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Positive and Negative Affect as Links Between Social Anxiety and Depression:

Predicting Concurrent and Prospective Mood Symptoms in Unipolar and Bipolar Mood Disorders

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Abstract

The co-occurrence of social anxiety and depression is associated with increased functional impairment and a more severe course of illness. Social anxiety disorder is unique among the anxiety disorders in sharing an affective profile with depression, characterized by low levels of positive affect (PA) and high levels of negative affect (NA). Yet it remains unclear how this shared affective profile contributes to the covariation of social anxiety and depressive symptoms. We examined whether self-reported PA and NA accounted for unique variance in the association between social anxiety and depressive symptoms across three groups (individuals with remitted bipolar disorder, type I [BD; n = 32], individuals with remitted major depressive disorder [MDD; n = 31], and nonpsychiatric controls [n = 30]) at baseline and follow-ups of 6 and 12 months. Low levels of PA, but not NA, accounted for unique variance in both concurrent and prospective associations between social anxiety and depression in the BD group; in contrast, high levels of NA, but not PA, accounted for unique variance in concurrent and prospective associations between social anxiety and depression in the MDD group. Limitations include that social anxiety and PA/NA were assessed concurrently and all measurement was self-report. Few individuals with MDD/BD met current diagnostic criteria for social anxiety disorder. There was some attrition at

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Conflict of Interest Statement

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follow-up assessments. Results suggest that affective mechanisms may contribute to the high rates of co-occurrence of social anxiety and depression in both MDD and BD. Implications of the differential role of PA and NA in the relationship between social anxiety and depression in MDD and BD and considerations for treatment are discussed.

Keywords

social anxiety; depression; bipolar disorder; positive affect; negative affect

Social anxiety and depression commonly co-occur. For instance, 41% of a treatment-seeking sample with major depressive disorder (MDD) also met diagnostic criteria for social anxiety disorder (SAD; Brown, Campbell, Lehman, Grisham, & Mancill, 2001), and 49% of an epidemiological sample with bipolar disorder (BD) met criteria for SAD (Sala et al., 2012). The emergence of depression in SAD results in greater functional impairment (Kessler, Stang, Wittchen, Stein, & Walters, 1999; Stein et al., 2001), greater risk for alcohol and substance dependence (Nelson et al., 2000), and higher rates of suicidality (Mineka, Watson, & Clark, 1998) than when SAD occurs alone. Similarly, the experience of social anxiety in mood disorders, such as BD and MDD, is associated with a more severe course of illness, greater functional impairment, higher rates of drug use, and more suicide attempts than mood disorders without social anxiety (Klein Hofmeijer-Sevink et al., 2012; Lamers et al., 2011; Sala et al., 2012; Saunders, Fitzgerald, Zhang, & McInnis, 2012). Thus, it is of substantial importance to investigate potential variables that may account for the association between social anxiety and depression. As of now, an empirically informed model of this association is underdeveloped.

Understanding the relationship between social anxiety and depression necessitates the study of shared psychological mechanisms. Theoretical models implicate emotional deficits and dysregulation in the development and maintenance of both mood and anxiety disorders (e.g., Heimberg, Brozovich, & Rapee, 2014; Hofmann, Sawyer, Fang, & Asnaani, 2012). For instance, individuals with depression demonstrate poor understanding of their emotions and are less accepting of their emotions than those without depression (Ehring, Tuschen-Caffier, Schnülle, Fischer, & Gross, 2010; Mennin, Holaway, Fresco, Moore, & Heimberg, 2007). Similarly, socially anxious individuals are characterized by difficulty identifying their emotions, describing their emotions, and differentiating between different types of emotion (Kashdan & Farmer, 2014; Mennin et al., 2007; Spokas, Luterek, & Heimberg, 2009; Turk, Heimberg, Luterek, Mennin, & Fresco, 2005). Thus, emotional difficulties may underlie both depression and social anxiety.

Researchers have proposed that high negative affect (NA) and low positive affect (PA) are core components of both social anxiety and depressive disorders (Gross & Jazaieri, 2014; Hofmann et al., 2012). Indeed, depression has long been characterized by high NA and low PA, with persistent sad mood and decreased experiences of pleasure being key characteristics of the disorder (American Psychiatric Association [APA], 2013; Clark & Watson, 1991). Anxiety, on the other hand, was originally defined by high NA and high physiological arousal (Clark & Watson, 1991). However, social anxiety, unlike other anxiety

disorders, is also characterized by low PA (Brown, Chorpita, & Barlow, 1998; Hughes et al., 2006). Thus, the affective characteristics of social anxiety (i.e., high NA and low PA) more closely resemble those of depression than of other types of anxiety. This pattern of PA and NA may partially explain the relationship between social anxiety and depression.

NA and PA in Social Anxiety

Compared to individuals low in social anxiety, socially anxious individuals report higher levels of trait NA (Watson, Clark, & Carey, 1988) and exhibit greater intensity and instability of NA over the course of a given day (Farmer & Kashdan, 2014). Furthermore, in situations involving potential social threat (e.g., a social interaction or conversation), socially anxious individuals experience more intense NA, as assessed by self-report and physiological reactivity, than individuals low in social anxiety (Kashdan & Farmer, 2014; Kashdan, Farmer, et al., 2013; Kashdan & Roberts, 2004; Moscovitch, Suvak, & Hofmann, 2010).

Research also highlights deficits in PA as a central feature of social anxiety (Brown et al., 1998; Gilboa-Schechtman, Shachar, & Sahar, 2014; Hughes et al., 2006; Kashdan, 2007). Socially anxious individuals exhibit lower levels of PA, on both a trait and daily basis, than individuals low in social anxiety (Kashdan & Steger, 2006). Low PA predicts social anxiety (Naragon-Gainey, Gallagher, & Brown, 2013), particularly during social interactions (Kashdan, Farmer, et al., 2013) and in response to daily stressors (Farmer & Kashdan, 2012). Moreover, a large meta-analysis demonstrated that social anxiety is consistently related to low PA, even after statistically controlling for depression (Kashdan, 2007).

NA and PA in MDD and BD

Like social anxiety, unipolar depression has been consistently associated with dysfunction related to NA. Compared to healthy controls, individuals with depression report higher NA on a trait level (Clark, Watson, & Mineka, 1994; Watson, Clark, & Carey, 1988) and on a day-to-day basis (Bylsma, Taylor-Clift, & Rottenberg, 2011; Mor et al., 2010). Deficits in positive emotion also play a central role in depression. A large meta-analysis found that depressed individuals exhibited lower levels of emotional reactivity to positive stimuli than nondepressed controls (Bylsma, Morris, & Rottenberg, 2008), and individuals in a current depressive episode reported experiencing less hedonic capacity in a probabilistic reward task (Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008). Individuals with depression also exhibit difficulty sustaining PA across time on both self-report and neurobiological indices (Heller et al., 2009; Shestyuk, Deldin, Brand, & Deveney, 2005).

The nature of NA and PA is less straightforward in BD. Individuals with BD do not seem to differ from healthy controls in their experience of NA (Gruber, 2011), a surprising finding given that BD is characterized by recurring depressive episodes (Judd et al., 2003). For instance, individuals with BD do not differ from healthy controls in their affective or physiological responses to stimuli designed to elicit negative emotion (Gruber, Johnson, Oveis, & Keltner, 2008), nor do they differ in NA in response to daily stressors (Myin-Germeys et al., 2003). Individuals with or at risk for BD do experience heightened and

sustained PA (Johnson, 2005; Johnson, Ruggero, & Carver, 2005; Lovejoy & Steuerwald, 1995), consistent with the elevated positive emotionality of mania (Gruber, 2011; Gruber et al., 2008). However, when measured at the trait level or for more extended time periods, individuals with BD exhibit lower levels of PA compared to healthy controls (Myin-Germeys et al., 2003), consistent with clinical observations of the depressive features of BD (e.g., Judd et al., 2003; Judd et al., 2005).

The Present Investigation

The links between social anxiety and depression are poorly understood. Thus, the present study investigated whether PA and NA account (in part) for the relationship between social anxiety and depression as a step toward an empirically driven model of this association.

We were interested in whether maladaptive patterns of NA and PA influence the relationship between symptoms of social anxiety and depression over time in both MDD and BD. Recent reviews have highlighted the mediating role of NA and PA in the development of affective disorders (e.g., Hofmann et al., 2012), and given recent meta-analytic evidence that psychotherapeutic interventions for depressed individuals increase PA and decrease NA, we wanted to explore the relationships of NA and PA with social anxiety and depression in adults with mood disorders (Boumparis, Karyotaki, Kleiboer, Hofmann, & Cuijpers, 2016). Specifically, we examined the contributions of trait NA and PA to the relationship between social anxiety and depressive symptoms, concurrently and prospectively, in adults with remitted MDD, adults with remitted BD, and healthy controls. ¹

The primary focus of our study was the dimensional relationships between social anxiety, depression, and affect. We examined individuals with remitted mood disorders (i.e., not currently in a depressed, hypomanic/manic or mixed mood episode) to best investigate enduring patterns of emotion regulation and affectivity independent of current symptom severity, consistent with prior research (Gruber, Kogan, Mennin, & Murray, 2013; Ong, Zaki, & Gruber, 2017). Involving individuals with remitted MDD and BD in a longitudinal design allowed us to probe whether social anxiety and affect were associated with increased depression concurrently and prospectively (i.e., 6 months and 12 months later) from a relatively asymptomatic baseline profile. Recent research has shown that interepisode affective intensity and instability predicts increases in depression in remitted BD individuals (Gershon & Eidelman, 2015). However, no research to date has examined PA and NA as prospective predictors of depression in remitted MDD, and no research has evaluated the role of social anxiety in either mood-disordered group.

We had two primary aims. The first was to examine the concurrent and prospective association between social anxiety and depressive symptoms in our sample. Given that large-scale studies indicate the onset of SAD precedes the development of MDD in up to 70% of comorbid cases (Fava et al., 2000; Kessler et al., 1999), we examined social anxiety as the predictor variable and depression as the outcome variable in our analyses. We

¹We also examined the associations between social anxiety and mania, but these analyses did not reveal significant findings. Given space constraints and our desire to give adequate focus to the depression analyses, we elected not to present the results on mania in this paper. All results are available upon request from the corresponding author.

hypothesized that social anxiety symptoms would be related to greater severity of depressive symptoms, both concurrently and prospectively (**Hypothesis 1**). Because research has demonstrated strong positive associations between social anxiety and depression in MDD, BD, and nonclinical samples (Naragon-Gainey, Watson, & Markon, 2009; Nolidin, Downey, Hansen, Schweitzer, & Stough, 2013), we collapsed across groups to examine the bivariate relationships between social anxiety and depression.

Our second aim was to examine the role of PA and NA in the relationship between social anxiety and depressive symptoms in individuals with remitted mood disorders and controls. The affective patterns of MDD parallel those of depression measured dimensionally, characterized by high NA and low PA (Brown et al., 1998; Bylsma et al., 2008; Clark et al., 1994; Watson, Clark, & Carey, 1988). Thus, we hypothesized that NA and PA would both account for unique variance in the association between social anxiety and depressive symptoms in remitted MDD (**Hypothesis 2a**). Given research suggesting that PA has a unique and important role in BD, whereas NA does not (Gruber, 2011; Myin-Germeys et al., 2003), we further hypothesized that low PA, but not NA, would account for unique variance in the relationship between social anxiety and depressive symptoms among individuals with remitted BD (**Hypothesis 2b**). Finally, given that nonclinical samples are not characterized by dysregulated affect, we did not expect NA or PA to account for unique variance in the relationship between social anxiety and depression in controls (**Hypothesis 2c**).

Method

Participants

Data for the present study were collected as part of a larger experimental protocol designed to examine emotion regulation in individuals with remitted BD relative to individuals with remitted MDD and individuals with no history of depression. Participants (60% female; $M_{age} = 31.22$, SD = 9.94) were individuals with remitted BD type I (n = 32), individuals with remitted MDD (n = 31), and healthy controls (CTL; n = 30) who did not meet diagnostic criteria for any current or past DSM-IV-TR (APA, 2000) Axis I disorder. As noted above, both MDD and BD clinical groups were currently remitted at the time of study entry in order to examine more enduring patterns of emotion regulation that were independent of current symptom status. Groups did not differ on any demographic variables (Table 1). Exclusion criteria included self-reported history of severe head trauma, stroke, neurological disease, medical illness (e.g., autoimmune disorder, HIV/AIDs), and current alcohol or substance abuse/dependence in the past 6 months. The Institutional Review Boards at Yale University and the University of Colorado Boulder approved the study procedures and all participants provided written informed consent.

Measures

Diagnostic Interview.—Diagnostic status was established through a combination of a structured diagnostic interview and clinician-administered symptom measures, administered by clinical psychology doctoral students and trained research assistants who were supervised by clinical psychology faculty. The same interviewer administered all clinician-rated assessments at the initial visit for a given participant. Diagnoses were assessed using the

Structured Clinical Interview for *DSM–IV* Patient Version (SCID-I/P; First, Spitzer, Gibbon, & Williams, 2002). Current and lifetime diagnoses were assessed for the CTL group, whereas only current comorbidities were assessed for the BD and MDD group due to practical time constraints. Approximately one-third of the SCID-I/P interviews (30%; n = 28) were independently reviewed by trained researchers and major discrepancies (i.e., errors) were resolved with informal consensus meetings. Final interrater reliability was greater than 90% for all current and lifetime MDD and BD diagnoses (all κ 's > 0.90). This suggests strong interrater agreement, though we note that the use of the "skip out" strategy (implemented when initial criteria for a diagnosis are not met) might have reduced opportunities for potential disagreement among raters (e.g., Talbot et al., 2012).

Interviewers further evaluated current mania and depressive symptoms in the past week using the Young Mania Rating Scale (YMRS; Young, Biggs, Ziegler, & Meyer, 1978) and the clinician-administered Inventory of Depressive Symptoms (IDS-C; Rush, Guillon, Basco, Jarrett, & Trivedi, 1996). For participants with past MDD or BD, remitted mood status (i.e., absence of a current manic, hypomanic, depressive, or mixed episode) was confirmed using the SCID-I/P mood modules for the past month and the symptom cutoffs for the past week on the YMRS (YMRS 7) and IDS-C (i.e., IDS-C 11; Gruber, Harvey, & Gross, 2012; Talbot, Hairston, Eidelman, Gruber, & Harvey, 2009).²

Beck Depression Inventory-Short Form (BDI-SF; Beck & Beck, 1972).—The BDI-SF is a 13-item questionnaire designed to assess the presence and severity of depression symptoms. The BDI-SF is an abbreviated 13-item version of the original 21-item Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961). Scores from the BDI-SF are highly correlated with scores from the standard BDI (r= .93), and the BDI-SF has demonstrated good internal consistency (Cronbach's α = .83), good convergent validity, and good discriminant validity (Reynolds & Gould, 1981). In the current sample, internal consistency of the BDI-SF was good at the baseline visit (α = .84), 6-month follow-up visit (α = .88), and 12-month follow-up visit (α = .83).

Brief Fear of Negative Evaluation Scale (BFNE; Leary, 1983).—The BFNE is a 12-item self-report measure of fear of negative evaluation rated on a Likert-type scale ranging from 1 (*not at all characteristic of me*) to 5 (*extremely characteristic of me*). The BFNE has been shown to have strong internal consistency (α = .90) and good convergent validity (Leary, 1983). Recent research has demonstrated that the eight items with straightforward wording yield stronger convergent and discriminant validity than the four reverse-scored items (Carleton, Collimore, McCabe, & Antony, 2011; Rodebaugh et al., 2011; Weeks et al., 2005). Therefore, only the eight straightforward items (BFNE-S) were used for data analyses. In the current sample, internal consistency for the 8-item BFNE-S was excellent (α = .95).

²Of note, three individuals with BD and seven individuals with MDD met full diagnostic criteria for current SAD during the diagnostic interview. This small sample is not surprising given the remitted nature of the sample. Nonetheless, this limited sample precluded us from making conclusions about SAD comorbidity, and we modeled social anxiety dimensionally rather than categorically.

Positive and Negative Affect Schedule (PANAS; Watson, Clark, & Tellegen,

1988).—The PANAS is a 20-item self-report measure that assesses trait levels of PA and NA. Participants were instructed to indicate the extent to which they typically experience 10 positively- and 10 negatively-valenced emotions on a 5-point Likert-type scale, ranging from 1 (*not at all characteristic of me*) to 5 (*extremely characteristic of me*). The PA and NA subscales have demonstrated excellent internal consistency (all a's > .85), as well as strong convergent validity (Watson, Clark, & Tellegen, 1988). In the current sample, internal consistency was good for both the PA (a = .84) and NA (a = .88) subscales.

Procedure

Participants were primarily recruited through online advertisements and flyers posted in mental health centers in the New Haven metropolitan area. Individuals interested in participation completed a preliminary telephone screening. Eligible individuals presented to the laboratory for an initial visit at Time 1 (T1) that included the diagnostic interview, experimental tasks not reported here, and a battery of self-report questionnaires (including the BFNE-S, BDI-SF, and PANAS) completed in person using the data collection program Qualtrics (Qualtrics, 2011). All eligible participants were invited to complete two follow-up assessments approximately six months (i.e., Time 2, T2) and 12 months (i.e., Time 3, T3) after their initial assessment.

At T2, participants completed an online battery of self-report questionnaires remotely, with current (i.e., past week) depressive symptoms measured using the BDI-SF. Retention rates were good, with 24 MDD individuals (77%), 23 BD individuals (72%), and 27 CTL individuals (90%) completing the 6-month follow-up assessment. T2 completers in the MDD group did not differ on any demographic variables, baseline social anxiety or depressive symptoms, or baseline trait NA but scored higher on baseline trait PA (M= 3.35, SD= 0.64) than noncompleters (M= 2.81, SD= 0.45), t(30) = -2.18, p= .04, d= 0.80. T2 completers in the BD group did not differ on any demographic variables, baseline social anxiety symptoms, or baseline trait affect scores but scored lower on baseline depressive symptoms (M= 15.76, SD= 2.96) than noncompleters (M= 20.44, SD= 5.64), t(28) = 2.36, p= .006, d= 0.89. T2 completers in the CTL group did not differ from noncompleters on any variables (p's > .05).

At T3, participants completed the same online battery of self-report questionnaires. Retention rates were similar to T2, with 23 MDD individuals (74%), 21 BD individuals (66%), and 26 CTL individuals (87%) completing the T3 follow-up. T3 completers in the MDD group did not differ from noncompleters on any variables (p's > .05). T3 completers in the BD group did not differ on any demographic variables, baseline social anxiety symptoms, or baseline trait affect scores but scored lower on baseline depressive symptoms (M= 15.42, SD = 2.89) than noncompleters (M= 20.18, SD = 5.10), t(28) = 3.28, p = .003, d = 1.24. T3 completers in the CTL group did not differ from noncompleters on any variables (p's > .05).

Data Analytic Plan

Analyses were conducted using Hayes' (2013) PROCESS macro for SPSS (IBM Corp., 2012). We hypothesized that trait PA or NA would account for unique variance in the relationship between self-reported social anxiety and depressive symptoms at each time point. When comparing effects among more than two groups, Hayes recommends creating dummy-coded variables to delineate the groups while still including all participants in the model, thereby retaining statistical power (Hayes, 2013). Accordingly, we created two dummy-coded variables with the CTL participants as the reference group. The PROCESS macro allows for the combination of effects into a single *conditional process* model. Such a model allows for the examination of variance accounted for by PA/NA in the context of a moderator. We entered our dummy-coded group variables as a multicategorical moderator of the variance accounted for by PA/NA in the relationship between social anxiety symptoms and depressive symptoms (PROCESS Model 16). Power analyses suggest that our sample size (N = 93) would be adequate to detect a medium effect size (i.e., $f^2 = .14$). Separate models were conducted for each time point.

PROCESS utilizes bootstrapping methods to estimate the intervening effects of the variable of interest with regard to the relation between other, distinct variables. Each of our analyses was performed using 5,000 bootstrap resamples to yield 95% confidence intervals (CIs) for the conditional indirect effects of self-reported PA and NA (Hayes, 2013). An estimated 95% CI that did not include zero would reflect that PA or NA accounted for unique variance in the relationship between self-reported social anxiety symptoms and self-reported mood symptoms for a particular diagnostic group.

It should be noted that the bootstrapping methods utilized by PROCESS are typically reserved for tests of mediation (Preacher & Hayes, 2008). However, because measures of social anxiety and measures of affect were collected simultaneously at the initial study visit, these data would not be appropriate for a fully mediational model, which requires temporal ordering and implies causal directionality (Maxwell & Cole, 2007). However, even when not applied to a causal mediation model, bootstrapping is advantageous in being able to make inferences about the indirect effect of any model that incorporates an intervening variable (i.e., PA and NA; Preacher & Hayes, 2008). When applied to cross-sectional data, these analyses highlight the unique variance accounted for by the intervening variable(s). Hayes (2013) also argues that using these analytic techniques with cross-sectional data is sometimes merited, particularly for hypothesis generation. For these reasons, we chose to use bootstrapping over a standard hierarchical regression model to examine the unique variance accounted for by PA and NA. Because we do not propose to make causal claims given the cross-sectional nature of some of our data, we refer to our analyses only as examining the unique variance accounted for by PA/NA in the relationship between social anxiety symptoms and depressive symptoms.

Lastly, we tested an alternative model in which the roles of social anxiety symptoms and affect were reversed. In the competing model, we examined the unique variance accounted for by social anxiety in the relationship between PA and NA and depressive symptoms. Given that models of comorbid social anxiety and depression have highlighted PA and NA as higher-order general factors, it is possible that affect may influence depressive mood

symptoms through worsening social anxiety symptoms (e.g., Mineka et al., 1998; Wang, Hsu, Chiu, & Liang, 2012). Evaluating this model provided an additional test of the hypothesized effects.

Results

Hypothesis 1: Bivariate Associations between Social Anxiety and Depressive Symptoms

Our results supported our first hypothesis. Across the entire sample, T1 social anxiety (BFNE-S) was significantly and positively correlated with depressive symptoms (BDI-SF) at T1 (r= .47, p< .001), T2 (r= .25, p= .03), and T3 (r= .28, p= .02). A complete report of the bivariate correlations for all symptom measures appears in Table 2.

Hypothesis 2a: Unique Variance Accounted for by NA/PA in the MDD Group

Our results supported this hypothesis for NA but not for PA. Analyses revealed a conditional indirect effect of NA for two of the three time points, such that NA accounted for unique variance in the relationship between social anxiety and depressive mood symptoms for the MDD group at T1 (b = 0.10, 95% CI[0.032, 0.201]) and T2 (b = 0.12, 95% CI[0.007, 0.275]), but not T3 (b = 0.07, 95% CI[-0.043, 0.248]). PA did not account for significant unique variance at T1 (b = 0.02, 95% CI[-0.014, 0.074]), T2 (b = 0.02, 95% CI[-0.013, 0.102]), or T3 (b = 0.02, 95% CI[-0.025, 0.063]).

Hypothesis 2B: Unique Variance Accounted for by NA/PA in the BD Group

This hypothesis was supported. For the BD group, NA did not account for significant unique variance at T1 (b = 0.07, 95% CI[-0.023, 0.151]), T2 (b = 0.09, 95% CI[-0.039, 0.257]), or T3 (b = 0.11, 95% CI[-0.110, 0.288]). However, PA accounted for significant unique variance in the relationship between social anxiety and concurrent T1 depressive symptoms (b = 0.05, 95% CI[0.014, 0.112]), as well as prospective depressive symptoms at T2 (b = 0.06, 95% CI[0.002, 0.270]) and T3 (b = 0.10, 95% CI[0.005, 0.296]).

Hypothesis 2c: Unique Variance Accounted for by NA/PA in the CTL Group

For the CTL group, NA did not account for significant unique variance in the social anxiety/depressive symptoms relationship at T1 (b = 0.08, 95% CI[-0.020, 0.294]), T2 (b = 0.01, 95% CI[-0.208, 0.213]), or T3 (b = 0.08, 95% CI[-0.001, 0.320]). Similarly, PA did not account for significant unique variance at T1 (b = 0.01, 95% CI[-0.001, 0.040]), T2 (b = 0.03, 95% CI[-0.011, 0.184]), or T3 (b = 0.02, 95% CI[-0.001, 0.097]). Data analytic results for each diagnostic group are displayed in Table 3.

Alternative Model

Results for the alternative model revealed that social anxiety did not account for unique variance in the relationship between affect and depressive symptoms for nearly all groups at all time points. The effect of social anxiety on the relationship between NA and depressive symptoms was significant only for the MDD group at T1 (b = 1.08, 95% CI[0.143, 2.363]). The effect of social anxiety on the relationship between PA and depressive symptoms was significant only for the MDD group at T1 (b = -0.56, 95% CI[-1.501, -0.057] and the CTL

group at T3 (b = -0.37, 95% CI[-1.394, -0.003].³ Given that the majority of the tested indirect effects were nonsignificant, and those that were significant were inconsistent, the hypothesized model provides more robust support for the relationships among social anxiety, affect, and depression.

Discussion

Understanding the association between social anxiety and depression is particularly important given the high prevalence rates, more severe clinical course, and increased functional impairment observed when social anxiety and depression co-occur (Lamers et al., 2012; Sala et al., 2012). Our study was the first to probe the ways in which social anxiety and trait-level affect contribute to depressive symptoms in individuals with remitted MDD and remitted BD. The pattern of results suggests that NA and PA are differentially implicated in the relationship of social anxiety to remitted MDD versus remitted BD. Specifically, NA accounted for unique variance in the relationship between social anxiety and depressive symptoms in remitted MDD, whereas PA accounted for unique variance in the relationship between social anxiety and depressive symptoms in remitted BD.

For our first aim, we examined the concurrent and prospective association between social anxiety and depressive symptoms. Our hypothesis that higher social anxiety would be related to more severe depression was supported. Social anxiety at baseline was positively associated with depressive symptoms at baseline, 6-month follow-up, and 12-month follow-up. These findings replicate and extend research on the positive association between social anxiety and depression. To our knowledge, only one other study has examined social anxiety in remitted BD or MDD samples, but that study did not examine the relationship between social anxiety and depression (Aydemir & Akkaya, 2011). Our findings demonstrate high levels of social anxiety among adults with remitted mood disorders and demonstrate a robust relationship between social anxiety and depressive symptoms at multiple time points.

For our second aim, we first investigated the contribution of NA and PA to the relationship between social anxiety and depressive symptoms in remitted MDD. We hypothesized that both NA and PA would account for unique variance in the association between social anxiety and depressive symptoms in MDD. Somewhat contrary to our hypothesis, NA, but not PA, accounted for significant variance in the relationship between social anxiety and depressive symptoms in the remitted MDD group at T1 and T2. Although social anxiety and depression share the affective profile of high NA and low PA, our findings suggest that it is only high levels of NA that contribute to depressive symptoms among socially anxious individuals with remitted MDD. Given that social anxiety precedes the development of a depressive episode in nearly three-quarters of cases (Kessler et al., 1999), our findings are important in furthering the understanding of this possible pathogenic pathway.

We next investigated the contribution of NA and PA to the relationship between social anxiety and depressive symptoms in remitted BD. We hypothesized that PA would negatively and uniquely account for the positive association between social anxiety and

³Results of the analyses of the alternative model are available from the corresponding author on request.

depressive symptoms among individuals with remitted BD. Consistent with our hypothesis, PA, but not NA, accounted for significant variance in this association at T1, T2, and T3. To our knowledge, our study is the first to examine the prospective effects of social anxiety and trait affect in a remitted BD sample. This is noteworthy given the high rates of co-occurrence between the two disorders (Sala et al., 2012) and recent emphases in the NIMH on understanding the role of positive valence systems in the etiology and maintenance of psychological disorders (e.g., Insel et al., 2010). Finally, and as expected, we found no significant conditional effects in the controls.

Our findings map onto literature that has identified PA dysfunction as a key characteristic of both social anxiety (e.g., Gilboa-Schechtman et al., 2014; Hughes et al., 2006; Kashdan, 2007) and BD (e.g., Gruber, 2011; Johnson, Gruber, & Eisner, 2007). Although individuals with BD often demonstrate heightened PA reactivity, many of these individuals have difficulty regulating this affect after initial responding. For instance, individuals with BD endorse more dysfunctional appraisals of their positive states compared with both MDD and healthy control groups (Kelly et al., 2011). Moreover, the tendency to appraise positive emotional states through a negative filter (i.e., dampening) is elevated in BD (compared with healthy and unipolar depressed populations) and has been associated with the onset of depressive symptoms in BD (Gilbert, Nolen-Hoeksema & Gruber, 2013).

Clinical Implications and Future Directions

All individuals with MDD or BD in the current study were remitted, and thus, findings may provide insight into factors that contribute to recurrence of mood episodes in individuals with MDD and BD. For instance, individuals with BD endorse more dysfunctional appraisals of their positive states compared with both MDD and healthy control groups (Kelly et al., 2011). Moreover, the tendency to appraise positive emotional states through a negative filter (i.e., dampening) is elevated in BD (compared with healthy and unipolar depressed populations) and has been associated with the onset of depressive symptoms in BD (Gilbert et al., 2013). Speculatively, a socially anxious individual with remitted BD might appraise social displays of positive emotion as excessive or embarrassing, leading to suppression of the expression of PA and potentially the suppression of the experience of PA. Indeed, individuals with social anxiety suppress their outward expression of positive emotion (Farmer & Kashdan, 2012), which in turn contributes to dampened PA, negative interpersonal consequences, and increased depression (Gross & John, 2003; Kashdan, Ferssizidis, Farmer, Adams, & McKnight, 2013; O'Toole, Jensen, & Fentz, 2014). Thus, for individuals with remitted BD, the suppression of positive emotion due to social concerns may be one way in which social anxiety confers risk for depression through changes in PA for individuals with remitted BD. This is in line with previous research demonstrating that dampening PA is associated with prospective onset of mood symptoms in remitted BD (Gilbert et al., 2013) and takes this one step further by associating this type of PA regulation specifically with social concerns.

Our results highlight the differential roles of NA and PA in the relationship between social anxiety and depressive symptoms in MDD compared to BD. Whereas high NA and low PA have both been identified as risk factors for depression broadly, it may be that high NA is a

risk factor specific to the co-occurrence of social anxiety and depressive symptoms among individuals with *unipolar*, rather than *bipolar*, depression. On the other hand, low PA may be a risk factor specific to the co-occurrence of social anxiety and depressive symptoms among individuals with bipolar depression. Thus, the shared affective profiles of social anxiety and depression may in part account for their high rates of co-occurrence, but our findings suggest that the specific affective components explaining that relationship are different for the depressive symptoms experienced in remitted MDD compared to remitted BD.

Our findings highlight the potential role of social anxiety in the recurrence and maintenance of depression among individuals with mood disorders. Clinical interventions for BD and MDD should include an assessment of social anxiety (even when mood symptoms have remitted) and potentially incorporate treatment components, such as cognitive restructuring and exposures, to target specific social fears. Addressing social anxiety in the larger treatment of BD and MDD may not only enhance recovery but may also prevent future recurrence of depressive episodes. These results also provide some specificity in targets for preventing recurrence of depressive symptoms in remitted MDD and BD samples. Specifically, focusing on decreasing elevated NA via behavioral activation or cognitive reappraisal strategies may help decrease the relationship between symptoms of social anxiety and future depressive symptoms in individuals with MDD, while targeting the dampening PA in the context of social anxiety may be important to prevent depressive recurrence in individuals with BD.

Although our study provides a novel examination of the role of affect in the relationship between social anxiety and depressive symptoms, several limitations must be acknowledged. First, the use of concurrently collected data (i.e., social anxiety and PA/NA) to explore a set of longitudinal associations prevented an examination of a true mediation model. Hayes (2013) argues that the use of these analytic techniques with concurrently collected data is sometimes merited, particularly for hypothesis generation, which is consistent with the novel nature of our analyses. Furthermore, to support our hypothesized model, we tested whether an alternative model might better account for relationships among the variables of interest. Conditional indirect effects from this alternative model were generally nonsignificant, providing further support for our original model. Nevertheless, future research should seek to replicate our findings with a fully longitudinal design in order to establish true mediating effects.

Second, only three individuals with BD and seven individuals with MDD met full diagnostic criteria for SAD, and thus, we elected to model social anxiety dimensionally, which precludes conclusions regarding comorbidity. Nevertheless, research suggests that even individuals with subthreshold social anxiety have significantly impaired functioning across multiple domains (Davidson, Hughes, George, & Blazer, 1994). Future research would benefit from examining a sample with a dual diagnosis of SAD and a mood disorder.

Third, both BD and MDD participants were specifically recruited to be in symptom remission at the time of testing. On the one hand, this represents a relative strength insofar as it enables better understanding of patterns of emotion regulation that may still be present during the remitted period and help explain interepisode dysfunction observed (e.g., Judd et

al., 2002; Kaplan et al., 2011). Indeed, up to 50% of BD individuals experience persistent difficulties during the remitted period that, furthermore, have been shown to predict depressive and manic symptom relapse (e.g., MacQueen et al., 2003). At the same time, future work is necessary to examine the relative influence of depressive and manic mood symptoms and their contribution to understanding affective links between social anxiety and depression.

Fourth, our study relied primarily on self-report measures. Although this methodology permits an understanding of subjective experience, future research should extend these findings with observational and clinician-assessed indices as well as laboratory assessments of psychophysiological mechanisms. Along related lines, our primary measure of social anxiety, the BFNE-S, requires interpretative caution. Fear of negative evaluation (FNE) is characterized by a sense of dread or apprehension about being judged unfavorably by others in social situations and is considered to be a core component of SAD (American Psychiatric Association, 2013; Heimberg et al., 2014). However, FNE could be considered a symptom of social anxiety rather than a representation of social anxiety itself, and as such, the BFNE may lack construct validity as a measure of social anxiety. Replication of our study using SAD symptom measures with high content validity, such as the self-report Social Interaction Anxiety Scale (Mattick & Clarke, 1998) or the clinician-administered Liebowitz Social Anxiety Scale (Liebowitz, 1987), would lend important support to our conclusions. Additionally, although our sample sizes were adequate, there was some attrition across time, and completers differed slightly from noncompleters (i.e., noncompleters were slightly less depressed than completers). Thus, future research would be well-served to replicate these findings and to do so in a larger sample to detect more subtle effects.

Despite these limitations, this investigation provided important insight into the transdiagnostic affective mechanisms that confer vulnerability for heightened depressive symptoms among individuals with a remitted mood disorder and elevated social anxiety. Our results suggest that the relationship between social anxiety and depression in MDD is partially explained by high levels of trait NA, whereas the relationship between social anxiety and depression in BD is partially explained by low levels of trait PA. Overall, our findings add specificity to the role of affect in the relationship between social anxiety and concurrent and prospective depression in remitted mood disorders.

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Table 1

Demographic and Self-Report Data by Diagnostic Group

	BD	MDD	CTL
Demographic			
Age in Years, $M(SD)$	30.81 (9.61)	31.42 (11.23)	31.45 (9.13)
Gender (% Female)	65.6	62.5	63.3
Race/Ethnicity, %			
Caucasian	87.5	90.6	90.0
African American	3.1	3.1	6.7
Asian American	6.3	0.0	0.0
Hispanic/Latino	3.1	6.3	3.3
Years of Education, $M(SD)$	15.08 (2.21)	15.16 (2.23)	15.95 (2.41)
Clinical			
YMRS, $M(SD)$	1.88 (1.90)	1.72 (1.87)	1.17 (1.05)
IDS-C, M (SD)	4.22 (3.27) ^a	5.03 (2.29) ^a	2.00 (1.98)
Age at Onset, Years	16.39	16.09	
Illness Duration, Years	14.61	15.34	
Measures of Interest			
BFNE-S (T1), $M(SD)$	23.34 (8.88) ^a	23.37 (7.59) ^a	15.50 (6.34)
PANAS-PA (T1), $M(SD)$	3.24 (0.56)	3.22 (0.64)	3.50 (0.66)
PANAS-NA (T1), $M(SD)$	1.72 (0.68) ^a	1.80 (0.63) ^a	1.24 (0.29)
BDI-SF (T1), $M(SD)$	17.17 (4.43) ^a	17.76 (3.30) ^a	14.07 (2.23)
BDI-SF (T2), $M(SD)$	16.70 (4.47)	16.67 (4.14)	16.11 (5.09)
BDI-SF (T3), $M(SD)$	17.14 (4.45) ^a	17.52 (4.21) ^a	14.69 (2.41)

Note. BD = Remitted Bipolar I Disorder Group; MDD = Remitted Major Depressive Disorder Group; CTL = Non-Psychiatric Control Group; YMRS = Young Mania Rating Scale; IDS-C = Inventory of Depressive Symptoms; BFNE-S = Brief Fear of Negative Evaluation Scale, Straightforward Items, PANAS-PA = Positive Affect Subscale, PANAS-NA = Negative Affect Subscale, BDI-SF = Beck Depression Inventory-Short Form.

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 $^{^{}a}\!\!$ Group mean is significantly (p < .05) different from the CTL group.

Table 2

Bivariate Correlations

Variables	T1 BFNE	T1 PANAS- PA	T1 PANAS-NA	T1 BDI-SF	T2 BDI-SF
T1 PANAS-PA	25*				
T1 PANAS-NA	.53***	17			
T1 BDI-SF	.47***	35 **	.53***		
T2 BDI-SF	.25*	30 **	.34**	.40**	
T3 BDI-SF	.28*	31 **	.41***	.56***	.61 ***

Note. BFNE-S = Brief Fear of Negative Evaluation Scale, Straightforward Items, PANAS-PA = Positive Affect Subscale, PANAS-NA = Negative Affect Subscale, BDI-SF = Beck Depression Inventory, Short Form. Sample sizes at T1 (n = 93), T2 (n = 74), and T3 (n = 70).

*p<.05;

** p < .01;

*** p<.001

Table 3

Conditional Indirect Effects by Group for the Effects of Positive and Negative Affect in the Relationship of Social Anxiety to Depressive Symptoms

Variable	Time Point	Group	Indirect Effect (b)	95% CI Lower Bound	95% CI Upper Bound
Positive Affect					
	Baseline				
		CTL	0.01	-0.001	0.040
		MDD	0.02	-0.014	0.074
		BD	0.05*	0.014	0.112
	6-Month				
		CTL	0.03	-0.011	0.184
		MDD	0.02	-0.013	0.102
		BD	0.06*	0.002	0.270
	12-Month				
		CTL	0.02	-0.001	0.097
		MDD	0.02	-0.025	0.063
		BD	0.10*	0.005	0.296
Negative Affect					
	Baseline				
		CTL	0.08	-0.020	0.294
		MDD	0.10*	0.032	0.201
		BD	0.07	-0.023	0.151
	6-Month				
		CTL	0.01	-0.208	0.213
		MDD	0.12*	0.007	0.275
		BD	0.09	-0.039	0.257
	12-Month				
		CTL	0.08	-0.001	0.320
		MDD	0.07	-0.043	0.248
		BD	0.11	-0.110	0.288

Note. BD = Remitted Bipolar I Disorder Group, MDD = Remitted Major Depressive Disorder Group, CTL = Non-Psychiatric Control Group; beta values for indirect effects are unstandardized;

^{*}indicates a significant (p < .05) conditional indirect effect as determined by the bootstrapped confidence intervals.