Published in final edited form as:

J Affect Disord. 2014 January ; 0: 483–490. doi:10.1016/j.jad.2013.07.039.

Self-referent information processing in individuals with bipolar spectrum disorders

Ashleigh Molz Adams^{a,*}, Benjamin G. Shapero^a, Laura H. Pendergast^a, Lauren B. Alloy^a, and Lyn Y. Abramson^b

^aTemple University, Philadelphia, PA, United States

^bUniversity of Wisconsin, Madison, MD, United States

Abstract

Background—Bipolar spectrum disorders (BSDs) are common and impairing, which has led to an examination of risk factors for their development and maintenance. Historically, research has examined cognitive vulnerabilities to BSDs derived largely from the unipolar depression literature. Specifically, theorists propose that dysfunctional information processing guided by negative self-schemata may be a risk factor for depression. However, few studies have examined whether BSD individuals also show self-referent processing biases.

Methods—This study examined self-referent information processing differences between 66 individuals with and 58 individuals without a BSD in a young adult sample (age $M = 19.65$, $SD = 1.74$; 62% female; 47% Caucasian). Repeated measures multivariate analysis of variance (MANOVA) was conducted to examine multivariate effects of BSD diagnosis on 4 self-referent processing variables (self-referent judgments, response latency, behavioral predictions, and recall) in response to depression-related and nondepression-related stimuli.

Results—Bipolar individuals endorsed and recalled more negative and fewer positive self-referent adjectives, as well as made more negative and fewer positive behavioral predictions. Many of these information-processing biases were partially, but not fully, mediated by depressive symptoms.

Limitations—Our sample was not a clinical or treatment-seeking sample, so we cannot generalize our results to clinical BSD samples. No participants had a bipolar I disorder at baseline.

Conclusions—This study provides further evidence that individuals with BSDs exhibit a negative self-referent information processing bias. This may mean that those with BSDs have selective attention and recall of negative information about themselves, highlighting the need for attention to cognitive biases in therapy.

© 2013 Elsevier B.V. All rights reserved.

*Correspondence to: Department of Psychology, Temple University, 1701 N. 13th Street, Philadelphia, PA 19122, United States. Tel.: +1 215 2048 766; fax: +1 215 204 5539. ashleigh.molz.adams@temple.edu (A. Molz Adams).

Conflict of interest

No contributing authors have any conflicts of interest.

Keywords

Bipolar spectrum disorders; Information processing; Self-schema

1. Introduction

Bipolar spectrum disorders (BSDs) are common and highly impairing psychological disorders (e.g., Goodwin and Jamison, 2007). As such, there has been increasing focus on understanding cognitive vulnerabilities and information processing deficits that may characterize individuals with BSDs. The purpose of this study is to examine self-referent encoding and recall in individuals with BSDs in comparison with healthy controls and to determine the extent to which identified differences are mediated by current mood symptoms.

1.1. Overview of bipolar spectrum disorders

Bipolar disorder accounts for over 96 million days of work loss in the United States every year (Kessler et al., 2006). In addition, mood episodes in individuals with bipolar disorder frequently occur more often than episodes in individuals with unipolar depressive disorders (Giles et al., 1986). Within the bipolar disorder category, several disorders form a spectrum of severity (Alloy et al., 2012; Akiskal et al., 1977; Birmaher et al., 2009; Goodwin and Jamison, 2007). Bipolar I disorder is the most severe, and is characterized by at least one manic episode; however, typically depressive episodes also occur (American Psychiatric Association (APA), 2000; Judd et al., 2002). Individuals with bipolar I are 12–15 times more likely than the general population to complete suicide (Angst et al., 2002). Bipolar II disorder is considered to be less severe and is characterized by a history of hypomanic episodes and at least one major depressive episode (American Psychiatric Association (APA), 2000). Cyclothymia, a more longstanding yet less severe disorder, is distinguished by mood variability and switching between milder hypomanic and depressive periods (American Psychiatric Association (APA), 2000). Despite the major personal, economic, and social impact of these disorders, compared to other mental illnesses, research on bipolar disorders is under represented (Hyman, 2000).

1.2. Cognitive risk factors for bipolar and unipolar depressive disorders

Given the wide-ranging detrimental effects of BSDs, there has been an emphasis on discovering risk factors for the development of these disorders. Early research examining the predictors of BSDs has been largely based on theories of unipolar depressive disorders (Alloy et al., 2005, 2006a, 2009). In the depression literature, two of the most influential theories of cognitive vulnerability (CV) are Beck's (1976, 1987) theory and the Hopelessness theory (Abramson et al., 1989). Beck's (1976, 1987) theory of depression suggests that some individuals have maladaptive schemas about the self, and extreme beliefs about the world that make them vulnerable to depression when they experience stressful life events. The Hopelessness theory (Abramson et al., 1989) proposes that individuals vulnerable to depression have characteristic tendencies to attribute negative life events to stable and global causes, infer that negative consequences will follow these events, and infer that the occurrence of a negative event in their lives means that they are fundamentally

flawed or worthless. Both theories suggest that when faced with negative life events, individuals with CVs are more likely to become depressed than those without these vulnerabilities; these theories have found considerable support in the literature (Alloy et al., 2006b; Gotlib and Joormann, 2010)

Research examining CV in BSDs has yielded support for cognitive styles that are similar to those seen in unipolar depression. Several studies suggest that cognitive styles during depressive states of BSD and those in unipolar depression are similar, as responses on measures of negative attitudes and thoughts about the self (Hollon and Kendall, 1980; Weissman and Beck, 1978) are not significantly different between these diagnostic groups during depressive episodes (Hill et al., 1989; Rosenfarb et al., 1998). Furthermore, several studies have found that those with BSD and unipolar depression also score similarly on these measures while in euthymic states (Hollon et al., 1986; Jones et al., 2005). As compared to healthy controls, individuals with BSD during a euthymic state have been shown to exhibit more negative inferential styles, dysfunctional attitudes, and greater sociotropy (Jones et al., 2005; Scott et al., 2000; Van der Gucht et al., 2009), as well as lower self-esteem and greater rumination (Van der Gucht et al., 2009; Jones et al., 2005), echoing comparisons of CV between healthy controls and individuals with unipolar depression (e.g., Weissman and Beck, 1978; Just and Alloy, 1997).

Although there are many similarities between individuals with BSDs and those with unipolar depression, research indicates that there are important differences as well. One cognitive difference found was that BSD patients had significantly more negative goal attainment dysfunctional attitudes on the DAS than unipolar depression patients while euthymic (Lam et al., 2004). This finding is consistent with the behavioral approach system (BAS) hyper-sensitivity model of BSD, which accounts for the occurrence of both hypomanic/manic and depressive episodes (Alloy et al., 2009; Alloy and Abramson, 2010; Depue and Iacono, 1989; Johnson, 2005; Urosevi et al., 2008). The BAS is a motivational system linked to goal-striving and approach to rewards (Gray, 1994). Individuals with BSD are hypothesized to have an overly sensitive BAS that leads to hypomanic/manic symptoms when the BAS becomes excessively activated by rewards or goals, and depressive symptoms when the BAS is excessively deactivated by cues of failure (Depue and Iacono, 1989; Urosevi et al., 2008). Notably, even while in hypomanic/manic states, individuals with BSDs continue to exhibit negative cognitive styles (French et al., 1996a). Other studies have found that hypomanic BSD patients' dysfunctional attitudes were not as extreme as those of unipolar depressed patients, but they were still more negative than those of healthy controls (Goldberg et al., 2008; Scott and Pope, 2003). Separate studies have shown that low self-esteem prospectively predicts both more depressive episodes (Johnson et al., 2000) as well as hypo/manic episodes (Scott and Pope, 2003) in BD. Yet another study found that more negative automatic thoughts only predicted a prospective increase in depressive, but not hypomanic, symptoms (Johnson and Fingerhut, 2004). Taken together, although investigation of CV in BD is underway, findings are still complex and inconclusive at this time.

1.3. Information processing in bipolar and unipolar depressive disorders

Alterations or deficits in information processing have been examined in a variety of ways, and there are both overt and covert methods; self-report measures explicitly ask participants to rate their thoughts and actions, whereas some studies have examined latency to respond to emotionally-laden stimuli (e.g., Greenberg and Alloy, 1989), and/or recall for learned words. Studies that have focused on emotionally neutral words have reported a variety of findings. Kieseppä et al. (2005) found that individuals with bipolar I disorder had significant impairments in processing speed, verbal learning, and verbal and visual memory as compared to their non-affected twins. However, Zalla et al., 2003 found that patients with BSDs had poor performance on the Stroop test, but not on verbal fluency or other executive functioning assessments, as compared to their first degree relatives. Slower processing speed has been suggested as a large contributing factor to the range of cognitive deficits shown in bipolar disorders. Antila et al. (2011) noted that processing speed was lowest in their BSD group, as compared to first-degree relatives of bipolar probands and healthy controls. Furthermore, controlling for processing speed, diagnostic differences in learning and recall were significantly lessened (Antila et al., 2011). Kieseppä et al. (2005) also noted that information processing speed could account for memory and learning deficits. Finally, Dittman et al., 2008 found that there were no significant differences on measures of psychomotor speed, working memory, or verbal learning, between a group of individuals with BSDs and a group of healthy controls.

Some investigations of information processing have begun to focus on emotionally-laden stimuli. This is an important direction research should take, in order to better understand the cognitive biases for mood and emotional content in those with bipolar spectrum disorders. The emotional Stroop test is commonly used for these investigations; in this task the participant must name the ink colors of emotionally-laden words. Lex et al. (2008a, 2008b) reported no information processing differences on the emotional Stroop test between individuals with BD I in remission and healthy controls. Using a test of encoding and recall for personality-relevant words, Pyle and Mansell (2010) reported that individuals at risk for BSDs recalled significantly more negative as well as positive words compared to those not at risk.

Beck's (1976)'s theory of depression states that there is a cognitive bias congruent with mood, and many studies have provided evidence for this in unipolar depression, with higher recall and encoding of depressive words (Blaney, 1986; see Haaga et al., 1991 for review), as well as an attentional bias for emotional stimuli, suggesting that depressed individuals selectively attend to emotional cues (Beevers et al., 2009). In this vein, Lim and Kim (2005) asked depressed individuals to perform the emotional Stroop test and found that depressed individuals showed interference (delayed color naming latency) for negative words, as well as a significant memory bias for negative words in a separate task. However, Greenberg and Alloy (1989) reported that depressed individuals had shorter latency to respond to depression-relevant words, but also rated friends more positively than themselves, compared to euthymic individuals. Within the bipolar spectrum literature, Lyon et al. (1999) found in an emotional Stroop task that bipolar depressed individuals exhibited slowed color naming for depressive words and recalled more negative words. Interestingly, Reilly-Harrington et

al. (1999) examined individuals with BSD and MDD during various mood states compared to healthy controls. They did not find differences in information processing between these groups; however, in combination with life events, information processing biases conferred risk for subsequent depressive symptoms for both BSD and MDD groups, and for subsequent hypomanic symptoms for the BSD group (Reilly-Harrington et al., 1999).

The hypothesis of a cognitive bias congruent with depressed mood has evidence from individuals with unipolar as well as bipolar depression, yet it remains unclear whether mania or hypomania is associated with more negative or more positive processing bias. There is mixed evidence for mood congruence in mania, as Murphy et al., 1999 reported that manic and depressed patients exhibited mood-congruent biases in a go/no-go task. Similarly, Lyon et al. (1999) reported on a sample of manic individuals and found that they performed similarly to depressed individuals in a Stroop test.

There are several clinical implications of a bias in information processing in bipolar spectrum disorders. These individuals may be at risk for misinterpreting events in the environment, which could trigger a manic or depressive episode. Understanding the biases that lead to these interpretations is crucial for therapeutic intervention. In addition, this understanding can broaden our understanding of similarities and differences between unipolar and bipolar disorders.

1.4. Limitations of prior research

Although some research has examined cognitive vulnerabilities and information processing associated with BSDs, further work is needed. Much of the research on cognitive vulnerabilities conducted in unipolar depressive disorders has been translated to BSDs. For instance, studies have shown that negative inferential style is similar in BSDs and depressive disorders (e.g., Scott et al., 2000). However, very few studies have examined cognitive vulnerabilities in BSD employing behavioral tasks, despite the fact that such research has shown promise in the depression literature (e.g., Beevers et al., 2009). Behavioral measures of information processing may be especially important in the study of cognitive vulnerabilities in BSDs, as self-report measures have yielded results that suggest high levels of social desirability and conformity in this population (Pardoen et al., 1993; Scott et al., 2000). Furthermore, researchers suggest that individuals with BSDs are likely to have negative thoughts and feelings about the self that are not detectable by self-report measures (Winters and Neale, 1985), adding evidence to the widely known “manic defense” theory (Neale, 1988). Another study examining a BSD sample found that implicit behavioral measures showed negative cognitive styles similar to a depressed sample, whereas explicit measures showed greater endorsement of positive words (Lyon et al., 1999). Research in this area is limited, and prior research employing behavioral measures has had some methodological limitations, including small samples with varying clinical characteristics between samples.

1.5. Present study

The current study takes an important step beyond prior research by examining the self-referent information processing differences between individuals with and without a BSD.

Furthermore, we collected hypomanic and depressive symptom information from all participants, and examined the extent to which mood symptoms mediated group differences in information processing biases. Research has shown that even in subsyndromal states, individuals with bipolar disorder experience mood symptoms that affect functioning (Bauer et al., 2010; Benazzi, 2004). Other research has found that during euthymic periods, those with BSDs routinely display cognitive deficits (Antila et al., 2011; Kieseppä et al., 2005) that may be influenced by subsyndromal mood symptoms (Robinson et al., 2006). The models investigated in this study allowed us to examine the extent to which diagnostic group, mood symptoms, and the relationship between the two, influenced information processing biases for self-referent, emotionally valenced content. Given the aforementioned research demonstrating negative cognitive styles in individuals with BSDs (Bentall and Thompson, 1990; French et al., 1996b; Scott and Pope, 2003), we hypothesized that our BSD participants would exhibit significantly more negative information processing biases than the healthy control participants. We further hypothesized that this effect would be partially, but not fully, mediated by concurrent levels of mood symptoms, highlighting an underlying cognitive vulnerability that is not totally dependent on mood state.

2. Methods

2.1. Participants and procedures

The current study employed a sample from the Longitudinal Investigation of Bipolar Spectrum Disorders Project (LIBS; Alloy et al., 2008, 2012). Participants for the LIBS Project were recruited from Temple University and the University of Wisconsin-Madison through a two-stage screening process. In phase I, 20,500 individuals consented for screening with the General Behavior Inventory (GBI; Depue et al., 1989), a first-stage case identification measure, to assess whether they might be eligible for the bipolar spectrum group or the healthy control group. Based on GBI cutoffs (see measures), eligible participants were invited for the diagnostic screening phase (phase II) of the study. The aim of the LIBS Project was to examine predictors of onset of first manic episode; therefore, individuals diagnosed with Bipolar I (i.e., those who had already experienced a manic episode) were excluded from participating in the study. Only participants who met criteria for Diagnostic and Statistical Manual of Mental Disorders 4th Edition (*DSM-IV*; American Psychiatric Association, 2000) or research diagnostic criteria (RDC; Spitzer et al., 1978) diagnoses of Bipolar II, Bipolar NOS, or Cyclothymia were invited to participate in the longitudinal part of the study and comprised the bipolar spectrum group. Participants who did not meet criteria for any *DSM-IV* or RDC Axis I psychiatric disorder and had no family history of BSDs were also asked to participate and constituted the healthy control group. Following the two-stage screening process, a total of 206 participants met criteria and were included in the bipolar spectrum group (age $M = 19.6$, $SD = 1.6$; 62.56% female; 68.9% Caucasian) and 208 participants were included in the healthy control group ([age $M = 19.7$, $SD = 1.5$; 59.22% female; 72.8% Caucasian] Alloy et al., 2008). These participants provided consent for the longitudinal portion of the study at Time 1, at which time they completed a variety of behavioral and symptom measures.

The current study used a subsample of the LIBS project Temple University participants, and includes 66 bipolar spectrum participants, and 58 control participants (see Table 1 for demographic information), for whom we had complete information on all measures of interest. This subsample did not significantly differ from the larger Temple University sample on measures of baseline depressive and hypomanic symptoms, gender, age, or ethnicity. The bipolar and control groups also did not differ significantly from each other on gender, ethnicity, or age (see Table 1 for *t*-tests).

2.2. Measures

Phase I—The revised GBI is a self-report measure used to differentiate potential bipolar and normal participants eligible to participate in the diagnostic interview. The GBI is a 73-item, self-report scale, in which participants rate bipolar symptoms (with intensity and duration) on a 4-point scale (1 = never, 4 = very often). We used Depue et al. (1989)'s scoring method and cutoff recommendations, such that participants with a score ≥ 11 on the depression (*D*) subscale and ≥ 13 on the hypomanic-biphasic (HB) subscale were identified as potential bipolar participants, and those with a *D* score < 11 and an HB score < 13 became the potential control group; these individuals were invited to participate in phase II. The GBI has good internal consistency ($\alpha=0.90-0.96$), test-retest reliability ($r = 0.71-0.74$), high specificity (0.99) and adequate sensitivity (0.78) for bipolar spectrum conditions. The GBI was validated through findings of similar rates of mood disorders in a large sample as in the general population, high specificity between mood disorders, and high correlations with interview ratings (Depue et al., 1989). In the LIBS project sample, the internal consistency was high for both the HB and D subscales (α 's=.95 and .87, respectively).

Phase II—Trained diagnostic interviewers conducted expanded Schedule for Affective Disorders and Schizophrenia—Lifetime (exp-SADS-L; Endicott and Spitzer, 1978) diagnostic interviews with every participant that came in for a Phase II visit. The interview was expanded to include both DSM-IV and RDC criteria. As stated above, those diagnosed with Bipolar II Disorder, cyclothymia, or Bipolar NOS, and those with no lifetime history of any Axis I disorder or family history of mood disorders, were all invited to participate. The SADS-L is a well-validated diagnostic interview that has high inter-rater reliability (Endicott and Spitzer, 1978), including in the LIBS Project ($\alpha > .96$ for BSD diagnoses; Alloy et al., 2008,2012). Depressive symptoms were assessed with the Beck Depression Inventory (BDI; Beck et al., 1979). The BDI is a 21-item self-report scale that assesses affective, cognitive, motivational, and somatic symptoms of depression. Total scores range from 0 to 63 with higher scores indicating more severe symptoms. The BDI has demonstrated good internal consistency, retest reliability, and concurrent validity with clinical depression ratings in clinical ($r=.72$) as well as nonclinical ($r=.60$) samples (Depue et al., 1981). In the current sample, internal consistency was good ($\alpha=.78$).

Hypomanic/manic symptoms were assessed with the Halberstadt Mania Inventory (HMI; Alloy et al., 1999). The HMI is a 28-item self-report questionnaire designed to assess current affective, cognitive, motivational, and somatic symptoms of hypomania/mania. In the larger study, the HMI demonstrated construct validity and was correlated ($r=.46$) with symptoms reported on diagnostic interview (Alloy et al., 2008). Similarly, in a sample of

undergraduates (Alloy et al., 1999), the HMI showed high internal consistency ($\alpha=.82$) and adequate discriminate validity with the BDI ($r = -.12, p < .001$). In the current sample, internal consistency was high ($\alpha=.82$).

Time 1—Participants completed the Self-Referent Information Processing (SRIP) task, which was previously used in the Cognitive Vulnerability to Depression study (see Alloy et al., 1997 for development). The first part of this task was conducted on a computer; participants were presented with a series of adjectives on a computer screen and instructed to press one of two buttons (“me” and “not me”) to indicate whether each word described them. Participants were asked to respond to each adjective as quickly as possible and their response times (RTs) were recorded. Adjectives were presented at random, and each participant saw 40 words in total, including 20 depression-related words, and 20 words unrelated to depression (Alloy et al., 1997). Immediately after viewing and responding to all the adjectives on the computer screen, participants were asked to read 10 more adjectives that “can be used to describe a person.” They were instructed to circle each one that pertained to themselves, and give specific behavioral examples from their past as to why that adjective described them. Next, participants were asked to read sentences describing behaviors and reactions that may or may not be true of them, and rate each sentence on a 0-100 scale, with 0 meaning extremely unlikely and 100 meaning extremely likely that they would behave in the manner described. After a delay of about an hour, participants were asked to recall as many adjectives as possible from the computer task, in any order. They were given 5 min to complete this task. Four types of scores were calculated from participants’ responses on the SRIP tasks: self-referent responses, behavioral predictions, recall, and response latency.

Self-referent responses were calculated by summing the number of times “like me” was endorsed for positive and negative adjectives. Participants’ responses to the behavioral predictions task were averaged separately for positive and negative statements relating to the self; i.e., higher scores on positive behavioral predictions signifies believing one is more likely to engage in positive behaviors. Research assistants coded the recalled words into negative and positive stimulus words, as well as intrusions. For our analyses, we calculated negative words recalled as a proportion of all words recalled, positive words recalled as a proportion of all words recalled, and number of all intrusions. Latency is the length of time to respond (“like me” or “not like me”) to target words, calculated separately for depression-relevant and depression-irrelevant words.

3. Results

3.1. Statistical analyses

Repeated measures multivariate analysis of variance (MANOVA) was used to examine multivariate effects of BSD diagnosis on a linear composite of 4 SRIP variables: latency in responding, number of times “like me” was endorsed in response to stimulus words, behavioral predictions, and proportion of stimulus words recalled. Each of these variables utilized four types of stimuli representing a 2 (valence) $\times$ 2 (content) design. Specifically, stimulus words were either positive or negative (valence) and relevant or irrelevant to depressive self-concept (content). Therefore, all SRIP variables were within subjects,

whereas diagnosis was a between subjects variable, leading to a mixed design. Diagnostic group $\times$ content $\times$ valence interactions were examined. Significant effects from the MANOVA were followed by univariate analyses of variance (ANOVAs) to determine which SRIP variables best accounted for the significant differences.

3.2. Primary analyses

The MANOVA revealed main effects for BSD diagnosis ($F(3,104) = 5.048, p = .003$), content ($F(4,103) = 131.145, p < .001$), and valence ($F(4,103) = 119.411, p < .001$), as well as interactions of content $\times$ valence ($F(4,103) = 35.086, p < .001$), content $\times$ BSD ($F(4,103) = 4.085, p = .004$), and valence $\times$ BSD ($F(4,103) = 10.011, p = .004$). Most notably, individuals with BSD performed differently than their healthy control counterparts on a linear composite of SRIP variables designed to tap self-schema. Specifically, individuals with BSD were more likely than controls to exhibit responses indicative of a negative self-schema when presented with adjectives that either had a negative valence or were relevant to depressive self-concept. We did not find a significant three way interaction between content, valence, and diagnosis ($F(4,103) = 0.858, p = .492$).

3.2.1. Self-referent responses—Follow-up ANOVAs were conducted to more fully examine significant effects identified from the MANOVA. Findings from all ANOVAs are reported in Table 2. In regard to self-referent responses, we found that a BSD diagnosis predicted a lower likelihood of responding ‘like me’ to positive adjectives ($F(1,122) = 21.39, p < .001$) and a higher likelihood of responding ‘like me’ to negative adjectives ($F(1,122) = 19.60, p < .001$).

Additionally, we investigated whether concurrent mood symptoms mediated the relationship between BSD diagnosis and frequency of “like me” responses to positive and negative stimulus words. Following recommendations for mediation analyses with small samples (MacKinnon et al., 2010), the nonparametric, bias-corrected bootstrap method with 5000 resamples to derive 95% confidence intervals for indirect effects was conducted (Preacher and Hayes, 2004). *Indirect*, an SPSS macro, was used to conduct all mediation analyses (Preacher and Hayes, 2008). Concurrent depressive symptoms fully mediated the relationship between BSD diagnosis and number of ‘like me’ responses to negative adjectives. The effect was positive (.67), such that higher BDI depressive symptoms led to more endorsements of negative words. The indirect effect was statistically significant ($p < .05$) and estimated to lie between .08 and 1.38 with 95% confidence. In regard to positive stimulus words, concurrent depressive symptoms fully mediated the relationship between BSD and number of positive words endorsed; the effect was negative (−1.09), such that higher BDI depressive symptoms led to fewer “like me” responses to positive stimuli. The effect was statistically significant ($p < .05$) and estimated to lie between −2.18 and −.34 with 95% confidence. Hypomanic symptoms did not mediate the effect of BSD diagnosis on number of positive or negative adjectives endorsed.

3.2.2. Behavioral predictions—Bipolar diagnosis predicted lower positive behavioral predictions ($F(1,106) = 6.965, p = .010$), as well as higher negative behavioral predictions ($F(1,106) = 26.583, p < .001$). Concurrent depressive symptom levels fully mediated the

relationship between bipolar diagnosis and negative behavioral predictions; the indirect effect was positive (7.79), statistically significant ($p < .05$), and estimated to lie between 1.54 and 14.75 with 95% confidence. This indicates that higher depressive symptoms led to higher negative behavioral predictions. Depressive symptoms did not have a significant indirect effect on positive behavioral predictions, and hypomanic symptoms did not have significant indirect effects on positive or negative behavioral predictions.

3.2.3. Recall—Similarly, bipolar diagnosis predicted lower likelihood of recalling positive adjectives ($F(1,122) = 28.907, p < .001$), as well as higher likelihood of recalling negative adjectives ($F(1,122) = 31.824, p < .001$). Recall variables were calculated as a proportion of each individual's total number of words recalled. Depressive symptoms partially mediated the relationship between bipolar diagnosis and proportion of negative words recalled, signifying that there was an indirect effect of depressive symptoms, but it did not negate the effect of bipolar diagnosis. The indirect effect was positive (.48), statistically significant ($p < .05$), and estimated to lie between -1.72 and $-.36$ with 95% confidence. This indicates that higher depressive symptoms led to higher recall of negative words. Depressive symptoms fully mediated the relationship between bipolar diagnosis and positive words recalled; the indirect effect was negative ($-.96$), statistically significant ($p < .05$), and estimated to lie between 0.024 and 0.973 with 95% confidence. This indicates that higher depressive symptoms led to fewer positive words recalled. Hypomanic symptoms did not have significant indirect effects on positive or negative words recalled.

3.2.4. Latency—Bipolar diagnosis predicted shorter latency to respond to depression-irrelevant stimuli, but this effect was non-significant. Healthy controls responded significantly more quickly to depression-relevant stimuli ($F(1,123) = 5.056, p = .026$). Neither depressive nor hypomanic symptoms had significant indirect effects on the latencies in responding to either type of stimuli.

4. Discussion

Prior research has shown that negative self-referent information processing biases are closely linked to unipolar depression. This study extends these previous findings to bipolar spectrum disorders, and the current results suggest that information processing biases regarding the self, particularly endorsing and recalling more negative and fewer positive adjectives as self-descriptive and making more negative and fewer positive behavioral predictions, are also linked to bipolar disorder. Moreover, in many instances, these information-processing biases are partially, but not fully, mediated by depressive symptoms. Hypo/manic symptoms do not appear to be influential. It is important to note here that individuals in the BSD group had significantly higher depressive symptoms at baseline ($t(59.45) = -6.17, p < .001$), whereas the difference in hypomanic symptoms was only marginally significant ($t(77.33) = -1.69, p < .10$). These findings suggest that whereas concurrent depressive, but not hypomanic, symptoms greatly affect self-referent information processing in the direction of greater negative biases, there is also some inherent vulnerability to negative self-referent processing in having a BSD diagnosis, as the effects of diagnostic group were not fully mediated by current depressive symptom levels in some analyses.

These findings are important inasmuch as we found that a bipolar spectrum diagnosis is characterized by an overall more negative self-referent processing style as compared to healthy controls. Depressive symptoms only partially mediate these effects, suggesting that whereas subsyndromal symptoms affect self-referent processing, a bipolar diagnosis itself may be associated with negative self-referent processing. This adds to the literature on cognitive vulnerabilities in BSDs by employing behavioral measures that assess implicit cognitive styles, suggesting that those with BSDs may process information about themselves more negatively. We also extend prior studies that found a negative processing bias in BSDs, even during euthymic states (e. g., Hollon et al., 1986; Jones et al., 2005; Scott et al., 2000). Given that this negative processing bias is partially, but not fully, mediated by subsyndromal depressive symptoms, future research should further examine the effects of subsyndromal mood symptoms. There are many possible clinical implications of negative self-referent information processing. Beck's (1976)'s theory holds that those with depression exhibit a negatively valenced bias in all types of information processing, leading to selective attention for negative information (Beevers and Carver, 2003) and interpreting ambiguous information in negative ways (Rude et al., 2002). If this bias is also seen in those with BSD, we should expect similar performance on these laboratory tasks and negative biases in everyday functioning.

We did find that healthy controls responded more quickly to depression-relevant stimuli; however, we did not have enough statistical power to examine this effect with regards to the actual responses (i.e., "like me" or "not like me"). We did not find that hypomanic symptoms mediated the effect of BSD diagnosis on self-referent processing. Perhaps hypo/manic symptoms at the subsyndromal level have little effect on information processing. However, it is important to note that this sample consisted of those with a BSD (primarily Bipolar II), and no participants had yet developed bipolar I disorder. Other research has shown that bipolar spectrum individuals are more prone to depressive episodes and subsyndromal depressive symptoms, rather than hypomanic symptoms and episodes (Judd et al., 2003a). Although the current study found a negative information processing bias, it is possible that hypomanic symptoms affect those with bipolar I disorder in an overly positive manner. The BAS model of BSDs holds that when faced with activating life events, there is an overreaction of the BAS system, leading to overactivity and euphoria. The current study suggests that individuals with BSDs may be prone to a slightly more negative interpretation of information relevant to themselves.

Several limitations of this study should be noted. We did not have enough statistical power to detect differences in latency by response type, which may have led to many more interesting results. Further research should include larger samples. Furthermore, our BSD sample was not a clinical or treatment-seeking sample, so we cannot necessarily generalize our results to clinical samples of individuals with BSDs. Future research should be conducted with clinical samples to replicate and extend our findings. Finally, our BSD sample only included individuals with the milder bipolar spectrum disorders, as no participants had a diagnosis of bipolar I disorder at baseline. With the full range of BSD diagnoses, we may see further differences in information processing.

The cognitive vulnerability literature discusses mood effects following activating life events. This study does not challenge that, but examines processing biases in euthymic states. Prior research on cognitive vulnerabilities has highlighted the importance of understanding basic differences in interpretation of the environment. The current study shows that while not emotionally activated by stressful life events or mood episodes, individuals with BSD are more negatively biased in self-referent information processing, which could explain their proneness to experiencing depressive episodes (Judd et al., 2003b). Although it is generally thought that those with BSD and MDD are distinct groups, their cognitive processing biases appear to be similar in that in general both groups are more inclined to encode the environment as negative while in a euthymic state. This has implications for practitioners in understanding individuals with BSD, as it may be essential to remember that these individuals process the world in a negative way, until activating life events prompt a swing to a hypomanic state.

The current study provides further evidence that individuals with a BSD exhibit a negative self-referent information processing bias in covert tasks, such as recall for adjectives, as well as overt tasks, such as behavioral predictions. Although we did not find many differences between the BSD and control groups in the latency to respond, latency may not be as important as the encoding and storage of self-referent information. Many of the group differences were partially mediated by depressive symptoms, highlighting the strong effects that subsyndromal symptoms have on information processing. However, even accounting for subsyndromal symptoms, we found an effect of diagnosis, suggesting that bipolar spectrum status alone is also associated with this negative processing bias.

Acknowledgments

This research was made possible by the members of the laboratories of Lauren Alloy at Temple University and Lyn Abramson at University of Madison-Wisconsin.

Role of funding source

This research was supported by National Institute of Mental Health Grants MH 52617 and MH 77908 to Lauren B. Alloy and MH 52662 to Lyn Y. Abramson. The National Institute of Mental Health had no role in data analysis, interpretation, or the writing of this manuscript.

References

- Abramson LY, Metalsky GI, Alloy LB. Hopelessness depression: a theory-based subtype of depression. *Psychological Review*. 1989; 96:358–372.
- Akiskal HS, Djenderedjian AH, Rosenthal RH, Khani MK. Cyclothymic disorder: validating criteria for inclusion in the bipolar affective group. *The American Journal of Psychiatry*. 1977; 134:1227–1233. [PubMed: 910973]
- Alloy LB, Urošević S, Abramson LY, Jager-Hyman S, Nusslock R, Whitehouse WG, Hogan M. Progression along the bipolar spectrum: a longitudinal study of predictors of conversion from bipolar spectrum conditions to bipolar I and II disorders. *Journal of Abnormal Psychology*. 2012; 121:16–27. [PubMed: 21668080]
- Alloy LB, Abramson LY. The role of the behavioral approach system (BAS) in bipolar spectrum disorders. *Current Directions in Psychological Science*. 2010; 19(3):189–194. [PubMed: 20606725]
- Alloy LB, Abramson LY, Murray LA, Whitehouse WG, Hogan ME. Self-referent information-processing in individuals at high and low cognitive risk for depression. *Cognition & Emotion*. 1997; 11:539–568.

- Alloy LB, Abramson LY, Urosevic S, Bender RE, Wagner CA. Longitudinal predictors of bipolar spectrum disorders: a behavioral approach system (BAS) perspective. *Clinical Psychology*. 2009; 16:206–226. [PubMed: 20161008]
- Alloy LB, Abramson LY, Urosevic S, Walshaw PD, Nusslock R, Neeren AM. The psychosocial context of bipolar disorder: environmental, cognitive, and developmental risk factors. *Clinical Psychology Review*. 2005; 25:1043–1075. [PubMed: 16140445]
- Alloy LB, Abramson LY, Walshaw PD, Cogswell A, Grandin LD, Hughes ME, Iacoviello BM, et al. Behavioral approach system and behavioral inhibition system sensitivities and bipolar spectrum disorders: prospective prediction of bipolar mood episodes. *Bipolar Disorders*. 2008; 10:310–322. [PubMed: 18271911]
- Alloy LB, Abramson LY, Walshaw PD, Cogswell A, Smith JM, Neeren AM, Hughes ME, et al. Behavioral approach system (BAS) sensitivity and bipolar spectrum disorders: a retrospective and concurrent behavioral high-risk design. *Motivation and Emotion*. 2006a; 30:143–155.
- Alloy LB, Abramson LY, Walshaw PD, Gerstein RK, Keyser JD, Whitehouse WG, Urosevic S, et al. Behavioral approach system (BAS)-relevant cognitive styles and bipolar spectrum disorders: concurrent and prospective associations. *Journal of Abnormal Psychology*. 2009; 118:459–471. [PubMed: 19685944]
- Alloy LB, Abramson LY, Whitehouse WG, Hogan ME, Panzarella C, Rose DT. Prospective incidence of first onsets and recurrences of depression in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology*. 2006b; 115:145–156. [PubMed: 16492105]
- Alloy LB, Reilly-Harrington N, Fresco DM, Whitehouse WG, Zechmeister JS. Cognitive styles and life events in subsyndromal unipolar and bipolar disorders: stability and prospective prediction of depressive and hypomanic mood swings. *Journal of Cognitive Psychotherapy*. 1999; 13(1):21–40.
- American Psychiatric Association (APA). *Diagnostic and Statistical Manual of Mental Disorders*. 4. Washington, DC: 2000. text rev
- Angst F, Stassen HH, Clayton PJ, Angst J. Mortality of patients with mood disorders: follow-up over 34–38 years. *Journal of Affective Disorders*. 2002; 68(2-3):167–181. [PubMed: 12063145]
- Antila M, Kieseppä T, Partonen T, Lönngqvist J, Tuulio-Henriksson A. The effect of processing speed on cognitive functioning in patients with familial bipolar I disorder and their unaffected relatives. *Psychopathology*. 2011; 44(1):40–45. [PubMed: 21042040]
- Bauer M, Glenn T, Grof P, Schmid R, Pfennig A, Whybrow PC. Subsyndromal mood symptoms: a useful concept for maintenance studies of bipolar disorder? *Psychopathology*. 2010; 43(1):1–7. [PubMed: 19893338]
- Beck, AT. *Cognitive Therapy of the Emotional Disorders*. New American Library; New York: 1976.
- Beck AT. Cognitive models of depression. *Journal of Cognitive Psychotherapy*. 1987; 1:5–37.
- Beck, AT.; Rush, AJ.; Shaw, BF.; Emery, G. *Cognitive Therapy of Depression*. Guilford; New York: 1979.
- Beevers CG, Carver CS. Attentional bias and mood persistence as prospective predictors of dysphoria. *Cognitive Therapy and Research*. 2003; 27:619–637.
- Beevers CG, Wells TT, Ellis AJ, McGeary JE. Association of the serotonin transporter gene promoter region (5-HTTLPR) polymorphism with biased attention for emotional stimuli. *Journal of Abnormal Psychology*. 2009; 118(3):670–681. [PubMed: 19685963]
- Benazzi F. Inter-episode mood lability in mood disorders: residual symptom or natural course of illness? *Psychiatry and Clinical Neurosciences*. 2004; 58(5):480–486. [PubMed: 15482578]
- Bentall RP, Thompson M. Emotional Stroop performance and the manic defense. *British Journal of Clinical Psychology*. 1990; 29:235–237. [PubMed: 2364203]
- Birmaher B, Axelson D, Goldstein B, Strober M, Gill MK, Hunt J, Houck P, et al. Four-year longitudinal course of children and adolescents with bipolar spectrum disorders: the course and outcome of bipolar youth (COBY) study. *The American Journal of Psychiatry*. 2009; 166:795–804. [PubMed: 19448190]
- Blaney PH. Affect and memory: a review. *Psychological Bulletin*. 1986; 99:229–246. [PubMed: 3515383]

- Depue RA, Krauss S, Spoont MR, Arbisi P. General behavior inventory identification of unipolar and bipolar affective conditions in a nonclinical university population. *Journal of Abnormal Psychology*. 1989; 98(2):117–126. [PubMed: 2708652]
- Depue RA, Iacono WG. Neurobehavioral aspects of affective disorders. *Annual Review of Psychology*. 1989; 40:457–492.
- Depue RA, Slater JF, Wolfstetter-Kausch H, Klein D, Goplerud E, Farr D. A behavioral paradigm for identifying persons at risk for bipolar depressive disorder: a conceptual framework and five validation studies. *Journal of Abnormal Psychology*. 1981; 90(5):381–437. [PubMed: 7298991]
- Dittman S, Hennig-Fast K, Gerber S, Seemüller F, Riedel M, Severus WE, et al. Cognitive functioning in euthymic bipolar I and bipolar II patients. *Bipolar Disorders*. 2008; 10:877–887. [PubMed: 19594503]
- Endicott J, Spitzer RL. A diagnostic interview: the Schedule for Affective disorders and Schizophrenia. *Archives of General Psychiatry*. 1978; 35:837–844. [PubMed: 678037]
- French CC, Richards A, Scholfield EJ. Hypomania, anxiety and the emotional Stroop. *British Journal of Clinical Psychology*. 1996a; 35:617–626. [PubMed: 8955547]
- French CC, Richards A, Scholfield EJC. Hypomania, anxiety and the emotional Stroop. *British Journal of Clinical Psychology*. 1996b; 35:617–626. [PubMed: 8955547]
- Giles DE, Rush AJ, Roffwarg HP. Sleep parameters in bipolar I, bipolar II, and unipolar depressions. *Biological Psychiatry*. 1986; 21(13):1340–1343. [PubMed: 3756280]
- Goldberg JF, Gerstein RK, Wenze SJ, Welker TM, Beck AT. Dysfunctional attitudes and cognitive schemas in bipolar manic and unipolar depressed outpatients: implications for cognitively based psychotherapeutics. *The Journal of Nervous and Mental Disease*. 2008; 196(3):207–210. [PubMed: 18340255]
- Goodwin, FK.; Jamison, KR. *Manic-Depressive Illness: Bipolar Disorder and Recurrent Depression*. second. Oxford University Press; New York: 2007.
- Gotlib IH, Joormann J. Cognition and depression: current status and future directions. *Annual Review of Clinical Psychology*. 2010; 6:285–312.
- Gray, JA. Three fundamental emotion systems. In: Ekman, P.; Davidson, RJ., editors. *The Nature of Emotion: Fundamental Questions*. Oxford University Press; New York: 1994. p. 243-247.
- Greenberg MS, Alloy LB. Depression versus anxiety: processing of self- and other-referent information. *Cognition & Emotion*. 1989; 3(3):207–223.
- Haaga DA, Dyck MJ, Ernst D. Empirical status of cognitive theory of depression. *Psychological Bulletin*. 1991; 110:215–236. [PubMed: 1946867]
- Hill CV, Oei TPS, Hill MA. An empirical investigation of the specificity and sensitivity of the Automatic Thoughts Questionnaire and Dysfunctional Attitudes Scale. *Journal of Psychopathology and Behavioral Assessment*. 1989; 11(4):291–311.
- Hollon SD, Kendall PC. Cognitive self-statements in depression: development of an automatic thoughts questionnaire. *Cognitive Therapy and Research*. 1980; 4(4):383–395.
- Hollon SD, Kendall PC, Lumry A. Specificity of depressotypic cognitions in clinical depression. *Journal of Abnormal Psychology*. 1986; 95(1):52–59. [PubMed: 3700847]
- Hyman SE. Goals for research on bipolar disorder: the view from NIMH. *Biological Psychiatry*. 2000; 48(6):436–441. [PubMed: 11018216]
- Johnson SL. Mania and dysregulation in goal pursuit: a review. *Clinical Psychology Review*. 2005; 25(2):241–262. [PubMed: 15642648]
- Johnson SL, Fingerhut R. Negative cognitions predict the course of bipolar depression, not mania. *Journal of Cognitive Psychotherapy*. 2004; 18(2):149–162.
- Johnson SL, Sandrow D, Meyer B, Winters R, Miller I, Solomon D, Keitner G. Increases in manic symptoms after life events involving goal attainment. *Journal of Abnormal Psychology*. 2000; 109:721–727. [PubMed: 11195996]
- Jones L, Scott J, Haque S, Gordon-Smith K, Heron J, Caesar S, Cooper C, et al. Cognitive style in bipolar disorder. *The British Journal of Psychiatry*. 2005; 187:431–437. [PubMed: 16260818]

- Judd LL, Akiskal HS, Schettler PJ, Coryell W, Endicott J, Maser JD, Solomon DA, et al. A prospective investigation of the natural history of the long-term weekly symptomatic status of bipolar II disorder. *Archives of General Psychiatry*. 2003a; 60(3):261–269. [PubMed: 12622659]
- Judd LL, Akiskal HS, Schettler PJ, Coryell W, Maser J, Rice JA, Solomon DA, et al. The comparative clinical phenotype and long term longitudinal episode course of bipolar I and II: a clinical spectrum or distinct disorders? *Journal of Affective Disorders*. 2003b; 73:19–32. [PubMed: 12507734]
- Judd LL, Akiskal HS, Schettler PJ, Endicott J, Maser J, Solomon DA, Leon AC, et al. The long-term natural history of the weekly symptomatic status of bipolar I disorder. *Archives of General Psychiatry*. 2002; 59(6):530–537. [PubMed: 12044195]
- Just N, Alloy LB. The response styles theory of depression: tests and an extension of the theory. *Journal of Abnormal Psychology*. 1997; 106(2):221–229. [PubMed: 9131842]
- Kessler RC, Akiskal HS, Ames M, Birnbaum H, Greenberg P, Hirschfeld RM, Jin R, et al. Prevalence and effects of mood disorders on work performance in a nationally representative sample of U.S. workers. *The American Journal of Psychiatry*. 2006; 163:1561–1568. [PubMed: 16946181]
- Kieseppä T, Tuulio-Henriksson A, Haukka J, Van Erp T, Glahn D, Cannon TD, Partonen T, et al. Memory and verbal learning functions in twins with bipolar-I disorder, and the role of information-processing speed. *Psychological Medicine*. 2005; 35(2):205–215. [PubMed: 15841678]
- Lam D, Wright K, Smith N. Dysfunctional assumptions in bipolar disorder. *Journal of Affective Disorders*. 2004; 79(1-3):193–199. [PubMed: 15023494]
- Lex C, Meyer TD, Marquart B, Thau K. No strong evidence for abnormal levels of dysfunctional attitudes, automatic thoughts, and emotional information-processing biases in remitted bipolar I affective disorder. *Psychology and Psychotherapy*. 2008a; 81:1–13. [PubMed: 17983484]
- Lex C, Meyer TD, Marquart B, Thau K. No strong evidence for abnormal levels of dysfunctional attitudes, automatic thoughts, and emotional information-processing biases in remitted bipolar I affective disorder. *Psychology and Psychotherapy*. 2008b; 81:1–13. [PubMed: 17983484]
- Lim S, Kim J. Cognitive processing of emotional information in depression, panic, and somatoform disorder. *Journal of Abnormal Psychology*. 2005; 114:50–61. [PubMed: 15709812]
- Lyon HM, Startup M, Bentall RP. Social cognition and the manic defense: attributions, selective attention, and self-schema in bipolar affective disorder. *Journal of Abnormal Psychology*. 1999; 108:273–282. [PubMed: 10369037]
- MacKinnon DP, Lockwood CM, Williams J. Confidence limits for the indirect effect: Distribution of the product and resampling methods. *Multivariate Behavioral Research*. 2010; 39:99–128. [PubMed: 20157642]
- Murphy FC, Sahakian BJ, Rubinsztein JS, Michael A, Rogers RD, Robbins TW, Paykel ES. Emotional bias and inhibitory control processes in mania and depression. *Psychological Medicine*. 1999; 29:1307–1321. [PubMed: 10616937]
- Neale, JM. Defensive functions of manic episodes. In: Oltmanns, TF.; Maher, BA., editors. *Delusional Beliefs: Wiley Series on Personality Processes*. John Wiley & Sons; Oxford: 1988. p. 138-156.
- Pardoen D, Bauwens F, Tracy A, Martin F, Mendlewicz J. Self-esteem in recovered bipolar and unipolar out-patients. *British Journal of Psychiatry*. 1993; 163:755–762. [PubMed: 8306117]
- Preacher KJ, Hayes AF. Asymptotic and resampling strategies for assessing and comparing indirect effects in multiple mediator models. *Behavior Research Methods*. 2008; 40:879–891. [PubMed: 18697684]
- Preacher KJ, Hayes AF. SPSS and SAS procedures for estimating indirect effects in simple mediation models. *Behavior Research Methods*. 2004; 36:717–731.
- Pyle A, Mansell W. Cognitive biases in hypomanic personality: preliminary findings indicating the relevance of self-versus-other encoding and high-versus-low levels of activation. *Behavioural and Cognitive Psychotherapy*. 2010; 38(4):459–472. [PubMed: 20519068]
- Reilly-Harrington NA, Alloy LB, Fresco DM, Whitehouse WG. Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. *Journal of Abnormal Psychology*. 1999; 108(4):567–578. [PubMed: 10609421]

- Robinson LJ, Thompson JM, Gallagher P, Goswami U, Young AH, Ferrier IN, Moore PB. A meta-analysis of cognitive deficits in euthymic patients with bipolar disorder. *Journal of Affective Disorders*. 2006; 93(1-3):105–115. [PubMed: 16677713]
- Rosenfarb IS, Becker J, Khan A, Mintz J. Dependency and self-criticism in bipolar and unipolar depressed women. *British Journal of Clinical Psychology*. 1998; 37:409–414. [PubMed: 9856294]
- Rude SS, Wenzlaff RM, Gibbs B, Vane J, Whitney T. Negative processing biases predict subsequent depressive symptoms. *Cognition & Emotion*. 2002; 16(3):423–440.
- Scott J, Pope M. Cognitive styles in individuals with bipolar disorders. *Psychological Medicine*. 2003; 33(6):1081–1088. [PubMed: 12946092]
- Scott J, Stanton B, Garland A, Ferrier IN. Cognitive vulnerability in patients with bipolar disorder. *Psychological Medicine*. 2000; 30(2):467–472. [PubMed: 10824667]
- Spitzer RL, Endicott J, Robins E. Research diagnostic criteria: rationale and reliability. *Archives of General Psychiatry*. 1978; 35(6):773–782. [PubMed: 655775]
- Urosevi S, Abramson LY, Harmon-Jones E, Alloy LB. Dysregulation of the behavioral approach system (BAS) in bipolar spectrum disorders: review of theory and evidence. *Clinical Psychology Review*. 2008; 28:1188–1205. [PubMed: 18565633]
- Van der Gucht E, Morriss R, Lancaster G, Kinderman P, Bentall RP. Psychological processes in bipolar affective disorder: negative cognitive style and reward processing. *British Journal of Psychiatry*. 2009; 194(2):146–151. [PubMed: 19182176]
- Weissman, AN.; Beck, AT. Proceedings from the Annual Meeting of the American Educational Research Association: Development and Validation of the Dysfunctional Attitude Scale: A Preliminary Investigation; Toronto, Canada. 1978.
- Winters KC, Neale JM. Mania and low self-esteem. *Journal of Abnormal Psychology*. 1985; 94(3): 282–290. [PubMed: 4031225]
- Zalla T, Joyce C, Szöke A, Schürhoff F, Pillon B, Komano O, et al. Executive dysfunctions as potential markers of familial vulnerability to bipolar disorder and schizophrenia. *Psychiatry Research*. 2003; 121:207–217. [PubMed: 14675740]

Table 1

Demographic and clinical characteristics of the sample.

	Bipolar spectrum	Healthy controls	<i>T</i>	
BDI	10.70 (8.10)	2.90 (3.16)	6.1**65	*
HMI	18.43 (11.69)	15.17 (6.87)	1.69	†
Age	19.76 (1.89)	19.53 (1.57)	0.718	ns
% male	36.36%	39.66%	0.374	ns
Ethnicity			χ^2	
White	50.00%	44.80%	3.982	ns
Black	24.20%	32.80%		
Hispanic	3.00%	0.00%		
Asian	3.00%	6.90%		
Biracial	19.70%	15.50%		

Note: Standard deviations in parentheses. BDI = Beck depressive inventory; HMI = Halberstadt Mania Inventory. ns $p > .10$.

† $p < .10$.

* $p < .05$.

Table 2

Findings from univariate ANOVAs of SRIP variables.

	Bipolar spectrum		Controls	
	<i>M</i>	(<i>SD</i>)	<i>M</i>	(<i>SD</i>)
# "Like me" Endorsements				
Positive	8.66	3.07	***	10.93 2.27
Negative	3.58	2.60	***	1.72 1.96
Response times				
DR	4136.99	3263.74	*	3076.32 1598.40
DI	3319.26	1466.28	†	3904.76 2356.36
Behavioral predictions				
Positive	72.27	13.22	*	78.39 10.62
Negative	40.66	18.84	***	23.70 14.96
Recall				
Positive	6.92	2.15	***	8.84 1.79
Negative	2.98	1.66	***	1.42 1.37

Note. DR=depression relevant; DI=depression irrelevant.

† $p < .10$.

* $p < .05$.

*** $p < .001$.