



Preliminary communication

Boiling at a different degree: An investigation of trait and state anger in remitted bipolar I disorder

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ABSTRACT

Background: Elevated anger is a prominent clinical feature of bipolar disorder (BD). However, it is unclear whether this feature is characterized by elevated trait anger (i.e., how much anger one experiences in general) and/or state anger (i.e., how much anger one experiences when provoked), how stable anger elevations are (i.e., whether they appear during remission), and whether they have prognostic significance.

Methods: The present study assessed trait anger as well as state anger during a neutral baseline and a validated laboratory anger provocation among adults with remitted bipolar I disorder (BD; $n=27$) and healthy controls (CTL; $n=29$). To examine prognostic significance, we assessed manic and depressive symptom severity one year later in a subsample of BD participants ($n=18$).

Results: Results revealed greater trait anger as well as state anger experience at baseline for the BD compared to the CTL group. No group differences emerged in anger during the provocation. Anger did not predict symptom severity, but greater positive emotion during the provocation predicted mania (but not depression) symptom severity.

Limitations: We utilized a relatively high functioning sample of remitted BD patients. Future studies should include BD patients with current mood episodes and more diverse functioning, to ensure generalizability of our results.

Conclusions: These findings suggest that BD is characterized by elevated trait and baseline state anger, but not greater responding to anger provocation. Persistently elevated anger may represent a marker of BD, and context-inappropriate positive emotion experience during anger provocation may constitute a vulnerability factor for mania severity.

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1. Introduction

Recent theoretical models posit that elevated anger represents a core clinical feature of bipolar disorder (BD) that persists across the lifespan (Lara et al., 2006; Alloy & Abramson, 2010). Elevated anger is associated with harmful consequences in BD, including increased rates of violent crime and suicidality (Oquendo et al., 2000; Fazel et al., 2010). Despite the centrality of anger in BD and its destructive consequences, our current understanding of the nature of anger in BD is limited. Specifically, it is currently unclear whether individuals with BD experience higher levels of anger at the baseline state level (regardless of environmental provocation), greater apparent trait-level anger in daily life due to more frequent environmental triggers

(e.g., higher levels of relationship or occupational instability), greater reactivity to anger provocation, or some combination of these elements. This question is of critical importance if effective interventions are to be designed to ameliorate the destructive effects of heightened anger in BD. For example, if the primary characteristic of heightened anger in BD is elevated reactivity to immediate, anger-provoking environmental events, psychosocial interventions aimed at reducing such events may be best suited for treatment. Alternatively, if baseline levels of anger are chronically elevated in BD, interventions aimed at reducing chronic anger and aggressive behavior by increasing self-regulation in daily life may represent superior first-line treatments.

To address this question, experimental studies are needed which examine both trait and state anger responses in participants with BD in carefully controlled laboratory settings, and examine the prognostic significance of anger and responding to angering events in this population. The present research employed such

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methods to elucidate the nature and prognostic significance of trait and state anger in BD.

Significant literature supports anger as a central feature of BD (American Psychiatric Association, 2013). Anger has been associated with heightened sensitivity of the Behavioral Approach System (BAS) (Harmon-Jones & Allen, 1998), a central process implicated in the etiology of BD (Urosevic et al., 2008). Rating scales used by clinicians to evaluate the presence of manic episodes in BD include descriptions such as “markedly impatient or irritable” as well as “episodes of anger or annoyance” (Young et al., 1978; Bech et al., 1979). Moreover, documented symptoms of depression in BD include “episodes of sudden, intense, and situationally inappropriate anger”, suggesting that this symptom may not be restricted to manic episodes (Perlis et al., 2004). One study found higher rates of hostile personality characteristics in a remitted BD group compared to unaffected relatives (Savitz et al., 2008), suggesting that heightened anger persists even when other symptoms of the disorder remit. Another reported that among euthymic individuals with BD, a larger number of previous episodes of depression and mania was associated with prolonged recovery (taking longer to return to baseline) after frustration (Wright et al., 2008).

These findings have been instrumental in establishing the clinical centrality of anger to the diagnosis of BD, and are beginning to elucidate the nature of this symptom. However, they do not clarify whether individuals with BD generally experience more anger in their daily lives, are more readily angered when provoked, or both. Clarifying this necessitates an approach that appreciates the nuances of measuring trait and state anger. Importantly, trait reports and state experiences of emotion can be dissociated from one another (Robinson & Clore, 2002). Trait self-report measures confound characteristics of the person with their life circumstances. For instance, a person could report elevated trait anger because they experience more anger (i.e., they are an angry person) or because they are in a dismissing relationship (i.e., frequent occurrence of anger-eliciting events). To account for potential and confounds of trait assessments with life circumstances, research is needed that assesses anger at the trait level alongside assessments of state anger under carefully controlled laboratory settings.

Finally, the clinical prognostic significance of state and trait anger are not well understood. No existing work has examined whether anger experience, or affective responses to angering events, might predict illness course among those with BD. This work, undertaken in the current study, may provide potential risk markers for future symptom changes, and inform the development of more effective treatments.

1.1. The present investigation

The present study aimed to address three critical empirical gaps in our understanding of anger in BD. First, we aimed to provide support for preliminary literature that suggests individuals with BD report greater trait anger compared to controls, and test whether this would translate to heightened state anger experience during a carefully controlled, neutral laboratory baseline. This approach allowed us to ensure that reports of heightened trait anger are not a function of artifacts related to trait reports. To this end, participants completed the State-Trait Anger Expression Inventory (STAXI-2; Spielberger, 1999) and watched a 2-min, emotionally neutral film clip followed by reporting affect experienced during the clip. Second, we aimed to test whether participants with BD compared to controls would experience elevated anger in response to a carefully controlled laboratory anger provocation that has been validated among healthy individuals (Mauss et al., 2006; Mauss et al., 2007a). Third, we aimed to understand the predictive power of anger and responses to provocation for symptom course by examining the extent to which

affect predicted mood symptoms in a subsample of the BD group one year later.

Based on existing literature (Savitz et al., 2008), we hypothesized that BD participants would be characterized by heightened trait anger, as indexed by higher scores on the Angry Temperament subscale of the State-Trait Anger Expression Inventory (STAXI-2; Spielberger, 1999), compared to the control group (Hypothesis 1). Based on findings demonstrating higher levels of hostile personality traits among individuals with BD (Savitz, 2008), and heightened reactivity to an anger-evoking event among individuals at risk for BD (Harmon-Jones et al., 2002), we hypothesized that the BD group would exhibit heightened self-reported state anger compared to the control group during the neutral baseline (Hypothesis 2a) and during anger provocation (Hypothesis 2b). Our third exploratory aim examined whether trait anger, or state affect at baseline or during provocation would predict manic or depressive symptom severity at a one-year follow-up assessment among BD participants, given that existing work implicates heightened anger in the course of mania (Johnson, 2005) as well as bipolar depression (Perlis et al., 2004) (Exploratory question 3).

2. Method

2.1. Participants

Participants were 27 individuals diagnosed with BD type I, currently remitted (neither manic nor depressed; remission duration = 15.84 months ($SD = 19.13$), and 29 healthy control participants who did not meet current or past criteria for any DSM-IV-TR Axis I disorder. Participants with remitted BD were chosen to minimize the potential confound of mood on our results, and to understand whether elevated anger would be a stable marker of BD. Participants were recruited using online advertisements and flyers posted in New Haven, CT and surrounding communities. Exclusion criteria were history of severe head trauma, stroke, neurological disease, severe medical illness, and alcohol or substance abuse in the past six months. Participant characteristics are listed in Table 1.

For BD participants, the average age of onset was 16.61 years ($SD = 7.00$) and average illness duration was 14.37 years ($SD = 10.07$). The lifetime average number of manic/hypomanic episodes was 12.02 ($SD = 21.82$), and the lifetime average number of major depressive episodes was 12.87 ($SD = 22.62$). The average number of psychotropic medications for the BD group was 2.07 ($SD = 1.54$), and included anticonvulsants ($n = 12$), lithium ($n = 7$), neuroleptics ($n = 11$), anxiolytics ($n = 7$), stimulants ($n = 3$), antidepressants ($n = 3$), and sedative-hypnotics ($n = 1$). BD participants were not excluded on the basis of comorbid disorders (aside from current substance or alcohol use disorders) given that BD is commonly comorbid with other disorders (Kessler et al., 2005), though we verified that BD was the primary, or most severe, diagnosis. BD participants had an average of 0.56 ($SD = 0.97$) current Axis I comorbidities including panic disorder ($n = 1$), agoraphobia ($n = 1$), social phobia ($n = 3$), specific phobia ($n = 3$), obsessive-compulsive disorder ($n = 2$), generalized anxiety disorder ($n = 2$), body dysmorphic disorder ($n = 1$), hypochondriasis ($n = 1$), and bulimia ($n = 1$). The CTL group did not meet criteria for any current or lifetime Axis I disorders assessed.

2.2. Assessments

2.2.1. Diagnostic evaluation

All Axis I diagnoses were confirmed using the Structured Clinical Interview for DSM-IV (SCID-IV; First et al., 2007). Approximately one-fourth ($n = 13$; 23.21%) of videotaped interviews were rated by

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