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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.

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 heighted 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

Table 1
Demographic and clinical characteristics.

	BD ($n=27$)	CTL ($n=29$)	Statistic	Effect size	Levene's test	Degrees of freedom
Demographic						
Age (Yrs)	30.89 (8.84)	31.89 (8.97)	$F=0.18$	$\eta_p^2<0.01$	$F=0.12$	1,53
Female (%)	66.67%	62.07%	$\chi^2=0.13$	$V=0.05$		
Caucasian (%)	88.89%	89.66%	$\chi^2=0.01$	$V=0.01$		
Education (Yrs)	15.06 (2.29)	16.05 (2.38)	$F=2.51$	$\eta_p^2=0.05$	$F=0.16$	1,53
Employed (%)	51.85%	68.97%	$\chi^2=1.72$	$V=0.18$		
Living Alone (%)	22.22%	13.79%	$\chi^2=0.68$	$V=0.11$		
Income > $50,000	25.93%	48.28%	$\chi^2=2.98$	$V=0.22$		
Married (%)	29.63%	17.24%	$\chi^2=1.20$	$V=0.15$		
Clinical						
YMRS	1.91 (2.01)	1.21 (1.76)	$F=1.93$	$\eta_p^2=0.04$	$F=0.26$	1,54
IDS-C	5.67 (3.63)	2.28 (2.33)	$F=17.58^*$	$\eta_p^2=0.25$	$F=9.74^*$	1,54
GAF	75.30 (6.06)	88.07 (3.08)	$F=101.01^*$	$\eta_p^2=0.65$	$F=13.96^*$	1,54
Working memory	10.44 (3.07)	12.34 (3.21)	$F=5.11^*$	$\eta_p^2=0.09$	$F=0.23$	1,54
Intellectual functioning	31.67 (4.20)	33.00 (2.84)	$F=1.96$	$\eta_p^2=0.04$	$F=8.50^*$	1,54

Note: BD=Bipolar disorder group; CTL=Healthy control group; YMRS=Young Mania Rating Scale; IDS-C=Inventory to Diagnose Depression. Income= Annual Household Income. GAF=Global assessment of functioning; Working Memory=Wechsler Adult Intelligence Inventory, 4th edition Letter-Number Sequencing Task. Mean values are displayed with standard deviations in parentheses where applicable.

* $p<0.05$.

an independent reviewer. Ratings matched 100% of primary diagnoses, and reliability was high across all diagnoses ($\kappa_{mean}=1.00$).

2.2.2. Mood symptoms

Current symptoms of mania were measured using the Young Mania Rating Scale (YMRS; Young et al., 1978). The YMRS is an 11-item, clinician-rated measure of mania symptoms with scores ranging from 0 to 60. Scores ≥ 7 represent clinically significant mania. Current symptoms of depression were measured using the Inventory of Depressive Symptomatology (IDS-C; Rush et al., 1996). The IDS-C is a 30-item, clinician-rated measure of current depressive symptoms with scores ranging from 0 to 84. Scores ≥ 11 represent clinically significant depression. Intra-class correlations (ICC; Shrout & Fleiss, 1979) for absolute agreement between the original interviewer and an independent rater for approximately one fifth of study participants ($n=12$; 21.43%) were strong for both the YMRS (0.98) and IDS-C (0.96). Current remitted mood status for the BD group was verified according to SCID-IV mood module criteria for the past month and cutoff scores on the YMRS (≤ 7), and IDS-C (≤ 11) for the past week. Controls also scored below these cutoffs.

2.2.3. Mood symptoms at follow-up

The Altman Self-Rating Mania Questionnaire (ASRM; Altman et al., 1997), employed to assess mania symptoms at follow-up, is a five-item self-report inventory with scores ranging from 0 to 20 ($M=4.06$ $SD=5.01$). Items probe participants' mood over the past week, indexing cheerfulness, self-confidence, need for sleep, talkativeness, and activity level. Scores ≥ 14 represent clinically significant mania. Internal consistency was strong in the present study ($\alpha=0.91$). The Beck Depression Inventory-Short Form (BDI-SF; Beck & Beck, 1972), used to assess depression at follow-up, is a 13-item self-report inventory with scores ranging from 0 to 39 ($M=4.05$, $SD=4.57$). Cutoff scores for mild, moderate, and severe depression using this measure are 5, 8, and 16, respectively. Items probe symptoms such as depressed mood, hopelessness, and suicidal ideation, over the past week. Internal consistency was strong in the present study ($\alpha=0.82$).

2.2.4. Global functioning

The Global Assessment of Functioning Scale (GAF; Luborsky, 1962) was used to assess global functioning in the past week. The GAF assesses psychosocial functioning on a scale from 1 (lowest) to 100 (highest). ICC for agreement between the original interviewer and an independent rater for one-fifth of study participants ($n=11$; 19.64%) was high (ICC=0.94).

2.2.5. Working memory

Given that the anger provocation involved a working memory component (see Procedure), we measured working memory as a potential confound using the letter-number sequencing subtest of the Wechsler Adult Intelligence Scale-IV (WAIS-IV; Pearson, 2008). Participants were read aloud a series of increasingly long lists of randomly ordered numerical digits and alphabetical letters. Then, participants were asked to verbally repeat all numbers (in numerical order) first, followed by all letters (in alphabetical order). Raw scores (ranging from 0 to 21) were calculated as the total number of trials correct, from which WAIS-IV age-normed scaled scores (ranging from 1 to 9) were computed for final analyses.

2.2.6. General intellectual functioning

The vocabulary subtest of the Shipley Institute of Living Scale (SILS; Zachary, 1986) was included as a conventional measure of general intellectual functioning, given the cognitive load imposed by the anger provocation. Items consisted of 40 multiple-choice questions in which participants were asked to select one of four words closest in meaning to the target word. Scores ranged from 0 to 40.

2.2.7. Trait anger

Trait anger was measured using the Angry Temperament Scale (e.g., "I am a hotheaded person") from the State-Trait Anger Expression Inventory (STAXI-2; Spielberger, 1999). This subscale was chosen to capture chronic, trait-level anger experience, rather than state-dependent anger that may fluctuate as a function of mood state or external provocation. The STAXI-2 is a 57-item scale measured on a 1 (*almost never*) to 4 (*almost always*) scale. In our sample, internal consistency was good ($\alpha=0.78$) for this subscale.

2.2.8. Laboratory measures of emotional experience

Self-reported positive affect (PA) and negative affect (NA) were assessed using the modified Differential Emotions Scale (mDES; Fredrickson et al., 2003). The mDES consists of 18 individual positive and negative emotion terms rated on a 1 (*not at all*) to 5 (*extremely*) scale. We examined the anger item as our target emotion. Remaining individual items were averaged to create PA

(amusement, awe, contentment, joy, gratitude, hope, love, pride, sympathy, interest; $\alpha_{mean}=0.93$) and NA composites (fear, disgust, embarrassment, guilt, sadness, shame, contempt; $\alpha_{mean}=0.60$).[1]

2.3. Procedure

The present study, which was part of a larger study at Yale University, consisted of two parts: an initial laboratory session (Part 1) and one-year follow-up assessment (Part 2). All procedures were approved by the Yale University Institutional Review Board and participants provided written and verbal consent before participation.

2.3.1. Part 1: Laboratory anger provocation paradigm

The laboratory experiment consisted of three components. First, participants underwent a diagnostic interview to determine eligibility using the SCID-IV (First et al., 2007). Immediately afterward, assessments of working memory and general intellectual functioning were obtained. Second, questionnaire measures were conducted and participants completed tasks not relevant to the present study. Approximately one week later ($M=8.05$, $SD=3.61$ days), participants returned to complete the anger provocation task. After reestablishing consent, current symptoms were reassessed to ensure remitted status using the YMRS and IDS-C. Participants were escorted to a $6' \times 7'$ room and seated in front of a 26″ computer monitor. After completing unrelated tasks, participants were oriented to the anger provocation task. Computerized software (MediaLab v2008; New York, NY) was used to guide participants through the experiment, present instructions, and collect questionnaire information.

The anger provocation consisted of a well-validated task that has been shown to provoke anger in healthy adults (Mauss et al., 2006, 2007a, 2007b). A pre-recorded voice, was transmitted over an intercom system to the experimental room. Participants were told that the voice belonged to an experimenter in the adjacent room. Over the intercom, participants were informed that they would be participating in a cognitive performance task. To establish a baseline, participants sat quietly while 60 s of physiological data was acquired (see Supplementary material). After, they watched an emotionally neutral film with scenes from *Denali* (110 s) and reported emotion experience using the mDES. Next, participants were asked to count backwards in steps of 7 or 13 from a large number (e.g., 13,279) as quickly as possible. After 60 s, participants were interrupted by the recorded voice of the 'experimenter'. This was repeated three times, starting from a different number each time. Between repetitions, the pre-recorded voice told participants that they were "producing artifacts" and that they had to "speak more loudly." The voice took an increasingly condescending and impatient tone, ultimately communicating that data would be unusable due to poor study compliance ("let's just stop here"). Spontaneous clarifications and questions from participants were answered using pre-recorded prompts. Next, participants self-reported their emotion experience using the mDES. To ensure participants ended the session in a calm state, they were shown a calming film depicting nature scenes from *Planet Earth* (210 s). Finally, experimenters gave a thorough debriefing.

2.3.2. Part 2: Longitudinal symptom follow-up

A follow-up assessment was conducted approximately one year after the anger provocation ($M=366.00$ days, $SD=17.65$). In this assessment, participants completed measures of mood symptom severity, including self-reported manic and depressive symptom severity using an online Qualtrics™ survey. Following previous studies (Gilbert et al., 2013), remotely completed online self-report measures were utilized to maximize retention and power for the follow-up assessment.

3. Results

3.1. Demographic and clinical characteristics

As listed in Table 1, BD and CTL participants did not significantly differ with respect to age, gender, ethnicity, education, annual household income, or marital status. The BD group scored lower on global functioning (GAF) and the working memory measure than the CTL group. Although both groups scored below mania (YMRS scores ≤ 7) and depression (IDS-C scores ≤ 11) cutoffs, BD participants scored higher than CTL participants on the IDS-C. Groups did not differ in general intellectual functioning.

3.2. Manipulation check: examining the effectiveness of the anger provocation

To ensure that our results mirrored those of prior work using the same task, we conducted repeated-measures ANOVAs examining Condition (Baseline, Provocation) main effects for the state emotion response variables (Anger, PA, NA). Prior to any analysis, skewness and kurtosis indices were examined for all three dependent variables. All three were positively skewed, and NA and Anger were leptokurtic. Thus, attempts were made to normalize the data using an inverse transformation for these variables. The transformed variables were used in all subsequent analyses, though non-transformed mean values are presented in Table 2 for ease of interpretation. Significant main effects of Condition emerged for all three, with Anger, $F(1, 55)=72.70$, $p<0.001$, $\eta_p^2=0.57$, and NA,$F(1, 55)=52.85$, $p<0.001$, $\eta_p^2=0.49$, increasing

Table 2
Means and standard deviations for emotion response variables across all participants.

	BD ($n=27$)	CTL ($n=29$)	Statistic	Effect size	Degrees of freedom
Baseline					
Anger	1.37 (0.63)	1.10 (0.56)	$F=4.11$*	$\eta_p^2=0.07$	1,52
NA	1.05 (0.11)	1.04 (0.15)	$F=1.13$	$\eta_p^2=0.02$	1,52
PA	2.36 (0.76)	2.70 (0.94)	$F=0.65$	$\eta_p^2=0.00$	1,52
Anger provocation					
Anger	2.59 (1.22)	2.55 (1.38)	$F=0.28$	$\eta_p^2=0.01$	1,52
NA	1.43 (0.36)	1.35 (0.52)	$F=0.56$	$\eta_p^2=0.01$	1,52
PA	1.79 (0.81)	1.83 (1.03)	$F=0.24$	$\eta_p^2=0.01$	1,52
Trait anger					
Experience	17.46 (4.62)	15.21 (3.64)	$F=4.91$*	$\eta_p^2=0.08$	1,53

Note: BD=Bipolar disorder group; CTL=Healthy control group; M=Mean, SD=Standard Deviation; PA=Positive effect, NA=Negative effect; Mean values are displayed with standard deviations in parentheses where applicable. *F* Values for task variables are reported controlling for working memory and IDS-C scores.

* $p<0.05$.

[1] We examined reliability estimates for the NA composite separately for the BD and CTL groups. Alpha values were comparable across both groups at baseline (BD=0.51, CTL=0.69; $Z=-1.03$, $p=0.30$) and during the task (BD=0.65, CTL=0.83, $Z=-1.44$, $p=0.15$).

from baseline (Anger: $M=1.23$, $SD=0.60$; NA: $M=1.05$, $SD=0.13$) to the provocation (Anger: $M=2.57$, $SD=1.29$; NA: $M=1.39$, $SD=0.45$). PA decreased from baseline ($M=2.54$, $SD=0.87$) to the provocation ($M=1.81$, $SD=0.93$), $F(1, 55)=55.76$, $p<0.001$, $\eta_p^2=0.50$.

3.3. Overview of main analyses

To address Hypothesis 1, a one-way analysis of variance (ANOVA) was run comparing both groups (BD, CTL) on the Angry Temperament variable from the STAXI-2 (Spielberger, 1999). This variable was positively skewed, and so an attempt was made to normalize the data using an inverse transformation, though non-transformed mean values are presented in Table 2 for ease of interpretation.

To address Hypothesis 2, three separate 2 (Condition: Baseline, Provocation) x 2 (Group: BD, CTL) repeated-measures ANOVAs were conducted for each individual emotion response variable, including anger, positive affect (PA), negative affect (NA). Additionally, a one-way ANOVA examining each time point (Baseline, Provocation) separately was conducted for each emotion response variable, given our a priori interest in examining group differences at baseline and during the provocation separately. Because groups differed in subsyndromal depressive symptoms, IDS-C was included as a covariate. Given that groups also differed in working memory, which was an important component of performance in the anger provocation, we also included these scores as covariates (though note that results remained generally consistent without covariates[2]). A Greenhouse–Geisser correction was used when assumptions for sphericity were not met and adjusted F and p values are reported. Effect sizes for significant results are reported as partial eta squared (η_p^2). All reported p values are two-tailed. Means and standard deviations are presented in Table 2.

Hypothesis 1: Group differences in trait anger

Results revealed a main effect of group, $F(1, 53)=4.91$, $p=0.03$, $\eta_p^2=0.09$, indicating that as predicted, the BD group reported greater trait anger than the CTL group.

Hypothesis 2: Group differences in state anger response

3.3.1. Emotional experience

For state anger, there was no main effect of Group and no significant Group x Condition interaction ($ps>0.10$). Given our strong a priori rationale for examining group differences in state anger separately at baseline (Hypothesis 2a) and during the anger provocation (Hypothesis 2b), we performed two separate one-way ANOVAs examining group differences in state anger at baseline and during the anger provocation. These analyses revealed that as predicted, the BD group ($M=1.37$, $SD=0.63$) reported greater anger than the CTL group ($M=1.10$, $SD=0.56$) at baseline, $F(1, 52)=4.11$, $p<0.05$, $\eta_p^2=0.07$. However, contrary to predictions, state anger in the BD group was not significantly different from the CTL group during the provocation ($p>0.60$). For PA and NA, there were no significant main effects of Group or significant Group x Condition interactions, and follow-up analyses revealed no group differences at baseline or during the provocation ($ps>0.10$).

[2] Significance levels for all analyses remained significant without covariates, except for two results. For PEP, the main effect of condition was reduced to a trend, $F(1,48)=3.39$, $p<0.07$,$\eta_p^2=0.07$. For HR, the main effect of condition became significant, $F(1,52)=154.92$, $p<0.01$, $\eta_p^2=0.75$, reflecting increased heart rate during the anger provocation ($M=77.72$, $SD=10.15$) compared to baseline ($M=68.95$, $SD=11.19$).

3.4. Secondary analyses for Hypotheses 1 and 2

We examined the role of two potential confounds on our main findings for Hypotheses 1 and 2, including comorbid anxiety disorders and medication status. First, we examined the role of comorbid anxiety disorders because anxiety disorders have been associated with heightened levels of anger (Hawkins & Cougle, 2011), anxiety symptoms and disorders are common in BD (Freeman et al., 2002), and comorbid Axis I anxiety disorders were prevalent in our BD sample ($n=8$; 29.6%). Thus, we re-ran all analyses covarying for the presence (yes or no) of at least one comorbid Axis I anxiety disorder. All results remained comparable. Second, because some studies have found effects of antipsychotic medications on subjective emotional experience (e.g., Gerlach & Larsen, 1999), we also re-ran all analyses covarying for antipsychotic medication, dummy coded present ($n=10$) or absent ($n=17$), at the time of testing. All results remained comparable.

Hypothesis 3: Predicting mania and depression symptoms prospectively

Approximately two-thirds of the original sample of BD participants completed the one-year follow-up study ($n=18$; 66.67%). Those who completed the follow-up did not differ from those who did not on any demographic variables, mania symptoms, working memory, or intellectual functioning ($ps>0.05$). However, non-completers reported significantly more severe depression symptoms on the IDS-C ($M=8.78$) than completers ($M=4.11$) at the time of the initial experiment ($p=0.001$). Three separate linear regressions were conducted, using trait anger experience, as well as self-reported emotion (Anger, PA, NA) at baseline and during the provocation, to predict self-reported mania (ASRM) and depression (BDI-SF) symptom severity. Neither Anger nor NA significantly predicted ASRM scores ($ps>0.05$).However, higher PA significantly predicted higher ASRM scores one year later, $b=1.67$, $t(43)=2.77$, $p=0.008$. None of the predictor variables (Anger, PA, NA) significantly predicted depressive symptoms ($ps>0.20$).

4. Discussion

The present investigation generates several important insights that promise to advance our understanding of anger in BD. First, the success of the provocation in eliciting anger provides critically needed validation for an anger-elicitation paradigm appropriate for the BD population. Second, in support of Hypothesis 1, that BD participants would be characterized by heightened trait anger compared to the CTL group, and Hypothesis 2a, that they would also exhibit heightened state anger compared to the CTL group during a neutral baseline, results from our trait and baseline state-level anger assessments suggest chronically heightened anger in BD. Inconsistent with Hypothesis 2b, heightened anger was not observed in the BD group during the provocation. Finally, results from prospective analyses within the BD group indicated that increased positive emotion reported during the provocation was predictive of increased mania symptoms one year later. We discuss each of these contributions below, and stress how these results bear implications for future empirical work in BD and for developing interventions for reducing anger and associated outcomes in BD.

Our first finding provides direct evidence supporting the validity of an experimentally rigorous, yet clinically appropriate, anger-elicitation paradigm in a BD sample. Specifically, the provocation was associated with increased self-reported anger and negative effect, and decreased positive effect. These results are consistent with previous findings using this paradigm with an undergraduate sample (Mauss et al., 2006), and indicate successful

translation to a community sample of healthy adults and individuals with BD.

Our second domain of findings relates to group-related differences in more chronic, as opposed to reactive, indices of anger in the BD group. Specifically, increased trait-level and baseline state level anger observed in the BD group dovetails with prior literature documenting strong associations between BD and heightened levels of chronic anger and aggressive behavior, as well as self-reported trait anger (Fazel et al., 2010; Elbogen and Johnson, 2009; Lara et al., 2006). Also consistent with these findings is the observation that the BD group reported greater anger experience at baseline relative to the CTL group. Attesting to the robustness of this effect, results remained consistent when controlling for comorbid anxiety, antipsychotic medication, depressive symptoms, and working memory performance. The fact that these group differences emerged in a remitted sample suggests that heightened anger may represent a marker characteristic of individuals with BD, rather than a characteristic of a specific mood state. However, it is plausible that chronically heightened anger emerges as a result of triggering events or environmental characteristics (e.g., higher levels of stress), which may be more common in the lives of those with BD. This explanation would account for the relative strength of the group differences in our trait-level findings (which reflect anger experiences outside the laboratory) compared to the group differences during our laboratory baseline.

Inconsistent with our predictions, there were no group differences in self-reported anger during provocation. The absence of self-reported differences is consistent with research documenting an absence of heightened self-reported anger in response to provocation among individuals at risk for BD (Harmon-Jones et al., 2002). Taken together, these findings are consistent with the notion that heightened anger in BD may exist at a chronic level, persistent across contexts, rather than a more transient, reactive level. This is congruent with recent theoretical models positing positive and approach-oriented emotion persistence as a central feature of the disorder (Gruber, 2011a, 2011b), and has critical implications for the expansion of existing treatment approaches aimed at appropriate anger regulation and expression (e.g., Willner et al., 2002). Specifically, this finding may shift treatment emphasis from mitigating reactivity to specific provocative events to regulating baseline anger experience in everyday life.

Our third domain of findings suggests a prospective link between emotion-related responding during provocation and clinical outcomes at a one-year follow-up assessment. Increased positive emotion reported during the provocation significantly predicted mania symptoms one year later, suggesting that context-incongruent emotional responding is predictive of mania symptom severity prospectively. These results fortify existing literature demonstrating that context-insensitive affective responding may not only be a characteristic of mood disorders (Rottenberg et al., 2005; Gruber, 2011a; Gruber, 2011b), but may also predict mood symptoms prospectively (Gruber et al., 2009). The lack of depression- and anger-related prospective findings indicate that negative affective responding, when it is contextually appropriate (i.e., when provoked) may not be predictive of mood symptoms prospectively, and suggest that anger management programs for individuals with BD may benefit from a focus on regulating and reducing context-incongruent emotional reactivity.

Our findings should be interpreted carefully given study limitations. First, the present study focused on currently remitted BD patients to examine patterns of anger associated with BD, independent of any confounding effects of current mood state. An important next step is to extend these findings to manic and depressed mood phases of BD. Future studies should also include comparison subjects beyond healthy volunteers, including psychiatric groups matched on Axis I diagnoses commonly comorbid to BD as well as with documented anger-related difficulties (e.g., remitted major depressive disorder group, disorders of impulse control) to more carefully tease apart group differences directly attributable to a BD diagnosis. Second, given that the BD sample included in the present study was relatively high functioning, future studies should examine whether anger may differ among lower-functioning samples of individuals with BD. Third, self-reported anger during our provocation relied on a single item. Although this helped ensure that participants were unaware of the intentional anger provocation, it may have made group differences more difficult to detect and thus, future studies should aim to assess state-related anger responses using a validated measure. Fourth, although our sample size is common in experimental psychopathology research, replicating these findings in larger samples will enable more careful examination of anger response profiles among BD subtypes with differing comorbidities and medication profiles.

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Conflicts of interest

No conflict declared.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.jad.2014.06.044.

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