

Feeling Without Thinking? Anger Provocation Task Predicts Impaired Cognitive Performance in Bipolar Disorder but not Major Depression or Healthy Adults

Sunny J. Dutra¹ · Greg J. Siegle² · Elizabeth J. Reeves³ · Iris B. Mauss⁴ · June Gruber⁵

Published online: 14 November 2015

© Springer Science+Business Media New York 2015

Abstract Elevated anger and impairments in executive functioning are prominent features of bipolar disorder (BD). Given that anger has been found to interfere with some aspects of cognition in healthy individuals, it is possible that heightened anger could compromise cognitive processing even more substantially among individuals with BD. Despite the important clinical and psychosocial implications of such an effect, the precise consequences of anger for cognition in BD are not well understood. To address this, the present study employed a validated anger provocation task and examined its impact on performance of an arithmetic task, assessing both accuracy (number of correct responses) and task engagement (number of responses made) among adults with remitted bipolar I disorder (BD; $n = 27$), healthy non-psychiatric controls (CTL; $n = 29$), and a clinical control group of adults with remitted depression (MDD; $n = 29$). Results revealed that individuals with BD uniquely declined in the number of responses made across the task. In addition, self-reported anger was predictive of reduced task performance among

individuals with BD. These results suggest that elevated anger may add to existing executive impairments in BD, compromising these individuals' ability to remain engaged in cognitively demanding tasks in the context of anger.

Keywords Anger · Cognitive functioning · Working memory · Emotion · Bipolar disorder · Depression

Introduction

Bipolar disorder (BD) is a chronic and debilitating psychiatric disorder associated with profound functional impairment, including poor occupational and social functioning (Woods 2000; Zarate et al. 2000). Anger, one of the cardinal symptoms of BD, may further impair executive function (e.g., Lerner and Tiedens 2006), leading to a particularly compromised state and potentially interfering with performance of cognitively demanding daily life tasks. Here, we examine the effects that elevated anger may have on cognitive performance in this population, with an eye toward improved understanding of the factors that may contribute to psychosocial impairment in BD.

BD is characterized by elevated levels of positive emotions (Alloy and Abramson 2010; Gruber 2011). A growing body of empirical evidence suggests that BD is associated with a greater magnitude of positive emotional responses during anticipation of, and in response to positive and rewarding stimuli (Gruber 2011). For example, individuals at risk for developing BD, as well as those with a history of BD but currently in remission, report elevated positive emotion during anticipation of, and in response to pleasant and rewarding stimuli across laboratory and daily life settings (Johnson et al. 2007). This heightened positive emotion experience has been observed across multiple

✉ June Gruber
june.gruber@colorado.edu

¹ Department of Psychology, Yale University, New Haven, CT, USA

² University of Pittsburgh School of Medicine, Pittsburgh, PA, USA

³ Department of Counseling, Higher Education, and Special Education, University of Maryland, Bethesda, MD, USA

⁴ Department of Psychology, University of California, Berkeley, Berkeley, CA, USA

⁵ Department of Psychology and Neuroscience, University of Colorado Boulder, 345 UCB, Muenzinger D321C, Boulder, CO 80309-0345, USA

types of positively valenced stimuli, such as autobiographical memories and emotion-eliciting films (Gruber et al. 2008, 2009).

In addition to elevated positive emotionality, BD is characterized by heightened approach- and achievement-motivation, and persistent reward pursuit (Johnson 2005; Nusslock et al. 2008). Individuals at risk for BD report elevated levels of reward (joy) and achievement-focused (pride) positive emotions (Gruber and Johnson 2009), score high on measures of incentive sensitivity, and endorse ambitious goals involving fame, wealth, and political influence (Johnson and Carver 2006). In addition, those at high risk for BD endorse elevated positive affect in response to false success feedback compared to those at lower risk (Meyer and Baur 2009), and euthymic individuals with BD report elevated positive affect at the prospect of earning rewards in their daily lives (Meyer et al. 2001) compared to healthy controls. Consistent with these findings, Farmer et al. (2006) found that when euthymic individuals with BD and a healthy control group were given false success feedback on their performance of a Go-No Go task, increases in happiness were more sustained among individuals with BD. Finally, students with a history of mania endorse that they anticipate great success in domains involving public recognition, more frequently than students without a history of mania (Johnson et al. 2009). In sum, BD appears to be associated with elevated levels of reward- and approach-related emotions, as measured by degree and duration of affective responses to multiple stimulus types, and by self-reported ambitious goal-setting.

One approach-related emotion, prominently elevated in BD despite its uniquely negative valence, is anger (Carver 2004; Carver and Harmon-Jones 2009; Lara et al. 2006). Anger has been defined as a transient emotional reaction to an eliciting stimulus, consisting of subjective, physiological, and behavioral components, and ranging in intensity from mild irritation to fury and rage (Spielberger et al. 1983). Importantly, anger can be measured at the state level, by examining transient anger reactivity to eliciting stimuli, or at the trait level, by examining stable individual differences in the tendency to experience anger (Deffenbacher et al. 1996). Individuals high in trait-level anger experience more intense and frequent episodes of state anger in their daily lives (Spielberger et al. 1983; Spielberger 1999; Spielberger and Reheiser 2010).

Anger is a unique emotion in its combination of negative valence and approach-orientation (Carver 2004; Carver and Harmon-Jones 2009). It is typically considered a negative emotion, both because it is triggered by events that are unpleasant, and because most people report that the subjective experience of anger is negative (Harmon-Jones 2004). However, its motivational properties differ from other negative emotions. While each emotion has distinct

motivational properties (Keltner et al. 2006), the majority of negative emotions motivate withdrawal-type behavioral responses such as avoidance (as in the case of fear, known to elicit avoidance and escape from the triggering stimuli; Ohman and Mineka 2001) and submission (as in the case of shame; Gilbert and McGuire 1998). Anger, on the other hand, motivates approach of the triggering stimulus (Carver and Harmon-Jones 2009). Prominent models of anger describe it as a motivating force, helping individuals to overcome challenges and obstacles to goal attainment (Carver 2004; Frijda 1987; Levine 1996). Evolutionary accounts posit that it functions socially to motivate others to repair transgressions (Keltner et al. 2006), and repair conflicts of interest in favor of the angry individual (Sell et al. 2009). Consistent with this conceptualization, anger is characterized by short-term ‘attack’-type behaviors and subsequent reconciliation, whereas contempt is associated with both short- and long-term social rejection and exclusion of the offending individual (Fischer and Roseman 2007).

Given that BD is characterized by elevated approach-related emotions, it is perhaps unsurprising that heightened anger is a central clinical feature of BD that persists across the lifespan (Lara et al. 2006). Extreme anger has been well documented across manic, depressive, and remitted mood phases in BD (Lara et al. 2006). For example, symptom-rating scales for BD frequently include items such as, “more impatient or irritable” (Bech et al. 1979) and displaying “hostile, uncooperative behavior” (Young et al. 1978). During periods of depression, individuals with BD have been found to report elevated irritability and sudden episodes of intense anger (Deckersbach et al. 2004; Perlis et al. 2004). Remitted individuals with BD self-report elevated trait levels of anger (Dutra et al. 2014), and have been found to display higher rates of trait hostility compared to unaffected relatives (Savitz et al. 2008), suggesting a trait-like pattern of elevated anger. In addition, recent findings indicate that a subset of individuals with remitted BD, those with elevated levels of emotion-relevant impulsivity, report particularly severe and ongoing struggles with aggressive behavior as well as anger (Johnson and Carver 2016).

Significant literature documents that among healthy adults, anger is associated with diminished cognitive functioning, including reduced depth of processing and increased use of heuristics when evaluating persuasive messages (e.g., elevated reliance on superficial cues of an argument, such as the qualifications of its author, rather than argument quality) (Lerner and Tiedens 2006). For example, in one study, after a laboratory anger induction involving recalling an autobiographical event, participants were more likely to judge peers as guilty of misconduct based on ambiguous evidence compared with those who

had completed a sadness induction (Bodenhausen et al. 1994). In a similar study, incidental anger was found to decrease the amount of assistance participants recommended providing in a welfare case, relative to neutral emotion (Small and Lerner 2008). Further, in a replication and extension of this work, the authors found that limiting participants' cognitive resources eliminated this group difference, implying that differences in depth of processing drove this effect. This work suggests that anger may influence cognitive functioning, compromising an individual's depth of on-line information processing and interfering with engagement in tasks requiring elaborative processing. Thus, if cognitive functioning declines as anger increases, elevated anger in BD may compromise affected individuals' ability to make effective decisions in daily life.

The cognitive impact of anger may be particularly costly for individuals with BD, given the existing impairments in multiple domains of cognitive functioning associated with the disorder (Martinez-Aran et al. 2004; Robinson et al. 2006). Some evidence suggests that a more severe degree of cognitive impairment in BD is associated with a more severe illness course. For example, Robinson and Ferrier (2006) found a negative relationship between the number of manic episodes and verbal declarative memory performance. Consistent with this finding, Martinez-Aran et al. (2004) found that verbal memory impairment was related to illness duration, number of previous manic episodes, suicide attempts and hospitalizations.

While some deficits in cognitive performance among individuals with BD have been observed to persist across mood states, some may vary according to mood state at the time of testing. For example, Quraishi and Frangou (2002) found that while symptomatic patients with BD demonstrated widespread cognitive impairments (e.g., planning, set-shifting, and abstract concept formation), remitted individuals with BD were selectively impaired in areas of verbal memory and sustained attention. However, other findings suggest broader cognitive impairments during euthymic states in BD. A recent meta-analysis found deficits in response inhibition, set-shifting, executive functioning, verbal memory and sustained attention among euthymic individuals with BD (Bora et al. 2009). These deficits have been associated with poor functional outcomes, and may help to explain the impairments in community and daily life functioning observed among euthymic individuals with BD (Green 2006; Martinez-Aran et al. 2004). Thus, it is critical to better understand the factors that may contribute to such cognitive impairments in BD.

The Present Research

The present investigation had two main aims. In our first aim, we sought to examine whether individuals with BD

would demonstrate a unique pattern of impaired cognitive performance in the context of anger, compared to a never-psychiatric control group and a clinical comparison group of individuals with remitted major depressive disorder (MDD). Here, our focus was on cognitive performance outcomes, and specifically whether the trajectory of cognitive performance in the context of anger was unique for individuals with BD.

For this analysis, a remitted MDD group was chosen as a clinical comparison group as this population is also characterized by impaired cognitive functioning and episodes of intense anger (Austin et al. 2001; Brody et al. 1999). As such, inclusion of remitted individuals with MDD as a clinical comparison group provided an ideal means for examining whether anger and baseline cognitive deficits may interact in a particularly deleterious way in BD compared to a related mood disorder. In addition, comparing each of these groups with a group of community participants with no history of Axis I psychopathology allowed us to differentiate between patterns of anger-related responding associated with a specific disorder, mood disorders more generally, and those associated with healthy functioning. Self-reported anger, as well as positive and negative affect in order to examine the specificity of our results, were monitored during the anger provocation consisting of a 'rude experimenter' administering an arithmetic task.

We hypothesized that the BD group's performance would decline over the course of the anger provocation to a greater extent than our comparison groups (Hypothesis 1). This hypothesis is based on the prediction that the BD group's existing impairments in executive functioning would be exacerbated by the increasing cognitive load of anger over the course of the task. We predicted that anger's effects on cognition would be stronger in BD than in MDD, given existing data showing higher levels of executive dysfunction among remitted individuals with BD than with a history of unipolar depression (e.g., Smith et al. 2006). Specifically, while remitted individuals with MDD have demonstrated impairments in executive functioning compared to healthy controls, remitted individuals with BD show significantly more impairment than remitted individuals with MDD on tests of executive functioning and verbal memory.

For our second aim, we focused solely on the BD group to investigate the extent to which subjectively experienced anger might contribute to impaired cognitive performance in our main population of interest. Here, our focus was on exploring anger as a potential mechanism, specifically driving cognitive performance in our population of interest. Following from our prediction that existing executive functioning impairments in BD may be exacerbated by anger, we hypothesized that self-reported anger would have

a deleterious effect on cognitive performance for our BD group (Hypothesis 2).

Importantly, use of remitted clinical samples in our study allowed us to examine the role of anger in cognitive performance without the confound of mood symptoms at the time of testing. If group differences emerge in the context of remission, they are less likely attributable to current mood symptoms and more likely attributable to more stable features of the disorder.

Method

Participants

Participants were 27 individuals diagnosed with BD type I who were currently remitted (neither manic, depressed nor mixed for >1 month), 29 individuals diagnosed with MDD who were currently remitted (not depressed for >1 month), and 29 healthy controls (CTL) who did not meet current or past criteria for any DSM-IV-TR Axis I disorders. Participants were recruited using online advertisements and flyers posted in the New Haven, CT region. Exclusion criteria for both groups included history of severe head trauma, stroke, neurological disease, severe medical illness (e.g., autoimmune disorder, HIV/AIDS), and alcohol or substance abuse assessed during the clinical interview over the past 6 months. Participants were not excluded from the BD or MDD groups on the basis of comorbid Axis I disorders (aside from current substance or alcohol use disorders in the past 6 months) given that these diagnoses are commonly comorbid with other disorders (e.g., Kessler et al. 2005), though we verified that BD and MDD were the primary diagnoses for each respective group. The CTL group did not meet criteria for any current or lifetime Axis I disorders assessed. Groups did not differ significantly in age, gender, ethnicity, years of education, or employment status ($ps > 0.05$). See Table 1.

Measures of Clinical Functioning

Diagnostic Evaluation

All diagnoses were confirmed using the Structured Clinical Interview for DSM-IV (SCID-IV; First et al. 2007). Trained clinical psychology faculty, doctoral candidates, and post-baccalaureate research fellows administered the SCID-IV (First et al. 2007). Approximately one-fourth ($n = 22$; 25.9 %) of videotaped interviews were rated by an independent reviewer who watched the videos offline. Ratings matched 100 % of primary diagnoses, and reliability was high across all Axis I diagnoses ($\kappa_{\text{mean}} = 1.00$). BD participants were currently remitted (neither manic nor

depressed) for an average of 15.85 months ($SD = 19.13$), and MDD participants were currently remitted for an average of 39.83 months ($SD = 40.81$), at the time of testing. Clinical characteristics of BD and MDD groups are presented in Table 1.

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 current manic symptoms with scores ranging from 0 to 60, with higher scores indicating greater severity. Scores ≥ 7 represent clinically significant symptoms. The YMRS has demonstrated acceptable internal consistency ($\alpha \geq 0.8$), and clinical sensitivity comparable to similar measures (Fristad et al. 1995; Young et al. 1978). Current depression symptoms were measured using the Inventory of Depressive Symptomatology—Clinician Rated (IDS-C; Rush et al. 1996), a 30-item, clinician-rated measure of current depressive symptoms with scores ranging from 0 to 84, with higher scores indicating greater depressive severity. Scores ≥ 11 represent clinically significant symptoms. The IDS-C has demonstrated high internal consistency ($\alpha \geq 0.89$), and sensitivity and specificity equal to or greater than other standard measures of depression (Rush et al. 1996; Trivedi et al. 2004).

Intra-class correlations for absolute agreement between the original interviewer and an independent rater who watched interview videos offline for approximately one fifth of study participants ($n = 18$; 21.18 %) were strong for both the YMRS (0.96) and IDS-C (0.95). Remitted mood status (i.e., neither manic, depressed, nor mixed mood state) 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. The CTL group also scored below these cutoffs.

Global Functioning

The Global Assessment of Functioning Scale (GAF; Luborsky 1962) was used to assess functioning in the past week. The GAF assesses overall psychological, social, and occupational functioning on a scale from 1 (lowest) to 100 (highest). ICC for absolute agreement between the original interviewer and an independent rater for one-fifth of study participants ($n = 18$; 21.18 %) was high (ICC = 0.96).

Medication

Participants self-reported medication types and dosages at the first laboratory visit. Psychotropic medications included

Table 1 Demographic and clinical characteristics

	BD (<i>n</i> = 27)	MDD (<i>n</i> = 29)	CTL (<i>n</i> = 29)	Statistic	Effect Size	Levene's Test	Degrees of Freedom
Demographic							
Age (years)	30.89 (8.84)	30.46 (10.61)	31.89 (8.98)	$F = 0.17$	$\eta_p^2 < 0.01$	$F = 0.88$	2,82
Female (%)	66.67	65.52	62.07	$\chi^2 = 0.14$	$V = 0.04$		
Caucasian (%)	88.89	93.10	89.66	$\chi^2 = 8.17$	$V = 0.22$		
Education (years)	15.06 (2.29)	15.24 (2.26)	16.05 (2.38)	$F = 1.47$	$\eta_p^2 = 0.04$	$F = 1.47$	2,82
Employed (%)	51.85	51.72	68.97	$\chi^2 = 8.40$	$V = 0.22$		
Clinical							
YMRS	1.91 (2.01)	1.55 (1.64)	1.21 (1.76)	$F = 1.05$	$\eta_p^2 = 0.03$	$F = 1.05$	2,82
IDS-C	5.67 (3.63)	5.72 (3.10)	2.28 (2.33)	$F = 12.01^*$	$\eta_p^2 = 0.23$	$F = 4.15^*$	2,82
GAF	75.30 (6.06)	78.97 (7.09)	88.07 (3.08)	$F = 38.17^*$	$\eta_p^2 = 0.48$	$F = 13.15^*$	2,82
Working Memory	10.44 (3.07)	12.03 (2.78)	12.34 (3.21)	$F = 2.83$	$\eta_p^2 = 0.07$	$F = 0.23$	2,82
Intellectual functioning	31.67 (4.20)	33.76 (3.46)	33.00 (2.84)	$F = 2.51$	$\eta_p^2 = 0.58$	$F = 4.27^*$	2,82
Age of Onset	16.61 (7.00)	15.74 (6.97)		$F = 0.22$	$\eta_p^2 < 0.01$	$F = 0.02$	1,54
Manic Episodes	12.02 (21.82)	5.67 (7.71)		$F = 2.61$	$\eta_p^2 = 0.11$	$F = 7.04^*$	1, 54
Depressive Episodes	12.87 (22.62)	39.83 (40.81)		$F = 7.49$	$\eta_p^2 = 0.12$	$F = 5.22^*$	1,53
Time Remitted (mos.)	15.85 (19.13)	0.55 (0.87)		$F = 21.08^*$	$\eta_p^2 = 0.28$	$F = 9.47^*$	1,54
Psychotropic Meds	2.07 (1.54)	0.69 (0.93)		$F = 0.28$	$\eta_p^2 = 0.01$	$F = 0.23$	1,54
Comorbid Diagnoses	0.56 (0.97)						

mean values are displayed with standard deviations in parentheses where applicable

BD = bipolar I disorder group; MDD = major depressive disorder group; CTL = healthy control group; YMRS = Young Mania Rating Scale; IDS-C = Inventory to Diagnose Depression; GAF = global assessment of functioning; Working Memory = Wechsler Adult Intelligence Inventory, 4th edition; Intellectual Functioning = Shipley Institute of Living Scale; Age of Onset = Age of BD or MDD Onset; Manic Episodes = number of lifetime manic or hypomanic episodes; Depressive Episodes = number of lifetime major depressive episodes; Time Remitted = number of months remitted prior to study participation; Psychotropic Meds = number of psychotropic medications; Comorbid Diagnoses = number of comorbid DSM-IV-TR axis I diagnoses

* $p < 0.05$

lithium (BD $n = 7$), anticonvulsants (BD $n = 12$, MDD $n = 2$) antidepressants (BD $n = 3$, MDD $n = 9$), neuroleptics (BD $n = 11$, MDD $n = 1$), anxiolytics (BD $n = 7$, MDD $n = 3$), stimulants (BD $n = 3$, and sedative-hypnotics (BD $n = 1$). Levels of each class of medication were recorded using the Somatotherapy Index and compiled to calculate an Intensity of Somatotherapy Score for each participant (Bauer et al. 1997). Average number of psychotropic medications are reported in Table 1.

Self-Reported Anger, Positive Affect, and Negative Affect

Self-reported emotion was assessed using the modified Differential Emotions Scale (mDES; Cohn et al. 2009). The mDES consists of 18 positive and negative emotion terms rated on a 1 (*not at all*) to 5 (*extremely*) scale. We examined the anger item individually as our target emotion. The remaining items were averaged to create a PA composite (amusement, awe, contentment, joy, gratitude, hope, love, pride, sympathy, interest; $\alpha_{\text{mean}} = 0.93$) and

NA composite (fear, disgust, embarrassment, guilt, sadness, shame, contempt; $\alpha_{\text{mean}} = 0.60$).¹

Task Performance Measures

Task performance measures were recorded off-line by a research assistant who watched video recordings of each participant during the anger provocation task. The trained coder rated two dimensions of cognitive performance including: (1) accuracy and (2) engagement. Accuracy was operationalized as the total number of correct responses (numbers verbally reported by participants which reflected accurate calculations, following the directions of the experimenter). Engagement was operationalized as the total number of responses made (numbers verbally reported by participants).

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

Baseline Cognitive Measures

Baseline Working Memory

We measured baseline 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 letters. After the list was read aloud, participants were asked to verbally repeat back 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 were computed for final analyses. The Letter-Number Sequencing task has demonstrated high internal consistency ($\alpha \geq 0.88$) acceptable test-retest reliability ($r = 0.78$), and contributes substantially to a working memory factor among WAIS-IV subtests (loading for the total group = 0.77) (Sattler and Ryan 2009; Wechsler 2008; Weiss et al. 2010).

Baseline Intellectual Functioning

The Shipley Institute of Living Scale (SILS; Zachary 1986) was included as a measure of general intellectual functioning. The vocabulary subtest of the SILS was administered, consisting of 40 multiple-choice questions in which the participant is asked to select one of four words closest in meaning to the target word. Scores range from 0 to 40. In previous research, the SILS has demonstrated acceptable internal consistency, with reliability estimates ranging from 0.78 to 0.89 (acceptable) (Szyhowski 2008).

Procedure

Participants first provided written and verbal informed consent. The study consisted of three parts. First, participants completed a diagnostic interview to determine eligibility using the SCID-IV (First et al. 2007). Baseline assessments of working memory and intellectual functioning were obtained immediately after the SCID by the same trained interviewer. Second, approximately 1 week later ($M = 7.95$ days, $SD = 3.09$), participants returned to the lab. After confirming informed consent, current symptoms were reassessed to ensure continued remitted status for all groups using the YMRS (≤ 7) and IDS-C (≤ 11). Prior to the present anger elicitation task, participants completed a series of unrelated tasks including a physiological baseline measurement, and completion of several computer tasks. Computer tasks included the ‘reading the mind in the eyes’ task (Baron-Cohen et al. 2001), viewing brief emotion-eliciting films and self-reporting emotional reactivity, an

autobiographical memory task, and ‘karaoke’ singing task. Third, participants were oriented to the anger provocation task by the experimenter and questions were answered. The anger provocation task, a previously validated task that has been shown to reliably provoke anger (Mauss et al. 2006, 2007a; b), allows for concurrent assessment of cognitive functioning, as it requires participants to make arithmetic calculations. Computerized software (MediaLab v2008; New York, NY, USA) was used to guide participants through the experiment, present instructions, and collect questionnaire information. Participants were observed remotely via digital cameras from a separate room. During the task, a pre-recorded voice, used in prior work by Mauss and colleagues (2006), was transmitted over an intercom system to the experimental room by the experimenter. Participants were told that this pre-recorded voice belonged to an experimenter in the adjacent room. Over the intercom, participants were informed that they would be participating in a cognitive task.

To establish an emotional baseline, participants first watched an emotionally neutral baseline film with scenes from *Denali* (110 s), following prior research (Mauss et al. 2006), and self-reported anger, PA, and NA was subsequently collected. Next, participants began the cognitive component of the task in which they were asked to count backwards in steps of 7 or 13 from a high 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 (e.g., 15,293) for a total of three cognitive trials. In between the three trials, 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 suggesting that data collection be terminated (“let’s just stop here”). Spontaneous clarifications and questions from participants were answered using pre-recorded prompts as needed (e.g., “yes” and “no”). At the end of the task, participants self-reported their emotion experience using the mDES. Of note, self-reported emotion was measured only twice, at baseline and at the end of the task. Next, participants remained seated for 120 s and viewed a calming clip from *Planet Earth* (210 s) to facilitate mood recovery. Finally, experimenters gave a thorough debriefing and answered questions.

Results

Manipulation Check: Group Differences in Self-Reported Anger

To ensure that the anger provocation task elicited significant increases in anger across all three groups, a 3 (Group:

BD, MDD, CTL) \times 2 (Task: Baseline, Task) repeated-measures ANOVA was conducted on self-reported anger. Results revealed a main effect of Time, with anger increasing significantly from baseline to task, $F(1,81) = 95.53$, $p < 0.001$, $\eta_p^2 = 0.54$. Neither the main effect of Group $F(1,81) = 0.48$, $p = 0.62$, $\eta_p^2 = 0.01$ nor the Group \times Task interaction was significant, $F(2,81) = 0.22$, $p = 0.80$, $\eta_p^2 = 0.01$.

Aim 1: Group Differences in Task Performance

To address Aim 1, two separate 3 (Task Trial: 1, 2, 3) \times 3 (Group: BD, MDD, CTL) repeated-measures ANOVAs were conducted on Number of Correct Responses (accuracy) and Number of Responses (engagement). 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.

For Number of Correct Responses, a significant main effect of Task Trial emerged, $F(2,164) = 26.81$, $p < 0.001$, $\eta_p^2 = 0.25$ (see Table 2 for Group and Trial mean values). The main effect of Group was not significant, $F(2,82) = 0.90$, $p = 0.41$, $\eta_p^2 = 0.02$, and the Group \times Task Trial interaction was not significant, $F(4,164) = 0.23$, $p = 0.92$, $\eta_p^2 = 0.01$.

For Number of Responses, a significant Group \times Task Trial interaction effect emerged, $F(4,164) = 3.05$, $p = 0.02$, $\eta_p^2 = 0.019$, reflecting a unique pattern of declining numbers of responses over the course of the task in the BD group (see Table 2 for Group and Trial mean values, and Fig. 1 for graphical representation of results). A main effect of Task Trial also emerged, $F(2,164) = 12.30$, $p < 0.001$, $\eta_p^2 = 0.13$, reflecting fewer responses in trial 3 compared to

trials 1 and 2. The main effect of Group was not significant, $F(2,82) = 0.24$, $p = 0.78$, $\eta_p^2 = 0.01$.

Aim 2: Anger as a Predictor of Task Performance in BD

To address Aim 2, two hierarchical regressions were conducted within the BD group, examining the role of subjective anger in predicting task performance. Self-reported anger at baseline was entered into the first step, and self-reported anger at the end of the task was entered into the second step in order to examine the role of anger reactivity to the manipulation in predicting task performance, above and beyond effects of baseline anger. One regression was run with Total Number of Correct Responses as the dependent variable, and another was run with Total Number of Responses. In this way, we tested whether anger during the task, above and beyond baseline levels of anger, predicted task performance. Results indicated that, consistent with Hypothesis 2, self-reported anger during the task predicted fewer Correct Responses ($\beta = -0.47$, $t = -2.07$, $p = 0.049$) and fewer Total Responses ($\beta = -0.54$, $t = -2.48$, $p = 0.02$).

To examine the specificity of these results to anger as opposed to positive and negative affect more generally, the same analyses were performed using the self-reported PA and NA composites from the mDES. PA did not predict Number of Correct Responses ($\beta = 0.12$, $t = 0.39$, $p = 0.70$) or Total Number of Responses ($\beta = -0.01$, $t = -0.04$, $p = 0.97$). NA also did not significantly predict Number of Correct Responses ($\beta = -0.11$, $t = -0.47$, $p = 0.65$) or Total Number of Responses ($\beta = -0.23$, $t = -1.00$, $p = 0.33$).

Table 2 Mean values for cognitive performance variables during anger elicitation task

	BD (<i>n</i> = 27)	MDD (<i>n</i> = 29)	CTL (<i>n</i> = 29)	All (<i>n</i> = 85)
Engagement (number of responses)				
Trial 1	12.04 (7.26)	10.07 (4.99)	10.14 (5.19)	10.72 (5.87)
Trial 2	8.74 (7.69)	12.03 (5.05)	11.76 (7.42)	10.89 (6.88)
Trial 3	7.11 (6.62)	8.66 (6.76)	7.69 (5.43)	7.84 (6.23)
Accuracy (number of correct responses)				
Trial 1	5.85 (6.14)	6.41 (5.50)	8.07 (6.34)	6.80 (6.01)
Trial 2	6.96 (7.29)	7.76 (6.33)	8.72 (6.62)	7.84 (6.70)
Trial 3	3.93 (4.47)	3.97 (3.86)	5.21 (3.85)	4.38 (4.06)

Mean values are displayed with standard deviations in parentheses where applicable

BD = bipolar I disorder group; MDD = major depressive disorder group; CTL = healthy control group

Task Engagement (Number of Responses) Across Task Segments

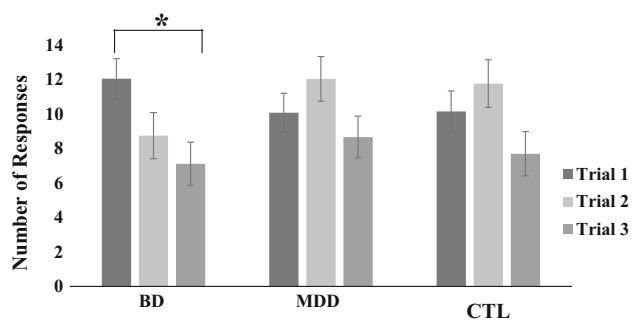


Fig. 1 Task engagement (number of responses) across task segments. Number of responses given by each group within task trials. As the task progressed, participants in the BD group gave fewer responses over time, resulting in a significantly decreased number of responses from Trial 1 to Trial 3. No significant differences emerged in the number of responses given over time within the MDD or CTL groups

Discussion

The present study examined the effects of anger on cognitive performance among individuals with BD. Cognitive performance was measured by accuracy and engagement in an arithmetic task during a validated anger provocation. Two main aims were explored in a group of individuals with remitted BD, a clinical comparison group of participants with remitted MDD, and healthy controls.

Our first aim examined group differences in cognitive task performance over the course of the anger provocation task. Results indicate a reduction in task engagement (number of responses) over time in our BD group, compared with the MDD and CTL groups, and suggest that in context of anger, some aspects of cognitive performance may decline uniquely among individuals with BD. Notably, while the number of responses provided by the BD group declined, no group differences emerged in arithmetic accuracy. This finding suggests the possibility of slowed cognitive processing (but otherwise intact performance) in the context of anger in BD. Given that perceptions of anger have been found to capture attention in a manner that takes priority over other tasks (Hansen and Hansen 1988), one possibility is that this attentional load is particularly taxing on the processing efficiency of individuals with BD, given their existing executive deficits in this specific area (Fleck et al. 2005). This explanation is consistent with our findings of reduced rate of responding to task prompts in the BD group, despite intact arithmetic accuracy of responses.

Our second aim was to examine whether subjective anger experienced during the task predicted task performance for individuals with BD. Results indicated that greater anger was associated with fewer correct responses, and fewer responses overall. These results provide support for the idea that the subjective experience of anger confers a cognitive vulnerability among individuals with BD. Taken together, the findings described herein shed important light on the role of anger in cognitive performance in BD, and guide future research in this area.

Our findings should be interpreted within the confines of several caveats. First, although our sample size is common in experimental studies with severe psychiatric samples, replication in a larger sample size will enable examination of unique patterns of anger response among BD subtypes with various comorbidities and medication profiles. Second, to ensure an ecologically valid sample, the majority of our BD patients were on medication at the time of testing. Still, future studies could more carefully test for effects of psychotropic medication on anger and cognition by recruiting BD participants carefully matched on medication subclasses. Third, though our patient samples met criteria for clinical remission at the time of testing ($YMRS \leq 7$

and $IDS-C \leq 11$), both patient groups reported significantly greater subthreshold depression symptoms compared to the healthy control group. Future studies could address this potential confound by comparing subgroups of patient samples varying on subthreshold mood symptoms. A next step in this line of work would be to compare individuals with BD in remitted states with those in manic and depressed states. Doing so would allow for better understanding of the extent to which the cognitively impairing effects of anger may vary across mood states in BD. Finally, although we excluded individuals meeting criteria for substance abuse or dependence in the 6 months preceding study participation, it is possible that substance dependence prior to this time could have influenced our results. Future efforts at replication and extension of our findings could aim to evaluate this possibility. Nonetheless, the present study addressed a critical yet understudied area regarding the interaction between anger and cognition in BD. In addition, inclusion of the clinical control group allowed for better understanding of anger's effects on cognition within BD specifically, alongside a clinical group also characterized by some dysregulated anger as well as cognitive impairment.

The findings described here have important implications for our understanding of BD, and for future research building on these findings to improve functioning in daily life and treatment efficacy for individuals with BD. Future research could examine more closely the mechanisms involved in cognitive task disengagement in response to anger provocation in BD; in particular, the extent to which individual components of anger provocation (e.g., perception of anger in others, subjective anger experience, physiological aspects of anger) contribute to task disengagement. It is important to consider alternative explanations of the present findings. One possibility for future work to explore is that cognitive difficulties observed in the BD group may be linked to emotion regulatory capacity deficits which, in turn, might cause these individuals to experience and report higher levels of anger when provoked. In this case, identifying and treating these cognitive impairments is an important step forward to hopefully reduce anger and irritability, an approach which has demonstrated promise among individuals diagnosed with attention deficit hyperactivity disorder (Connor et al. 2002).

Our findings could also be extended by examining relationships between anger, cognition, and real-world psychosocial outcomes, such as effective participation in treatment, social relationships, and occupational functioning. Specifically, it would be important to know whether there may be a critical threshold of preserved cognitive capacity for effective engagement in cognitive therapies, which may be diminished by anger, cognitive deficits, or

some combination of both. If this were the case, incorporating anger monitoring and anger reduction strategies into existing cognitive-behavioral treatments for BD could significantly increase treatment tolerability and efficacy. As such, continued investigation of the role of anger in cognitive performance in BD promises to improve our understanding of the disorder, and our ability to effectively treat and restore functioning to those affected.

Acknowledgments This study was supported by a NARSAD Young Investigator Grant from the Brain and Behavior Research Foundation (JG) and a CTSA Grant Number UL1 RR024139 from the National Center for Research Resources (NCRR) and the National Center for Advancing Translational Science (NCATS), and NIH roadmap for Medical Research (JG). Its contents are solely the responsibility of the authors and do not necessarily represent the official view of NIH. We thank Amelia Aldao, Kirsten Gilbert, Franziska Goer, Yoona Kang, Melissa Lavoie, and Amanda Purcell for special assistance.

Compliance with Ethical Standards

Conflict of Interest Sunny J. Dutra, Greg J. Siegle, Elizabeth J. Reeves, Iris B. Mauss, and June Gruber declare that they have no conflict of interest.

Informed Consent This study was approved by the Yale University Human Research Protection Program. Participants provided written and verbal informed consent at the first study session, prior to beginning any study procedures. Informed consent was re-confirmed at the beginning of the second study session.

Animal Rights No animal studies were carried out by the authors for this article.

References

- Alloy, L. B., & Abramson, L. Y. (2010). The role of the behavioral approach system (BAS) in bipolar spectrum disorders. *Current Directions in Psychological Science*, *19*, 189–194.
- Austin, M. P., Mitchell, P., & Goodwin, G. M. (2001). Cognitive deficits in depression: Possible implications for functional neuropathology. *British Journal of Psychiatry*, *178*, 200–206.
- Baron-Cohen, S., Wheelwright, S., Hill, J., Raste, Y., & Plumb, I. (2001). The “reading the mind in the eyes” test revised version: A study with normal adults, and adults with Asperger syndrome or high-functioning autism. *Journal of Child Psychology and Psychiatry*, *42*, 241–251.
- Bauer, M. S., McBride, L., Shea, N., Gavin, C., Holden, F., & Kendall, S. (1997). Impact of an easy-access VA clinic-based program for patients with bipolar disorder. *Psychiatric Services*, *48*, 491–496.
- Bech, P., Bolwig, T. G., Kramp, P., & Rafaelsen, O. J. (1979). The Bech-Rafaelsen Mania Scale and the Hamilton Depression Scale. *Acta Psychiatrica Scandinavica*, *59*, 420–430.
- Bodenhausen, G. V., Sheppard, L. A., & Kramer, G. P. (1994). Negative affect and social judgment: The differential impact of anger and sadness. *European Journal of Social Psychology*, *24*, 45–62.
- Bora, E., Yucel, M., & Pantelis, C. (2009). Cognitive endophenotypes of bipolar disorder: A meta-analysis of neuropsychological deficits in euthymic patients and their first-degree relatives. *Journal of Affective Disorders*, *113*, 1–20.
- Brody, C. L., Haaga, D. A., Kirk, L., & Solomon, A. (1999). Experiences of anger in people who have recovered from depression and never-depressed people. *Journal of Nervous and Mental Disease*, *187*, 400–405.
- Carver, C. S. (2004). Negative affects deriving from the behavioral approach system. *Emotion*, *4*, 3–22.
- Carver, C. S., & Harmon-Jones, E. (2009). Anger is an approach-related affect: Evidence and implications. *Psychological Bulletin*, *135*, 183–204.
- Cohn, M. A., Fredrickson, B. L., Brown, S. L., Mikels, J. A., & Conway, A. M. (2009). Happiness unpacked: Positive emotions increase life satisfaction by building resilience. *Emotion*, *9*(3), 361–368.
- Connor, D. F., Glatt, S. J., Lopez, I. D., Jackson, D., & Melloni, R. H. (2002). Psychopharmacology and aggression. I: A meta-analysis of stimulant effects on overt/covert behaviors in ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, *41*, 253–261.
- Deckersbach, T., Perlis, R. H., Frankle, W. G., Gray, S. M., Grandin, L., Dougherty, D. D., et al. (2004). Presence of irritability during depressive episodes in bipolar disorder. *CNS Spectrums*, *9*, 227–231.
- Deffenbacher, J. L., Oetting, E. R., Thwaites, G. A., Lynch, R. S., Baker, D. A., Stark, R. S., et al. (1996). State-trait anger theory and the utility of the trait anger scale. *Journal of Counseling Psychology*, *43*, 131–148.
- Dutra, S. J., Reeves, E. J., Mauss, I. B., & Gruber, J. (2014). Boiling at a different degree: An investigation of trait and state anger in remitted bipolar I disorder. *Journal of Affective Disorder*, *168*, 37–43.
- Farmer, A., Lam, D., Sahakina, B., Roiser, J., Burke, A., O’Neill, N., et al. (2006). A pilot study of positive mood induction in euthymic bipolar subjects compared with healthy controls. *Psychological Medicine*, *36*, 1213–1218.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. (2007). *Structured clinical interview for DSM-IV-TR axis I disorders, research version, patient edition (SCID-VP)*. New York: Biometrics Research, New York State Psychiatric Institute.
- Fischer, A. H., & Roseman, I. J. (2007). Beat them or ban them: The characteristics and social functions of anger and contempt. *Journal of Personality and Social Psychology*, *93*, 103–115.
- Fleck, D. E., Shear, P. K., & Strakowski, S. M. (2005). Processing efficiency and sustained attention in bipolar disorder. *Journal of the International Neuropsychological Society*, *11*, 49–57.
- Frijda, N. H. (1987). Emotion, cognitive structure, and action tendency. *Cognition and Emotion*, *1*, 115–143.
- Fristad, M. A., Weller, R. A., & Weller, E. B. (1995). The Mania Rating Scale (MRS): Further reliability and validity studies with children. *Annals of Clinical Psychiatry*, *7*, 127–132.
- Gilbert, P., & McGuire, M. (1998). Shame, social roles and status: The psychobiological continuum from monkey to human. In P. Gilbert & B. Andrews (Eds.), *Shame: Interpersonal behavior, psychopathology and culture* (pp. 99–125). New York: Oxford University Press.
- Green, M. F. (2006). Cognitive impairment and functional outcome in schizophrenia and bipolar disorder. *The Journal of Clinical Psychiatry*, *67*, e12.
- Gruber, J. (2011). Can feeling too good be bad? Positive emotion persistence (PEP) in bipolar disorder. *Current Directions in Psychological Science*, *20*, 217–221.
- Gruber, J., Harvey, A. G., & Johnson, S. L. (2009). Reflective and ruminative processing of positive emotional memories in bipolar disorder and healthy controls. *Behaviour Research and Therapy*, *47*, 697–704.

- Gruber, J., & Johnson, S. L. (2009). Positive emotional traits and ambitious goals among people at risk for mania: The need for specificity. *International Journal of Cognitive Therapy*, 2, 176.
- Gruber, J., Johnson, S. L., Oveis, C., & Keltner, D. (2008). Risk for mania and positive emotional responding: Too much of a good thing? *Emotion*, 8, 23–33.
- Hansen, C. H., & Hansen, R. D. (1988). Finding the face in the crowd: An anger superiority effect. *Emotion*, 54, 917–924.
- Harmon-Jones, E. (2004). On the relationship of anterior brain activity and anger: Examining the role of attitude toward anger. *Cognition and Emotion*, 18, 337–361.
- Johnson, S. L. (2005). Mania and dysregulation in goal pursuit: A review. *Clinical Psychology Review*, 25, 241–262.
- Johnson, S. L., & Carver, C. S. (2006). Extreme goal setting and vulnerability to mania among undiagnosed young adults. *Cognitive Therapy & Research*, 30, 377–395.
- Johnson, S. L., & Carver, C. S. (2016). Emotion-relevant impulsivity predicts sustained anger and aggression after remission in bipolar I disorder. *Journal of Affective Disorders*, 189, 169–175.
- Johnson, S. L., Eisner, L. R., & Carver, C. S. (2009). Elevated expectancies among persons diagnosed with bipolar disorder. *British Journal of Clinical Psychology*, 48, 217–222.
- Johnson, S. L., Gruber, J., & Eisner, L. (2007). Emotion in bipolar disorder. In J. Rottenberg & S. L. Johnson (Eds.), *Emotion and psychopathology: Bridging affective and clinical science*. Washington, DC: American Psychological Association.
- Keltner, D., Haidt, J., & Shiota, M. N. (2006). Social functionalism and the evolution of emotions. In M. Schaller, J. A. Simpson, & D. T. Kenrick (Eds.), *Evolution and social psychology* (pp. 115–142). East Sussex: Psychology Press.
- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, 62, 617–627.
- Lara, D. R., Pinto, O., Akiskal, K., & Akiskal, H. S. (2006). Toward an integrative model of the spectrum of mood, behavioral and personality disorders based on fear and anger traits: I. Clinical implications. *Journal of Affective Disorders*, 94, 67–87.
- Lerner, J. S., & Tiedens, L. Z. (2006). Portrait of the angry decision maker: How appraisal tendencies shape anger's influence on cognition. *Journal of Behavioral Decision Making*, 19, 115–137.
- Levine, L. J. (1996). The anatomy of disappointment: A naturalistic test of appraisal models of sadness, anger, and hope. *Cognition and Emotion*, 10, 337–359.
- Luborsky, L. (1962). Clinicians' judgments of mental health. A proposed scale. *Archives of General Psychiatry*, 7, 407–417.
- Martinez-Aran, A., Vieta, E., Colom, F., Torrent, C., Sanchez-Moreno, J., Reinares, M., et al. (2004). Cognitive impairment in euthymic bipolar patients: Implications for clinical and functional outcome. *Bipolar Disorder*, 6, 224–232.
- Mauss, I. B., Cook, C. L., Cheng, J. Y., & Gross, J. J. (2007a). Individual differences in cognitive reappraisal: Experiential and physiological responses to an anger provocation. *International Journal of Psychophysiology*, 66, 116–124.
- Mauss, I. B., Cook, C. L., & Gross, J. J. (2007b). Automatic emotion regulation during anger provocation. *Journal of Experimental Social Psychology*, 43, 698–711.
- Mauss, I. B., Evers, C., Wilhelm, F. H., & Gross, J. J. (2006). How to bite your tongue without blowing your top: Implicit evaluation of emotion regulation predicts affective responding to anger provocation. *Personality and Social Psychology Bulletin*, 32, 389–602.
- Meyer, T. D., & Baur, M. (2009). Positive and negative affect in individuals at high and low risk for bipolar disorders. *Journal of Individual Differences*, 30, 169–175.
- Meyer, B., Johnson, S. L., & Winters, R. (2001). Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS scales with symptoms. *Journal of Psychopathology and Behavioral Assessment*, 23, 133–143.
- Nusslock, R., Alloy, L. B., Abramson, L. Y., Harmon-Jones, E., & Hogan, M. E. (2008). Impairment in the achievement domain in bipolar spectrum disorders: Role of behavioral approach system hypersensitivity and impulsivity. *Minerva Pediatrica*, 60, 41–50.
- Ohman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: Toward an evolved module of fear and fear learning. *Psychological Review*, 108, 483–522.
- Perlis, R. H., Smoller, J. W., Fava, M., Rosenbaum, J. F., Nierenberg, A. A., & Sachs, G. S. (2004). The prevalence and clinical correlates of anger attacks during depressive episodes in bipolar disorder. *Journal of Affective Disorders*, 79, 291–295.
- Quraishi, S., & Frangou, S. (2002). Neuropsychology of bipolar disorder: A review. *Journal of Affective Disorders*, 72, 209–226.
- Robinson, L. J., & Ferrier, I. N. (2006). Evolution of cognitive impairment in bipolar disorder: A systematic review of cross-sectional evidence. *Bipolar Disorders*, 8, 103–116.
- Robinson, L. J., Thompson, J. M., Gallagher, P., Goswami, U., Young, A. H., Ferrier, I. N., & Moore, P. B. (2006). A meta-analysis of cognitive deficits in euthymic patients with bipolar disorder. *Journal of Affective Disorders*, 93, 105–115.
- Rush, A. J., Gullion, C. M., Basco, M. R., Jarrett, R. B., & Trivedi, M. H. (1996). The Inventory of Depressive Symptomatology (IDS): Psychometric Properties. *Psychological Medicine*, 26(3), 477–486.
- Sattler, J. M., & Ryan, J. J. (2009). *Assessment with the WAIS-IV*. San Diego, CA: Jerome M. Sattler Publisher Inc.
- Savitz, J., van der Merwe, L., & Ramesar, R. (2008). Hypomanic, cyclothymic, and hostile personality traits in bipolar spectrum illness: A family-based study. *Journal of Psychiatric Research*, 42, 920–929.
- Sell, A., Tooby, J., & Cosmides, L. (2009). Formidability and the logic of human anger. *Proceedings of the National Academy of Sciences*, 106, 15073–15078.
- Small, D. A., & Lerner, J. S. (2008). Emotional policy: Personal sadness and anger shape judgments about a welfare case. *Political Psychology*, 29, 149–168.
- Smith, D. J., Muir, W. J., & Blackwood, D. H. R. (2006). Neurocognitive impairment in euthymic young adults with bipolar spectrum disorder and recurrent major depressive disorder. *Bipolar Disorders*, 8, 40–46.
- Spielberger, C. D. (1999). State-Trait Anger Expression Inventory-2 (STAXI-2). Professional Manual. Tampa, FL: Psychological Assessment Resources.
- Spielberger, C. D., Jacobs, G. A., Russell, S., & Crane, R. S. (1983). Assessment of anger: The state-trait anger scale. In J. N. Butcher & C. D. Spielberger (Eds.), *Advances in personality assessment* (Vol. 2). Hillsdale, NJ: Lawrence Erlbaum Associates Inc.
- Spielberger, C. D., & Reheiser, E. C. (2010). The nature and measurement of anger. In M. Potegal, G. Stemmler, & C. D. Spielberger (Eds.), *International handbook of anger* (pp. 403–412). New York: Springer.
- Szykowski, D. (2008). A psychometric study of the Shipley Institute of Living Scale. *Dissertation Abstracts International: Section B: The Sciences and Engineering*, 69(5-B), 3315.
- Trivedi, M. H., Rush, A. J., Ibrahim, H. M., Carmody, T. J., Biggs, M. M., Suppes, T., et al. (2004). The Inventory of Depressive Symptomatology, Clinician Rating (IDS-C) and Self-Report (IDS-SR), and the Quick Inventory of Depressive Symptomatology, Clinician Rating (QIDS-C) and Self-Report (QIDS-SR) in public sector patients with mood disorders: A psychometric evaluation. *Psychological Medicine*, 34, 73–82.

- Wechsler, D. (2008). *Wechsler Adult Intelligence Scale—fourth edition: Technical and interpretive manual*. San Antonio, TX: Pearson.
- Weiss, L. G., Saklofske, D. H., Coalson, D., & Raiford, S. E. (Eds.). (2010). *WAIS-IV clinical use and interpretation: Scientist-practitioner perspectives*. San Diego, CA: Academic Press.
- Woods, S. W. (2000). The economic burden of bipolar disease. *Journal of Clinical Psychiatry*, *61*, 38–41.
- Young, R. C., Biggs, J. T., Ziegler, E., & Meyer, D. A. (1978). A rating scale for mania: Reliability, validity, and sensitivity. *British Journal of Psychiatry*, *133*, 429–435.
- Zachary, R. A. (1986). *Shibley Institute of Living Scale (SILS): Revised Manual*. Los Angeles: Western Psychological Services.
- Zarate, C. A., Tohen, M., Land, M., & Cavanagh, S. (2000). Functional impairment and cognition in bipolar disorder. *The Psychiatric Quarterly*, *71*, 309–329.