Published in final edited form as:
J Clin Psychol. 2009 September ; 65(9): 925–941. doi:10.1002/jclp.20596.

Affective Reactivity in Response to Criticism in Remitted Bipolar Disorder: A Laboratory Analog of Expressed Emotion

Amy K. Cuellar, **Sheri L. Johnson**, and **Camilo J. Ruggero**
University of Miami

Abstract

Potential mechanisms to explain the relationship between Expressed Emotion (EE) and poor outcome within bipolar disorder are poorly understood. One possibility is that people with bipolar disorder have difficulty regulating their affect in response to criticism. The present study examined whether participants with bipolar disorder were more affectively dysregulated than control participants when presented with a criticism by a confederate. There was a trend for people with bipolar disorder to react more negatively to the criticism, but there was also evidence that they recovered as quickly as controls. Exploratory analyses found that female gender, the perception of the criticism as more negative, being disabled, and having fewer positive relationships predicted greater reactivity to criticism among people with bipolar disorder.

Keywords

bipolar disorder; expressed emotion; affective reactivity; family criticism; emotion regulation

Despite gains in pharmacotherapy (Goldberg, 2004), data on the prognosis of bipolar disorder are sobering. Even among patients with therapeutic blood levels of lithium, approximately 50% of individuals with bipolar disorder relapse within the first year (Keller, Lavori, Coryell, Endicott, & Mueller, 1993; Tohen, Waternaux, & Tsuang, 1990) and 70% relapse within 5 years (Gitlin, Swendsen, Heller, & Hammen, 1995). Considering these gaps, Prien and Potter (1990) urged researchers to examine psychosocial factors in bipolar disorder.

One psychosocial factor that has been shown to strongly influence the course of bipolar disorder is expressed emotion (EE; Butzlaff & Hooley, 1998). Defined as family overinvolvement, criticism, and hostility towards the patient, almost 20 years of research have documented that EE exerts a significant effect on the course of bipolar disorder (Butzlaff & Hooley, 1998, Miklowitz, Goldstein, Nuechterlein, Snyder, & Mintz, 1988; Miklowitz, Simoneau, Sachs-Ericsson, Warner, & Suddath, 1996; Priebe, Wildgrube, & Mueller-Oerlinghausen, 1989; Rosenfarb et al., 2001), even among medication-adherent participants (O'Connell, Mayo, Flatow, Cuthbertson, & O'Brien, 1991). Expressed emotion is particularly linked to bipolar depression, as opposed to mania (Miklowitz, Wisniewski, Miyahara, Otto, & Sachs, 2005; Yan, Hammen, Cohen, Daley, & Henry, 2004). Within bipolar disorder, poor outcome appears

Correspondence concerning this article should be addressed to: Amy K. Cuellar, Michael E. DeBakey VA Medical Center, 116MHCL, 2002 Holcombe Blvd., Houston, TX 77030; AmyK.Cuellar@va.gov.
Amy K. Cuellar is now at the Michael E. DeBakey VA Medical Center/The Menninger Department of Psychiatry and Behavioral Sciences, Baylor College of Medicine, Houston, TX.
Sheri L. Johnson is now at the Department of Psychology, The University of California, Berkeley.
Camilo J. Ruggero is now at the Department of Psychiatry and Human Behavior, The Warren Alpert Medical School of Brown University, Providence, RI.

most related to the criticism and hostility components of EE (de Jesus Mari & Streiner, 1994). The effects of EE are stronger on mood disorders than schizophrenia (Butzlaff & Hooley, 1998; Hooley & Hoffman, 1999). Relapse rates are predicted by fewer critical comments in mood disorders compared with schizophrenia, suggesting a greater sensitivity to EE in mood disorder. Such a heightened sensitivity might suggest there are unique elements of bipolar disorder that increase vulnerability to EE.

What aspects of the bipolar disorder diathesis make patients more vulnerable to criticism? With other psychopathologies, specific intrinsic vulnerabilities of particular disorders have helped to predict response to environmental stressors. For example, maladaptive negative cognitions predispose individuals to depression following a life event (Brown & Beck, 2002). Similarly, hypersensitivity to threat predicts anxiety (Finlay-Jones & Brown, 1991; Sookman, Pinard, & Beck, 2001) and hyperarousal or overstimulation in schizophrenia predicts relapse in response to life events (Pallanti, Quercioli, & Pazzagli, 1997; Tarrier & Turpin, 1992). In sum, studies of cognitive and affective processes have helped explain how environmental risk factors become translated into symptoms of a disorder.

One potential mechanism that could link EE to negative outcomes in bipolar disorder might be increased affective reactivity. Not only are episodes of the disorder characterized by extremes of emotion, but mood lability is a robust predictor of the onset of the disorder (Angst, Gamma, & Endrss, 2003). In addition, people with bipolar disorder appear to have greater mood dysregulation in response to daily stressful events (Myin-Germeys et al., 2003). Although affect dysregulation could also involve reactivity to positive events, because criticism is negatively valenced, we choose to focus on negative reactivity. Affective dysregulation might be evidenced in more pronounced affective responses to EE. Several reviews of the neurobiology of bipolar disorder have emphasized the involvement of brain regions involved in reactivity and regulation of emotions (Cuellar, Johnson, & Winters, 2005; Depue & Zald, 1993; Howland & Thase, 1999; Leibenluft, Charney, & Pine, 2003; Winters, Johnson, & Cuellar, 2008). Similarly, models of bipolar disorder continue to emphasize negative emotional reactivity as a potentially key behavioral expression of neurobiological risk (Hasler, Drevets, Gould, Gottesman, & Manji, 2006; Leibenluft et al., 2003; Phillips, Drevets, Rauch, & Lane, 2003). Despite the dominance of theories of emotion dysregulation, relatively few studies have been conducted to examine how people with bipolar disorder respond affectively to emotionally evocative stimuli (see Johnson, Gruber, & Eisner, 2007, for a review).

Studies of brain activation in response to negative stimuli have provided some, but not entirely consistent support, for deficits among people with remitted bipolar disorder. That is, although some researchers have found that compared to nonmood disordered participants, people with bipolar disorder respond to sad stimuli with greater brain activation in prefrontal cortex regions (Chang et al., 2004; Lawrence et al., 2004), other studies have found diminished activity in the prefrontal cortex among bipolar groups compared to nonbipolar groups in response to negative subtitles (Malhi, Ivanovski, & Szekeres, 2004) or sad mood (Kruger, Seminowicz, Goldaple, Kennedy, & Mayberg, 2003). Hence, the body of evidence concerning neural responses to negative stimuli in bipolar disorder remains fairly inconsistent.

Research focused on emotional reactivity has been conducted using analog samples of people at risk for bipolar disorder (as defined by elevated scores on measures of subsyndromal symptoms). Those studies have found no significant differences on self-reports, facial behavior, or psychophysiological measures in response to film clips (Gruber, Johnson, Oveis, & Keltner, 2008), eye blinks in response to negative pictures (Sutton & Johnson, 2002), or confidence ratings after a false failure feedback (Stern & Berrenberg, 1979). Two findings in support of greater reactivity to negative stimuli have emerged in studies with diagnosable bipolar disorder. That is, researchers have found that people diagnosed with bipolar spectrum

disorders demonstrate greater cognitive deficits in response to completing unsolvable anagrams (Ruggero & Johnson, 2006), and more prolonged cortisol dysregulation after completing difficult math tasks (Depue, Kleiman, Davis, Hutchinson, & Krauss, 1985). Although these studies are consistent with greater reactivity to negative stimuli, they are not specific demonstrations of emotional reactivity per se and they included participants with mild levels of depression at the time of testing. Hence, it remains unclear whether emotional reactivity to stressors can be documented among persons diagnosed with bipolar disorder during periods of remission.

Although variations in the study methods (e.g., functional magnetic resonance imaging [fMRI] outcome measures vs. self-reports or psychophysiological data, clinical vs. analog participants) might explain cross-study discrepancies, another possible reason for inconsistent findings is the types of emotion-eliciting stimuli used in these studies. Stimuli such as pictures, film clips, and faces might not evoke as much emotion as a stressor that is personally engaging and interpersonal in nature, given that interpersonal life events and family EE are particularly powerful predictors of the course of bipolar disorder (cf. Butzlaff & Hooley, 1998; Johnson & Roberts, 1995).

The goal of this study was to examine whether people with bipolar disorder demonstrate increased affective reactivity to criticism compared to a healthy control group. More specifically, we were interested in negative affective reactivity, given the previously established links between EE and depressive symptoms. In this study, we used a laboratory analog of EE: criticism from a confederate. Although we considered measuring affective reactions to actual family criticisms, presenting standardized criticisms allowed us to limit the confounds between bipolar and nonbipolar groups in the variables that produce criticism and responses to criticisms (e.g., patient characteristics) and cause reactions to criticism (e.g., family transactions). In addition, there is evidence to suggest that EE does not necessarily need to be expressed by a relative to influence course of illness (cf., Ball, Moore, & Kuipers, 1992). The present study tested two aspects of self-reported affective responses to a standardized criticism: reactivity and recovery. We hypothesized that (a) participants with bipolar disorder will display greater negative affective reactivity than control participants in response to criticism; and (b) among those who react to the criticism, participants with bipolar disorder compared to those in the control group will display a slower recovery slope of their negative affect (NA), as assessed in the 9 minutes after the criticism. The principal outcome measure of NA will be the negative affect scale created from the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1981).

Method

Participants

Two groups of participants were recruited: those with bipolar disorder in remission ($n = 35$) and those with no history of a mood disorder ($n = 35$). To increase the homogeneity of diagnosis, only participants with bipolar I disorder were recruited for the current study. Participants were recruited in the South Florida community through support groups and print and Internet advertisements. Participants (58.67% female) were between the ages of 19 and 61, with a mean age of 39.97 ($SD = 11.94$). Exclusion criteria for both groups included the presence of schizoaffective disorder, a psychotic disorder, a mood disorder due to a general medical condition, substance-induced mood disorder, or the presence of significant subsyndromal mood symptoms or a current mood episode (see the *Procedures* section for remission criteria). Because approximately 60% of people with bipolar disorder have had a history of substance abuse disorders (Cassidy, Ahearn, & Carroll, 2002) and we were invested in maintaining the generalizability of the current study, participants were not excluded based on having a comorbid substance use disorder. Nevertheless, all participants had diagnoses of a primary

mood disorder; substance-induced mood disorders and mood disorders due to a medical condition were excluded.

Procedures

Participants completed written informed consent. All diagnostic instruments were administered either by an advanced clinical psychology graduate student or a doctoral level psychologist. Diagnoses were established by interview using the mood disorder and psychosis modules of the Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1996). The Bech-Rafaelsen Mania Scale (BRMS; Bech, Bolwig, Kramp, & Rafaelsen, 1979) and the Modified Hamilton Rating Scale for Depression (MHRSD; Miller, Bishop, Norman, & Maddever, 1985) were used to select participants in remission, defined as BRMS less than 7 and MHRSD less than 10. Among participants with bipolar disorder, the adequacy of pharmacotherapy was assessed with the Somatotherapy Index (Bauer et al., 1997).

After participants were interviewed, they completed baseline self-report measures of mood and interpersonal support in the following order: demographic information, Interpersonal Support Evaluation List (ISEL; Cohen, Mermelstein, Kamarck, & Hoberman, 1985), Levels of Expressed Emotion (LEE; Gerlsma, van der Lubbe, & van Nieuwenhuizen, 1992), and the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1981).

Criticism analog—After completing baseline measures, participants received the analog criticism. Recent research has examined reactivity in mood disorders to maternal criticism (Hooley, Gruber, Scott, Hiller, & Yurgelun-Todd, 2005) or using familial interactions to examine transactional relationships between patients and relatives (Rosenfarb et al., 2001). Because we are interested in differences in reactivity to real world criticism, we had considered using family interactions as a stimulus. We decided that using a standardized critical comment from a stranger would allow for tighter comparisons across individuals that would allow us to control for differences in family members, family histories of interactions, and other variables that could elicit differences in reactivity.

We aimed to write a script that was critical enough to elicit a reaction, yet not so critical as to cause more than a temporary affective shift. To write a script that was sufficiently critical, but not overly so, we solicited input from experts in the EE field, the Institutional Review Board, and comments following pilot testing with undergraduate students. An initial, milder preliminary script was presented to experts in the EE field with instructions to provide feedback as to the realism of the script and how well it captured the elements of hostility or criticism in EE. The script also was pilot tested with undergraduate students who were asked to rate how critical the script was. The feedback from both experts in the field and the undergraduate students was that the original script needed to be made more critical to increase its external validity. The script used in the current study was created based on this feedback and was rated as being more realistic and sufficiently critical to achieve a reaction. The Institutional Review Board provided feedback that the criticism was not overly harsh as to elicit more than a temporary affective shift. As a result of this process, the following procedures were used.

Participants were told that they were being asked to participate in an experiment studying the ways people provide and receive emotional support. They were told that they had been assigned to the role of receiving emotional support. They were asked to think of a midrange difficulty that they had been experiencing lately. Examples of midrange difficulties, such as family, finances, housing, school, work, friendships, or romantic relationships were provided for guidance. To protect participants, they were asked not to talk about severe problems, such as death, abuse, physical disabilities, or mental illness. Participants most frequently chose to discuss interpersonal relationships (37.14%), followed closely by finances (28.57%).

Remaining topics (34.29%) included problems such as health, education, housing, work, legal, and other difficulties. A chi-square revealed no significant differences between the participants with bipolar disorder and the control participants on the topic selected for discussion, $\chi^2(2, 69) = 3.03$, $p = .22$.

The experimenter asked the participant to explain his or her difficulty to the confederate for 2 minutes. In the presence of the participant, the experimenter told the confederate that his or her role was to provide the participant with a supportive comment after the description. The rationale for telling the confederate beforehand that his or her role was to provide a supportive comment was to replicate what likely happens in EE: Those relationships that are seen as emotionally close typically have the expectation of being supportive. At the end of 2 minutes, the confederate provided a standardized response of wincing and stating, "I see this is a difficult situation for you, but I think you're overreacting. Maybe you need to focus on what you've done to help create this problem. Look, don't you think there are more important things to worry about than (participant's problem)? There are people with some real problems. Don't you think you're being a little bit selfish?" Confederates were upper-level undergraduate psychology students who were trained by one of the investigators (AKC) to deliver the criticism in a believable, standardized fashion. The confederates were trained until they were rated as comparable in level of criticism, tone of the criticism, and nonverbal gestures by the same investigator. Confederates included men and women, as well as various ethnic groups (e.g., Hispanic, Asian, African American, and White), and were randomly assigned to participants. Post hoc chi-square analyses showed no significant differences between participant gender in terms of confederate gender, $\chi^2(1, 68) = 0.60$, $p = .81$, or participant ethnicity and confederate ethnicity, $\chi^2(12, 68) = 13.21$, $p = .35$.

After the feedback and before completing the final mood assessment, participants completed poststimuli self-reports in the following order: the POMS for immediate mood state, rating of support from the confederate, the Perceived Criticism scale (PC; Hooley & Teasdale, 1989), the Rating of Support (ROS, designed for this study), and the Causal Dimension Scale II (CDS; McAuley, Duncan, & Russell, 1992). Nine minutes after the first postcriticism POMS, participants completed the POMS again, regardless of which other questionnaires were completed or whether all the questionnaires had been completed.

At the end of the experiment, the experimenter fully debriefed participants about the experiment, including the nature of deceptive elements. After the debriefing, the confederate interacted with the participant, emphasizing that that the feedback given did not reflect his or her true feelings about the participant and provided actual supportive comments regarding the participant's problem.

Measures

Several measures were gathered to determine whether participants met the inclusion and exclusion criteria for the study.

Structured Clinical Interview for DSM-IV (SCID; First et al., 1996)—Diagnostic status was assessed using the mood and psychosis modules of the SCID, one of the most widely used standardized diagnostic interview instruments. The SCID coverage includes the age of onset and number of lifetime episodes of both mania and depression. Episodes that were secondary to medical concerns or substance abuse did not meet inclusion criteria in the current study. The experimenter was trained to reliability prior to the recruitment of participants in the current study. Interrater reliability for the current study was established by randomly selecting three tapes for review by the research team. Interrater reliability for four raters was high for the diagnosis of bipolar disorder ($\kappa = .94$).

Bech-Rafaelsen Mania Scale (BRMS; Bech et al., 1979)—Severity of mania during the past week was assessed using the BRMS, one of the most widely used instruments to assess symptoms of mania (Bech, 2002). Bech recommends a cutoff score of ≥10 to indicate hypomania. To assure that participants had no significant current mood symptoms, we decided to use a stricter criterion for remission. Participants who reported significant symptoms on the BRMS (≥7) were excluded from participation in the study. The 11 items on this scale cover symptoms such as flight of ideas, elevated mood, decreased need for sleep, and heightened sexual interest. The BRMS was administered using a standardized interview format, with each item rated from 0 (not present) to 4 (severe) using behavioral anchors for each scale point (Johnson & Miller, 1997). Reliability for the current study was high for three randomly selected interviews for four raters (interclass correlation = .92). The BRMS also is a highly sensitive instrument, detecting naturalistic and treatment-related changes in mania (Bech, 2002).

Modified Hamilton Rating Scale for Depression (MHRSD; Miller et al., 1985)—Severity of depressive symptoms in the past week was measured using the MHRSD, a commonly used interview designed to reliably and validly rate changes in depressive symptoms. Participants who reported significant symptoms on the MHRSD (≥10) were excluded from participation in the study. The 17-item interview assesses symptoms such as depressed mood, guilt, and insomnia and is highly correlated with the original HRSD. Interrater reliability for four raters on three tapes in the current study was high, ICC = .97, and individual item reliability greater than that for the original HRSD. The MHRSD is correlated with alternate measures of depression, with an interclass correlation of .84 with the original HRSD. In addition, the MHRSD is sensitive to changes in symptoms (Miller et al., 1985).

Rating of Support (ROS)—The ROS was a self-report measure designed for the current study to measure the perceived criticism of the confederate's comments to examine whether the participant thought that the stimulus was credible. The ROS included four questions concerning the supportiveness of the comments, the criticism of the comments (reverse scored), perceived liking of the participant by the confederate, and perceived dislike of the participant by the confederate (reverse scored). Each item was rated on a 10-item Likert scale. Internal consistency for the ROS was $\alpha = .73$ in the current sample.

Profile of Mood States-Short Version (POMS; Shacham, 1983)—The short version of the POMS, a 37-item self-report measure of affect, was used to assess mood state at baseline, immediately after the confederate's comments, and after a 9-minute delay. The POMS was the main outcome variable for analyses. For each word, participants were asked to describe how much they are experiencing the emotion “right now” on a scale of 0 (not at all)to 4 (extremely). The instrument includes six different subscales (depression, vigor, confusion, tension, anger, and fatigue). For the purposes of this study, the depression, tension, and anger subscales were combined to form a measure of NA. The negative affect scale achieved excellent internal consistency, with internal consistencies for Time 1 = .97, Time 2 = .97, and Time 3 = .96.

Somatotherapy index (Bauer et al., 1997)—Pharmacological treatment was assessed by using the Somatotherapy Index, a 6-point rating scale designed to assess the treatment for bipolar disorder. The Somatotherapy Index was coded as a potential confound of affective dysregulation within the bipolar sample if type of medication regimen is a significant predictor of affect. Coding was based on information provided by the participant in an interview format, which included the medication names, dosages, rates of compliance, and latest blood serum levels. High interrater reliability has been reported, a = .96 (Bauer et al., 1997).

Interpersonal Support Evaluation List (ISEL; Cohen et al., 1985)—Participants completed the self-esteem support subscale of the ISEL, a self-report measure designed to

assess perceived social support. This measure was used to control for the potential confound of between-group differences in quality of social support. The self-esteem support subscale was selected because it was the only subscale predictive of relapse in a longitudinal study of bipolar disorder (Johnson, Meyer, Winett, & Small, 2000). An example item from the self-esteem support subscale includes, "There is someone who takes pride in my accomplishments." The subscale consists of 10 items that are rated on a 4-point Likert scale, with anchors ranging from definitely false to definitely true. The ISEL has a demonstrated 6-month test-retest reliability of .74 (Cohen et al., 1985), and internal consistency in the current sample was high ($\alpha = .80$).

Level of Expressed Emotion Scale (LEE; Gerlsma et al., 1992)—The LEE is a self-report measure of perceived family EE. The LEE was used to control for the potential confound of between-group differences in family EE. Although the original LEE was 60 true/false items (Cole & Kazarian, 1988), Gerlsma and colleagues (1992) performed a factor analysis on the LEE and developed a 33-item version, which eliminated items that did not load onto one of three factors: lack of emotional support, intrusiveness/control, and irritability. In the 33-item version used in the current study (Gerlsma et al., 1992), items are rated on a 4-point Likert scale and scores include total perceived EE, as well as the subscales lack of emotional support, intrusiveness/control, and irritability. Reported 2-month test-retest reliabilities for the subscales range from .74 to .83 (Startup, 1999). The LEE has been shown to predict symptoms of depression at baseline and at a 6-month follow up (Gerlsma & Hale, 1997). Internal consistency for the current sample is high, with $\alpha = .92$ for the total perceived EE, $\alpha = .89$ for lack of emotional support, $\alpha = .73$ for intrusiveness/ control, and $\alpha = .81$ for irritability.

Perceived Criticism (PC; Hooley & Teasdale, 1989)—The LEE does not measure an important component of EE—criticism. The PC, a measure of family criticism, has been found to be more predictive of relapse in depression than either EE as measured by the Camberwell Family Interview or marital distress (Hooley & Teasdale, 1989). The PC is a self-report measure with two items rated on a 10-point Likert scale to assess how critical the participant considers his or her closest other to be of him or her and how critical the participant considers him- or herself to be of the closest other.

Causal Dimension Scale-II (CDS-II; McAuley et al., 1992)—To measure self-blaming attributional style as a potential confound, participants rated the cause of the confederate's comments on a modified version of the CDS-II, a self-report measure. First, participants are asked to write the cause of the confederate's reaction and whether that feedback was fair. Next, participants rated how critical the feedback was and how legitimate the feedback was on a 10-point Likert scale. Four modified CDS-II items rated on a 9-point Likert scale followed. All items begin with a prefix, such as "Is the critical part of the feedback," with anchors including *caused by your behavior* at one end of the Likert scale and *caused by something about his/her personality* at the other end of the scale. Internal consistency for the four modified CDS-II items in this study was $\alpha = .90$.

Results

Preliminary Analyses

Prior to testing primary hypotheses, preliminary data analyses were carried out to ensure assumptions were met and to screen for possible outliers. Outlier analyses did not suggest the need to remove any participant from subsequent tests. All assumptions regarding the primary analyses (e.g., normal distributions) were met. For all analyses, α was set to .05 and two-tailed tests were used.

Analyses were conducted to assess potential confounds that could predict mood reactivity and recovery. No significant differences were found between groups on level of education, $t(65) = 1.24$, $p = .22$; gender, $\chi^2(1, 70) = .06$, $p = .81$; or family role, $\chi^2(3, 64) = 2.74$, $p = .43$. In addition to the demographic items, no significant differences exist between groups on ratings of support, $t(67) = -1.00$, $p = .32$; family intrusiveness, $t(67) = -.09$, $p = .93$; current depressive symptoms, $t(67) = -1.40$, $p = .17$; or the believability of the feedback, $t(65) = 1.22$, $p = .23$. Although the bipolar group had significantly greater current manic symptoms, $t(67) = -3.77$, $p<.001$, there was no significant correlation between manic symptoms and mood reactivity, $r = .12$, $p<.31$.

The Somatotherapy Index was used to evaluate the types of psychiatric medication used. Medications were analyzed categorically by type (i.e., lithium, anticonvulsant, antidepressant, antipsychotic, and benzodiazepine). No psychiatric medication was a significant predictor of mood recovery or reactivity. Other variables considered as potential confounds included demographic variables, current mood symptoms, and variables relevant to relationships, such as the ISEL, LEE, PC, and CDS. To be considered a confound, the variable must have been associated with both the outcomes (i.e., mood reactivity or recovery) and it must have been associated with group. No variables met both criteria, so none were considered confounds.

Preliminary analyses were also conducted to examine if there were differences in baseline NA before the experimental manipulation. Participants with bipolar disorder reported greater NA than the control participants did at baseline, $t(68) = -2.70$, $p = .01$, as well as at Time 2, $t(67) = -3.71$, $p<.001$.

Finally, an experimental manipulation check was carried out to determine if the criticism influenced mood. Matched *t* tests indicated significantly greater NA, $t(68) = -2.17$, $p = .03$, and significantly less vigor (or positive affect), $t(68) = 4.19$, $p<.0001$, after the criticism compared to before, suggesting that the criticism was effective in influencing mood. Of note, 60% of the sample reacted to the criticism with increased NA, and 69% of the sample reacted to the criticism with decreased vigor. Of those who reacted with greater NA, slightly greater than half (54.76%) had a diagnosis of bipolar disorder. Similarly, nearly half (52.08%) of those who reacted with decreased vigor had a diagnosis of bipolar disorder.[1] In addition, the ROS was used to determine whether the standardized criticism was seen as critical. In a paired-sample *t* test, participants rated the criticism as significantly more criticizing than supportive, $t(69) = -6.54$, $p<.0001$. Partial correlations also were examined between affect immediately after the stimulus and the ROS while controlling for baseline affect. Negative affect at Time 2 after controlling for NA at Time 1 was significantly correlated with the ROS ($r = .58$, $p<.006$), suggesting that those who found the criticism more convincing were more likely to have a negative reaction.

Analysis Plan for Tests of Primary Hypotheses

To test hypotheses, a piecewise hierarchical linear model (HLM) was estimated (Raudenbush, Bryk, & Congdon, 2004). This model allowed us to model our two hypotheses, that is, that groups would differ on the initial negative affective reaction to the criticism as well the slope of recovery to baseline NA after the initial reaction. The negative affect scale from POMS was the principal outcome for these analyses.

Our piecewise HLM model had two levels of equations. The Level 1 equation for this model estimates a change in NA as a function of time for each person. Affect was measured at three time points: baseline (Time 1), immediately after the criticism (Time 2), and at 9 minutes after

[1]As noted earlier, 33% of the sample did not show an expected change in vigor following the criticism. Of these, 22% showed a paradoxical increase in vigor. There was no significant difference between the bipolar and control groups on vigor reactivity or recovery slopes.

the criticism (Time 3). The Level 1 equation was $NA_{ti} = \pi_{i0} - \pi_{i1}$ (time unit)+ π_{i2} (time unit) $+e_{ti}$. In this equation, NA reflects the score of person *i* at time *t*. The first coefficient (π_{i1}) represents the change in NA between baseline (Time 1) and just after the criticism (Time 2). The second coefficient (π_{2i}) represents the change in NA scores from immediately after the criticism (Time 2) to 9 minutes after the criticism (Time 3).

The Level 2 equations were estimated to explore how having a diagnosis affects the slope of the initial reaction to the criticism (π_{i1}) as well as the slope of the recovery from the criticism (π_{2i}). These consisted of the following three equations: $\pi_{i0} = \beta_{00} + \beta_{01}$ (group)$+r_0$ for the intercept equation, $\pi_{i1} = \beta_{10} + \beta_{11}$ (group)$+r_1$ for the slope of initial reactivity, and $\pi_{i2} = \beta_{20} + \beta_{21}$ (group)$+_r$ for the slope of the recovery. In each equation, group membership (i.e., group with bipolar coded 1 versus control coded 0) is used to predict the Level 1 slopes (as well as the intercept). Hypotheses focused on whether the Level 2 coefficient for diagnostic group affected either the slope of the initial reaction or the slope of recovery.

Some participants did not react to the criticism (see below for results). Because of this, we estimated the piecewise model twice. For the first model, we included all participants to evaluate group differences in the initial reaction to the criticism. The second model was used to consider recovery from an initial reaction. Because it would be invalid to consider recovery among participants who never reacted to the criticism, the second model was estimated after excluding participants who did not react to the criticism. Hence, two models are presented: first, to examine group differences in the slope of reactivity and second, to consider group differences in the slope of recovery among those who reacted.

Initial Reaction to Criticism

As shown in Table 1, there was a trend for participant diagnosis (group) to be a predictor of the slope of the initial reaction to the criticism. Specifically, the bipolar group reacted to the criticism with greater NA than the control group (i.e., the slope of their NA reaction was steeper). This trend is of note, given that the bipolar group reported higher levels of negative affect before the experiment started.

Analyses of Recovery

As mentioned, 40% of participants did not show an affective reaction to the criticism (i.e., that is, NA after the criticism was higher, 30%, or equal to 10%, what it was before the criticism[1]). After deleting nonreactive participants, the data set included 42 participants, of whom 23 belonged to the bipolar group (66% of original bipolar sample) and 19 to the control group (54% of original control sample). There was no significant difference in reactivity between the bipolar and control group in terms of the proportion of participants who reacted to the criticism, $\chi^2(1, 70) = .95$, $p = .33$, or the magnitude of their reactivity, $t(68) = -.97$, $p = .34$. Although the remaining participants each evidenced increased NA after the criticism, they varied in the degree of reactivity. Accordingly, initial reactivity was controlled for in considering slopes of recovery.

The Level 1 and Level 2 equations used for this model were similar to the ones used above with the difference being that we now shift the focus of analysis to the slope of recovery rather than initial reactivity. As shown in Table 2, before correcting for differences in initial reactivity, the bipolar group appears to have a steeper slope of recovery. However, this group difference was explained by differences in the initial reactivity: after controlling for reactivity, there was no group difference in the slope of recovery, $t(68) = 0.01$, $p = 0.99$.

Exploratory Analyses: Predictors of Reactivity

Because of the importance of predicting what characteristics cause people with bipolar disorder to be more reactive to criticism, exploratory analyses were carried out to determine which variables predicted reactivity. First, analyses were conducted to determine whether the people who reacted perceived the criticism as more legitimate or reported different attributions for the criticism on the CDS. Of note is that participants who reacted to the criticism did not differ significantly on their perception of the legitimacy of the criticism or on attributions of responsibility for the critical feedback.

We next conducted stepwise discriminant function analysis to identify potential individual differences that predicted reactivity within the bipolar group. Using stepwise selection, five variables entered the model as predictors of reactivity. The whole model was a significant predictor of reactivity, $\lambda = .24$, $\chi^2(4) = 33.80$, $p<.0001$. The five variables entered were the following (standardized coefficient weights in parentheses): gender (.50), perceived criticism of participant's closest other (.61), ROS (1.16), disability status (.71) and LEE Lack of Support subscale (−.84). Eighty-eight percent of the sample was correctly classified based on these variables.

Discussion

It is well documented that high levels of family criticism predict poor outcome in bipolar disorder. The current study examines whether people with remitted bipolar I disorder demonstrate more affective dysregulation in the face of criticism compared to those without bipolar disorder. Participants were exposed to a standardized criticism, and their initial affective reactions, as well as their recovery from those reactions, were examined. Participants with bipolar disorder and those without bipolar disorder did not differ significantly in their initial reactions or their recovery after receiving a criticism. That is, current findings suggested only a nonsignificant trend for the participants with bipolar disorder to have a stronger negative affective reaction to criticism than the control participants. Group differences did not reach the threshold for statistical significance.

Only a subset of participants demonstrated reactivity. What could cause some participants with bipolar disorder to be more reactive to the criticism? A first thought might be that the participants who reacted to the criticism saw the feedback as more valid. Nevertheless, those who reacted to the criticism did not report that they found the feedback to be more legitimate nor did they perceive themselves to be more responsible for the criticism. Within the bipolar group, there were four predictors of greater reactivity. The first such predictor included interpreting the criticism as more critical, which supports the well-researched attribution theory (Abramson, Metalksy, & Alloy, 1989; Abramson, Seligman, & Teasdale, 1978; Seligman, Abramson, Semmel, & von Baeyer, 1979). The finding that women with bipolar disorder were more reactive mirrors the findings that women in the general population show greater psychological reactivity to hostile marital interactions (Kiecolt-Glaser, Malarkey, Cacioppo, & Glaser, 1994) and a greater vulnerability to depression (see Nolen-Hoeksema, 2002). Surprisingly, we also found that less reactivity to criticism was associated with *less* familial supportiveness as measured by the LEE. Although this finding might be attributable to participants becoming inured to criticism due to experiencing it on a regular basis with family, it is difficult to integrate with the large body of findings suggesting that family EE leads to greater relapse (Butzlaff & Hooley, 1998, Miklowitz et al., 1988, ***1996; Rosenfarb et al., 2001; Priebe et al., 1989). Finally, participants in the bipolar group who were receiving disability payments were likely to be more reactive to criticism, consistent with previous findings that severity of psychopathology predicts perceptions of family criticism (Chambless, Bryan, Aiken, Steketee, & Hooley, 2001). Given that analyses of individual differences in

reactivity were post hoc, there is a need for replication. In addition, it must be noted that none of these differences are necessarily specific to bipolar disorder.

In sum, individuals with bipolar disorder did not appear to be more reactive to criticism than controls were. However, a subset of people with bipolar disorder was more sensitive to criticism. It remains to be seen whether people who were more sensitive to this analog of family criticism would be at greater risk of relapse when faced with high EE within a family context.

The absence of greater reactivity within the bipolar group as a whole is somewhat surprising, given the propensity for people with bipolar disorder to experience relapses in the face of family criticism. Difficulty identifying group differences in this study might relate to several methodological issues. First, for ethical reasons, the current study measured reaction to a mild analog—a short-lived criticism from a stranger. It may be easier to dismiss remarks from a stranger than chronic criticism from family members. Indeed, 40% of participants did not react to the criticism, and many participants verbalized thoughts about why the confederate's statements were so negative that included attributions about the confederate's mood, personality, and even in that the confederate must be mentally ill.

Although some might wonder about using a more serious stressor, we feel compelled to argue against this. Most participants, regardless of diagnostic status, reported full recovery of their mood within the first 9 minutes after hearing the criticism within this study. Nevertheless, one incident points toward possible mood effects over a longer period of time, even though several steps were taken to protect participants, such as evaluating mood state after debriefing and providing instructions to call the research team if distress emerged. Despite these attempts to protect participants, one participant informed the investigators weeks after his participation that he developed paranoid ideation the day after, even though he denied NA 9 minutes after the criticism. Although the negative incident appears to have been an idiosyncratic response, it points to the possibility that the effects of criticism might be quite different if measured over a longer period.[2] That is, the effects of criticism might take days rather than minutes to emerge, and so understanding these effects might require employing a daily diary approach (Bolger & Zuckerman, 1995). It is also impossible to rule out the opposite, that differences in recovery occur much more rapidly than the 9-minute follow-up used in this study.

Beyond concerns about the mildness of the criticism and the time frame, reported NA may not be the most sensitive outcome variable. Other researchers have found evidence that cortisol (Depue et al., 1985) or cognitive performance (Elliott, Sahakian, Herrod, Robbins, & Paykel, 1997; Ruggero & Johnson, 2005) are particularly sensitive indices of stress reactivity within bipolar disorder. Indeed, across studies of emotional reactivity, researchers have been able to document that people with bipolar disorder demonstrate elevated stress reactivity in cognitive, biological, and behavioral measures, but to date, have shown no effects for self-reported affect (see Johnson et al., 2007). Perhaps biological or cognitive outcome variables would provide more sensitive indices of stress reactivity within bipolar disorder.

It should also be noted that heightened emotional reactivity to stressors within bipolar disorder has only been documented when studies included participants with current symptoms of depression (Johnson et al., 2007). We deliberately excluded participants with even mild depressive symptoms. By doing so, we may have excluded the very people who might have

[2]The incident was reported to the Institutional Review Board and all input was taken about the best ethical practices to follow. Two of the experimenters (AKC and SLJ) met with the participant to alleviate any remaining distress, and then took further precautions to ensure the participant's safety and well-being. We contacted all participants with bipolar disorder to check for negative sequelae of participation. No other participant reported negative effects of the study, and seven participants reported receiving significant benefit. We decided that it would be important to note this concern in the literature, as we believe other researchers should use caution if considering implementing stress paradigms to study bipolar disorder.

been most reactive to criticism. We also did not assess comorbid conditions, which could be related to reactivity.

In sum, a set of methodological issues might have limited the ability to observe reactivity within this study. Some of these issues stem directly from attempts to strengthen the study by using a tightly controlled analog design and excluding participants with mild symptoms. Nonetheless, the small number of people demonstrating reactivity limited ability to study group differences, and limited statistical power to examine the slope of recovery.

Conclusion

The current study is unique in its attempt to examine affect dysregulation as a potential vulnerability to the effects of EE. Although there is ample evidence that EE leads to relapse, people with bipolar disorder did not show elevated reactivity to criticism in the current study. Although an increasing range of theory and evidence suggests that brain regions that are integrally tied to emotion are involved in bipolar disorder, current results join a host of previous findings that did not support negative emotional reactivity as a behavioral correlate of bipolar disorder during periods of remission (Chang et al., 2004; Gruber et al., 2008; Stern & Berrenberg, 1979; Sutton & Johnson, 2002). Although this may reflect methodological issues, it also might be that affective dysregulation can only be demonstrated in a subset of people with bipolar disorder, suggesting that EE could be a moderator of course in bipolar disorder. Several participant and stressor variables should be examined in future research. Given that variables such as disability status, gender, and less positive close relationships are significant predictors of reactivity, future research should directly measure these parameters as potential predictors of sensitivity to EE. If confirmed, understanding individual differences in emotional reactivity with bipolar disorder could have significant implications for designing psychosocial treatments for this disorder.

Acknowledgments

We thank Ryan Joseph-Smerilson, Keith Walker, Kenia Gomez, Jason Esteves, and Kelli Yee for their assistance as the confederates in this study.

References

Abramson LY, Metalksy GI, Alloy LB. Hopelessness depression: A theory-based subtype of depression. Psychological Review 1989;96:358–372.

Abramson LY, Seligman ME, Teasdale JD. Learned helplessness in humans: Critique and reformulation. Journal of Abnormal Psychology 1978;87:49–74. [PubMed: 649856]

Angst J, Gamma A, Endrss J. Risk factors for the bipolar and depression spectra. Acta Psychiatrica Scandinavica 2003;108:15–19. [PubMed: 12807372]

Ball RA, Moore E, Kuipers L. Expressed emotion in community care staff: A comparison of patient outcome in a nine month follow-up of two hostels. Social Psychiatry and Psychiatric Epidemiology 1992;27:35–39. [PubMed: 1557680]

Bauer MS, McBride L, Shea N, Gavin C, Holden F, Kendall S. Impact of an easy-access VA clinic-based program for patients with bipolar disorder. Psychiatric Services 1997;48:491–496. [PubMed: 9090732]

Bech P. The Bech-Rafaelsen Mania Scale in clinical trials of therapies for bipolar disorder: A 20-year review of its use as an outcome measure. CNS Drugs 2002;16:47–63. [PubMed: 11772118]

Bech P, Bolwig TG, Kramp P, Rafaelsen OJ. The Bech-Rafaelsen Mania Scale and the Hamilton Depression Scale: Evaluation of homogeneity and inter-observer reliability. Acta Psychiatrica Scandinavica 1979;59:420–430. [PubMed: 433633]

Bolger N, Zuckerman A. A framework for studying personality in the stress process. Journal of Personality and Social Psychology 1995;69:890–902. [PubMed: 7473036]

Brown, GP.; Beck, AT. Dysfunctional attitudes, perfectionism, and models of vulnerability to depression. In: Flett, GL.; Hewitt, PL., editors. Perfectionism: Theory, research, and treatment. American Psychological Association; Washington, DC: 2002. p. 231-251.

Butzlaff RL, Hooley JM. Expressed emotion and psychiatric relapse. Archives of General Psychiatry 1998;55:547–552. [PubMed: 9633674]

Cassidy F, Ahearn EP, Carroll BJ. Substance abuse in bipolar disorder. Bipolar Disorders 2002;3:181–188. [PubMed: 11552957]

Chambless DL, Bryan AD, Aiken LS, Steketee G, Hooley JM. Predicting expressed emotion: A study with families of obsessive-compulsive and agoraphobic outpatients. Journal of Family Psychology 2001;15:225–240. [PubMed: 11458630]

Chang K, Adleman NE, Dienes K, Simeonova DI, Menon V, Reiss A. Anomalous prefrontal-subcortical activation in familial pediatric bipolar disorder: A functional magnetic resonance imaging investigation. Archives of General Psychiatry 2004;61:781–792. [PubMed: 15289277]

Cohen, S.; Mermelstein, R.; Kamarck, T.; Hoberman, HM. Measuring the functional components of social support. In: Sarason, IB.; Sarason, BR., editors. Social support: Theory, Research, and applications. Martinus Nijhof; The Hague, The Netherlands: 1985.

Cole JD, Kazarian SS. The Level of Expressed Emotion Scale: A new measure of expressed emotion. Journal of Clinical Psychology 1988;44:392–397. [PubMed: 3384966]

Cuellar AK, Johnson SL, Winters R. Distinctions between bipolar and unipolar depression. Clinical Psychology Review 2005;25:307–339. [PubMed: 15792852]

de Jesus Mari J, Streiner DL. An overview of family interventions and relapse on schizophrenia: Meta-analysis of research findings. Psychological Medicine 1994;24:565–578. [PubMed: 7991739]

Depue RA, Kleiman RM, Davis P, Hutchinson M, Krauss SP. The behavioral high-risk paradigm and bipolar affective disorder: VIII. Serum free cortisol in nonpatient cyclothymic subjects selected by the General Behavior Inventory. American Journal of Psychiatry 1985;142:175–181. [PubMed: 3970242]

Depue, RA.; Zald, DH. Biological and environmental processes in nonpsychotic psychopathology: A neurobehavioral perspective. In: Costello, CG., editor. Basic issues in psychopathology. Guilford Press; New York: 1993. p. 127-237.

Elliott R, Sahakian BJ, Herrod JJ, Robbins TW, Paykel ES. Abnormal response to negative feedback in unipolar depression: Evidence for a diagnosis specific impairment. Journal of Neurology, Neurosurgery, and Psychiatry 1997;63:74–82.

Finlay-Jones R, Brown GW. Types of stressful event and the onset of anxiety and depressive disorders. Psychological Medicine 1991;11:803–815. [PubMed: 7323236]

First MB, Spitzer RL, Gibbon M, Williams JBW. Structured Clinical Interview for DSM-IV Axis I Disorders-Patient (Edition (SCID-I-I/P Version 2.0)). 1996

Gerlsma C, Hale WW III. Predictive power and construct validity of the Level of Expressed Emotion (LEE) scale: Depressed out-patients and couples from the general community. British Journal of Psychiatry 1997;170:520–525. [PubMed: 9330017]

Gerlsma C, van der Lubbe PM, van Nieuwenhuizen C. Factor analysis of the Level of Expressed Emotion Scale, a questionnaire intended to measure “perceived expressed emotion”. British Journal of Psychiatry 1992;160:385–389. [PubMed: 1562866]

Gitlin MJ, Swendsen J, Heller TL, Hammen C. Relapse and impairment in bipolar disorder. American Journal of Psychiatry 1995;152:1635–1640. [PubMed: 7485627]

Goldberg, JF. The changing landscape of psychopharmacology for bipolar disorder. In: Johnson, SL.; Leahy, R., editors. Psychological treatments for bipolar disorder. Guilford Press; New York: 2004.

Gruber J, Johnson SL, Oveis C, Keltner D. Risk for mania and positive emotional responding: Too much of a good thing? Emotion 2008;8:23–33. [PubMed: 18266513]

Hasler G, Drevets WC, Gould TD, Gottesman II, Manji HK. Toward constructing an endophenotype strategy for bipolar disorders. Biological Psychiatry 2006;60:93–105. [PubMed: 16406007]

Hooley JM, Hoffman PD. Expressed emotion and clinical outcome in borderline personality disorder. American Journal of Psychiatry 1999;156:1557–1562. [PubMed: 10518166]

Hooley JM, Gruber SA, Scott LA, Hiller JB, Yurgelun-Todd DA. Activation in dorsolateral prefrontal cortex in response to maternal criticism and praise in recovered depressed and healthy control participants. Biological Psychiatry 2005;57:809–812. [PubMed: 15820239]

Hooley JM, Teasdale JD. Predictors of relapse in unipolar depressives: Expressed emotion, marital distress, and perceived criticism. Journal of Abnormal Psychology 1989;98:229–235. [PubMed: 2768657]

Howland, RH.; Thase, ME. Affective disorders: Biological aspects. In: Millon, T.; Blaney, PH.; Davis, RD., editors. xford textbook of psychopathology. Oxford University Press; London: 1999. p. 166-202.

Johnson, SL.; Gruber, J.; Eisner, LR. Emotion and bipolar disorder. In: Rottenberg, J.; Johnson, SL., editors. Emotion and psychopathology: Bridging affective and clinical science. American Psychological Association; Washington, DC: 2007. p. 123-150.

Johnson SL, Meyer B, Winett C, Small J. Social support and self-esteem predict changes in bipolar depression but not mania. Journal of Affective Disorders 2000;58:79–86. [PubMed: 10760562]

Johnson SL, Miller I. Negative life events and time to recovery from episodes of bipolar disorder. Journal of Abnormal Psychology 1997;106:449–457. [PubMed: 9241946]

Johnson SL, Roberts JE. Life events and bipolar disorder: Implications from biological theories. Psychological Bulletin 1995;117:434–449. [PubMed: 7777648]

Keller MB, Lavori PW, Coryell W, Endicott J, Mueller TI. Bipolar I: A five-year prospective follow up. Journal of Nervous and Mental Disease 1993;181:238–245. [PubMed: 8097229]

Kiecolt-Glaser, JK.; Malarkey, WB.; Cacioppo, JT.; Glaser, R. Stressful personal relationships: Immune and endocrine function. In: Kiecolt-Glaser, JK.; Glaser, R., editors. Handbook of Human Stress and Immunity. Academic Press; San Diego: 1994. p. 321-339.

Kruger S, Seminowicz D, Goldaple K, Kennedy SH, Mayberg HS. State and trait influences on mood regulation in bipolar disorder: Blood flow differences with an acute mood challenge. Biological Psychiatry 2003;54:1274–1283. [PubMed: 14643095]

Lawrence NS, Williams AM, Surguladze S, Giampietro V, Brammer MJ, Andrew C, et al. Subcortical and ventral prefrontal cortical neural responses to facial expressions distinguish patients with bipolar disorder and major depression. Biological Psychiatry 2004;55:578–587. [PubMed: 15013826]

Leibenluft E, Charney DS, Pine DS. Researching the pathophysiology of pediatric bipolar disorder. Biological Psychiatry 2003;53:1009–1020. [PubMed: 12788246]

Malhi GS, Ivanovski B, Szekeres V. Bipolar disorder: It's all in your mind? The neuropsychological profile of a biological disorder. Canadian Journal of Psychiatry 2004;49:813–819.

McAuley E, Duncan TE, Russell DW. Measuring causal attributions: The revised Causal Dimension Scale (CDSII). Personality and Social Psychology Bulletin 1992;18:566–573.

McNair, PM.; Lorr, M.; Droppleman, LF. POMS Manual. 2nd ed.. Educational and Industrial Testing Service; San Diego: 1981.

Miklowitz D, Goldstein MJ, Nuechterlein K, Snyder K, Mintz J. Family factors and the course of bipolar affective disorder. Archives of General Psychiatry 1988;45:225–231. [PubMed: 3341878]

Miklowitz, DJ.; Simoneau, TL.; Sachs-Ericsson, N.; Warner, R.; Suddath, R. Family risk indicators in the course of bipolar affective disorder. In: Mundt, C.; Goldstein, MJ.; Hahlweg, K.; Fiedler, P., editors. Interpersonal factors in the origin and course of affective disorders. Gaskell/Royal College of Psychiatrists; London: 1996. p. 204-217.

Miklowitz DJ, Wisniewski SR, Miyahara S, Otto MW, Sachs GS. Perceived criticism from family members as a predictor of the one-year course of bipolar disorder. Psychiatry Research 2005;136:101–111. [PubMed: 16023735]

Miller IV, Bishop SB, Norman WH, Maddever H. The Modified Hamilton Rating Scale for Depression: Reliability and validity. Psychiatry Research 1985;14:131–142. [PubMed: 3857653]

Myin-Germeys I, Peeters F, Havermans R, Nicolson NA, deVries MW, Delespaul P, et al. Emotional reactivity to daily life stress in psychosis and affective disorder: an experience sampling study. Acta Psychiatrica Scandinavica 2003;107:124–131. [PubMed: 12534438]

Nolen-Hoeksema, S. Gender differences in depression. In: Gotlib, IH.; Hammen, CL., editors. Handbook of depression. Guilford Press; New York: 2002. p. 492-509.

O'Connell RA, Mayo JA, Flatow L, Cuthbertson B, O'Brien BE. Outcome of bipolar disorder on long-term treatment with lithium. British Journal of Psychiatry 1991;159:123–129. [PubMed: 1888958]

Pallanti S, Quercioli L, Pazzagli A. Relapse in young paranoid schizophrenic patients: A prospective study of stressful life events, P300 measures, and coping. American Journal of Psychiatry 1997;154:792–798. [PubMed: 9167506]

Phillips ML, Drevets WC, Rauch SL, Lane R. Neurobiology of emotion perception I: The neural basis of normal emotion perception. Biological Psychiatry 2003;54:504–514. [PubMed: 12946879]

Priebe S, Wildgrube C, Mueller-Oerlinghausen B. Lithium prophylaxis and expressed emotion. British Journal of Psychiatry 1989;154:396–399. [PubMed: 2597843]

Prien RF, Potter WZ. NIMH workshop report on treatment of bipolar disorder. Psychopharmacology Bulletin 1990;26:409–427. [PubMed: 2087538]

Raudenbush, SW.; Bryk, A.; Congdon, R. HLM 6: Hierarchical linear and nonlinear modeling. Scientific Software International; Lincolnwood, IL: 2004.

Rosenfarb IS, Miklowitz DJ, Goldstein MJ, Harmon L, Nuechterlein KH, Rea MM. Family transactions and relapse in bipolar disorder. Family Process 2001;40:5–14. [PubMed: 11288369]

Ruggero CJ, Johnson SL. Reactivity to a laboratory stressor among individuals with Bipolar I disorder in full or partial remission. Journal of Abnormal Psychology 2006;115:539–544. [PubMed: 16866593]

Seligman ME, Abramson LY, Semmel A, von Baeyer C. Depressive attributional style. Journal of Abnormal Psychology 1979;88:242–247. [PubMed: 500951]

Shacham S. A shortened version of the Profile of Mood States. Journal of Personality Assessment 1983;47:305–306. [PubMed: 6886962]

Sookman D, Pinard G, Beck AT. Vulnerability schemas in obsessive-compulsive disorder. Journal of Cognitive Psychotherapy 2001;15:109–130.

Startup M. Confirmatory factor analysis of the Level of Expressed Emotion (LEE) scale. British Journal of Medical Psychology 1999;72:421–424. [PubMed: 10524725]

Stern GS, Berrenberg JL. Skill-set, success outcome, and mania as determinants of the illusion of control. Journal of Research in Personality 1979;13:206–220.

Sutton SK, Johnson SJ. Hypomanic tendencies predict lower startle magnitudes during pleasant pictures. Psychophysiology 2002;39(Suppl):580.

Tarrier N, Turpin G. Psychosocial factors, arousal and schizophrenic relapse: The psychophysiological data. British Journal of Psychiatry 1992;161:3–11. [PubMed: 1638327]

Tohen M, Waternaux CM, Tsuang MT. Outcome in mania: A 4-year prospective follow-up of 75 patients utilizing survival analysis. Archives of General Psychiatry 1990;47:1106–1111. [PubMed: 2244795]

Winters RW, Johnson SL, Cuellar AK. Regulatory deficits in bipolar disorder and unipolar illness. Manuscript in preparation. 2008

Yan LJ, Hammen C, Cohen AN, Daley SE, Henry RM. Expressed emotion versus relationship quality variables in the prediction of recurrence in bipolar patients. Journal of Affective Disorders 2004;83:99–206.

Table 1

Response to Criticism Among Reactors: Effects of Diagnosis on Profile of Mood States (POMS) Negative Affect at Baseline and Immediately After the Criticism

Fixed effect	Coefficient	SE	t	*p* Value
Intercept (π_{i0})—Level 1				
Intercept (β_{00})—Level 2	0.44	0.09	4.78	0.00
Diagnosis (β_{01})—Level 2	0.74	0.19	3.82	0.00
Reactivity slope ($\pi_i 1$)—Level 1				
Intercept (β_{10})—Level 2	0.04	0.11	0.37	0.71
Diagnosis (β_{11})—Level 2	**0.29**	**0.17**	**1.76**	**0.08**
Recovery slope (π_{i2})—Level 1				
Intercept (β_{20})—Level	−0.02	0.11	−0.20	0.84
Diagnosis (β_{21})—Level 2	−0.53	0.26	−2.08	0.04

Note. The parameter for the line in bold reflects how a person's diagnosis influences the slope of their reactivity to the criticism. A positive coefficient means that the bipolar group (coded 1) has a steeper increase in negative affect after being criticized.

Table 2

Recovery from Criticism: Effects of Diagnosis on Profile of Mood States (POMS) Negative Affect at Baseline and Immediately After the Criticism for Participants who Reacted to the Criticism

Fixed effect	Coefficient	SE	t	*p* Value
Intercept (π_{i0})—Level 1				
Intercept (β_{00})—Level 2	0.72	0.14	5.12	0.00
Diagnosis (β_{01})—Level 2	0.77	0.26	2.93	0.01
Reactivity slope (π_{i1})—Level 1				
Intercept (β_{10})—Level 2	0.35	0.08	4.50	0.00
Diagnosis (β_{11})—Level 2	0.33	0.16	2.07	0.04
Recovery slope (π_{i2})—Level 1				
Intercept (β_{20})—Level	−0.08	0.20	−0.42	0.68
Diagnosis (β_{21})—Level 2	**0.80**	**0.37**	**− 2.16**	**0.04**

Note. These results only include participants who reacted to the criticism (i.e., had some initial increase in negative affect). The parameter for the line in bold reflects how a person's diagnosis influences their recovery after they reacted to the criticism.