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Attentional Biases and the Persistence of Sad Mood in Major Depressive Disorder

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Abstract

This study examined whether attentional biases for emotional information are associated with impaired mood recovery following a sad mood induction among individuals with and without major depressive disorder (MDD). Attentional biases were assessed with an exogenous cuing task using emotional facial expressions as cues among adults with (n = 48) and without (n = 224) current MDD. Mood reactivity and recovery were measured following a sad mood induction. Mood reactivity strongly predicted mood recovery; however, this relationship was moderated by attentional biases for negative emotional stimuli. Biases for sad and fear stimuli were associated with diminished mood recovery following mood induction across the sample. However, biases for sad stimuli were associated with significantly greater impairments in mood recovery among individuals with MDD than healthy controls. Furthermore, within the MDD group, impaired mood recovery was positively associated with depression severity. These results suggest that attentional biases maintain depression, in part, by facilitating the persistence of sad mood.

Keywords

major depressive disorder; attention; attentional bias; mood persistence; mood induction

Major depressive disorder (MDD) is a common disorder affecting approximately 121 million people worldwide (World Health Organization, n.d.). MDD is characterized as an emotional disorder that influences an individual's mood, motivation, sleep, eating, concentration, self-worth, and productivity (American Psychiatric Association, 2000). These symptoms have a significant impact on people with MDD and the people around them, leading to greater interpersonal problems, unemployment, substance abuse, delinquency, and risk for suicide (Kessler & Walters, 1998). Given the enormous impact at individual and societal levels, there is a clear need to better understand factors that maintain this disorder.

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Although the clinical presentation of MDD varies between individuals, one hallmark symptom is a persistent sad mood. Persistent sad mood involves feeling sad, down, depressed, or blue most of the day, nearly every day for a period of 2 weeks or longer (American Psychiatric Association, 2000). This symptom plays a defining role in the disorder; however, the mechanisms underlying mood persistence in MDD remain poorly understood. This gap in our understanding of MDD could undermine efforts to improve treatments aimed at interrupting persistent sad mood. In this article, we focus on identifying cognitive mechanisms associated with the persistence of sad mood in MDD.

Cognitive theories of depression posit that biases in the way depressed people process emotional information help perpetuate depressive symptoms (e.g., Beck, 1967; Ingram, 1984; Teasdale, 1988). These biases include, for example, preferential attention toward mood-congruent information in the environment (see also Segal & Shaw, 1986; Williams, Watts, MacLeod, & Mathews, 1997). According to cognitive theories, events that trigger sad mood interact with these biases to influence the generation of negative thoughts and feelings that, in turn, lead to more persistent sadness (e.g., Ingram, 1984; Teasdale, 1988). A growing body of research supports the idea that depressed individuals demonstrate cognitive biases for emotional information, including attentional biases (see Gotlib & Joormann, 2010, for review). Although this research suggests that biased attention could maintain sad mood, few studies have tested this possibility directly.

To date, research on the role of attentional biases in MDD has focused on characterizing the nature of such biases. This work followed influential research on the nature of attention for emotional stimuli in anxiety disorders (MacLeod, Mathews, & Tata, 1986; Mogg, Bradley, & Williams, 1995). Broadly, anxiety is associated with the rapid orienting of attention toward threatening (or fearful) stimuli (MacLeod et al., 1986). These biases are typically evident even when these stimuli are presented briefly (e.g., <500 ms; e.g., Mogg, Bradley, de Bono, & Painter, 1997; Mogg, Mathews, & Eysenck, 1992).

By contrast, depression is associated with elaborative attention toward mood-congruent (or sad) stimuli (Mogg & Bradley, 2005; see also Mathews & MacLeod, 2005; Wisco, 2009). Depressed individuals do not automatically orient toward sad stimuli; however, they demonstrate preferential, sustained attention toward these stimuli once they enter awareness (e.g., >1,000 ms; e.g., Bradley, Mogg, & Lee, 1997; Eizenman et al., 2003; Gotlib, Krasnoperova, Yue, & Joormann, 2004; Joormann, Talbot, & Gotlib, 2007; Kellough, Beevers, Ellis, & Wells, 2008; Leyman, De Raedt, Vaeyens, & Philippaerts, 2011; Siegle, Granholm, Ingram, & Matt, 2001).

But do these elaborative attentional biases help maintain depression, as posited by cognitive theories (e.g., Beck, 1967; Teasdale, 1988)? In this study, we employed a laboratory-based mood induction procedure to examine whether attentional biases for negative information are associated with more persistent sad mood. Depressed and nondepressed participants underwent a standardized mood induction procedure and we measured sad mood before, immediately after the induction (i.e., reactivity), and 12 min later (i.e., recovery). This design allowed us to explore the relationship between attentional bias and mood in a well-controlled environment.

We were primarily interested in how participants recovered from the mood induction procedure, an index of mood persistence. It is important to note that participants can recover from a mood induction quite differently. To illustrate, Figure 1 shows mood reactivity and recovery profiles for four study participants. All reported significant reactivity to the mood induction procedure; however, each demonstrated different levels of mood recovery. Figures 1a and 1b reflect “successful” levels of mood recovery for a nondepressed and depressed

participant, respectively. In each case, decrease in sad mood during recovery is equal to or greater than their initial mood reactivity. In contrast, Figures 1c and 1d reflect "impaired" levels of mood recovery: Decrease in sad mood during recovery is less than their initial mood reactivity. Therefore, mood recovery depends, in part, on each individual's mood reactivity. However, these plots also illustrate that recovery can be quite variable across individuals. The current study examined whether attentional biases contribute to difficulty with mood recovery, particularly among individuals with MDD.

We anticipated that attentional biases for negative stimuli (sad and fear) would be associated with impairments in mood recovery among individuals who reacted to the mood induction, but that biases for sad stimuli would correspond to unique disturbances in mood recovery among depressed individuals. Recent experimental evidence demonstrates that inducing a negative attentional bias (using both sad and fear stimuli) in healthy individuals leads to higher levels of sad mood following a laboratory stress manipulation (MacLeod, Rutherford, Campbell, Ebsworthy, & Holker, 2002). Furthermore, biases for negative stimuli (sad and fear) are associated with neuroticism and introversion (e.g., Chan, Goodwin, & Harmer, 2007; Derryberry & Reed, 1994), two personality dimensions predictive of higher levels of negative affect, including sad mood, in the general population (Clark, Watson, & Mineka, 1994; Eysenck, 1998). Therefore, we hypothesized that a general negative bias, for sad or fear stimuli, would be associated with impaired mood recovery among all individuals who reacted to the mood induction procedure. However, we expected biases for sad stimuli to exhibit a stronger association with impaired mood recovery among depressed versus nondepressed individuals. This prediction is based on the idea that specific, mood congruent biases play an active role in maintaining MDD (cf. Ingram, 1984; Teasdale, 1988). Finally, we hypothesized that impairments in mood recovery would be associated with greater depression severity among individuals with MDD. This prediction is consistent with the idea that impaired mood recovery contributes to a more severe and persistent episode of MDD.

Method

Participants

Participants were recruited using Internet, TV, and radio advertisement. The sample consisted of 291 community members from a large southwestern city in the United States who met the following inclusion criteria: (a) a *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., *DSM–IV*) diagnosis of MDD or no current or past MDD (control); (b) between the ages of 22 and 55 years; (c) normal or corrected-to-normal vision; and (d) ability to speak, read, and understand English sufficiently well to complete the procedures of the study. Exclusion criteria included (a) current or past *DSM–IV* diagnosis of alcohol or drug abuse in past 6 months; (b) current or past *DSM–IV* diagnosis of substance or alcohol dependence, bipolar disorder, psychotic disorder, obsessive– compulsive disorder, social phobia, panic disorder, posttraumatic stress disorder, and generalized anxiety disorder; or (c) a history of epilepsy or head trauma.

Three individuals who qualified for the MDD group and 15 individuals who qualified for the control group were removed from this analysis because of incomplete or missing data. One individual who qualified for the control group was subsequently removed from this analysis because his or her estimates of attentional bias were considered outliers even after applying our data reduction procedures (see Exogenous Cuing Task section): Attentional bias score for sad stimuli was greater than 14 standard deviations from the sample mean and attentional bias score for fear stimuli was greater than 6.5 standard deviations from the sample mean. Excluding these individuals did not substantively change the findings reported below. After removing these participants, the total sample size included in this analysis was 272 community members: 48 meeting criteria for the MDD group and 224 meeting criteria for

the control group. Demographic data about the sample is reported in the Results section and in Table 1.

Materials

Mini International Neuropsychiatric Interview (MINI)—The electronic version of the MINI was used as a screening interview to determine whether participants provisionally met criteria for study entry. The MINI is a short, structured screening interview that was developed for the *DSM–IV* (American Psychiatric Association, 1994) and the *International Classification of Diseases* (10th ed., *ICD–10*; World Health Organization, 1993) psychiatric disorders (Sheehan et al., 1998). The MINI has been validated against the Structured Clinical Interview for *DSM–IV* (SCID; First, Spitzer, Gibbon, & Williams, 2002) diagnoses and against the Composite International Diagnostic Interview for *ICD–10* (Lecrubier et al., 1997; Sheehan et al., 1998).

MINI interviewers were undergraduate research assistants who received at least 10 hr of training, wherein they learned interview skills, reviewed diagnostic criteria, and role-played interviews. Because this was a screening interview, brevity was important. Interviewers could terminate the interview as soon as the participant did not meet study criteria. Therefore, the full MINI was typically completed only for participants who met criteria for study entry. The average length of MINI screening interviews was approximately 15 min.

Structured Clinical Interview for *DSM–IV* (SCID)—To confirm key inclusion/exclusion criteria from the screening interview, participants completed the patient version of the SCID (First et al., 2002) during an in-person interview at the time of study participation. Three assessors conducted all interviews. Two assessors were doctoral graduate students with at least 2 years of clinical training and assessment experience. The third assessor was a full-time research assistant with a bachelor's degree in psychology. The third assessor participated in 15 hr of training and supervision led by graduate-level assessors, wherein she learned interview sills, reviewed diagnostic criteria for relevant *DSM–IV–TR* diagnoses (American Psychiatric Association, 2000), observed mock interviews, and role-played interviews. Twenty percent of all interviews were rated by an independent assessor who was a doctoral student in clinical psychology with at least 2 years of assessment experience. Agreement for MDD diagnosis between study interviewers and the independent assessor was excellent ($k = 1.00$, $p < 0001$).

Beck Depression Inventory—II (BDI–II)—The BDI–II (Beck, Steer, & Brown, 1996) is a widely used self-report questionnaire that assesses depression severity. The BDI–II consists of 21 items and measures the presence and severity of cognitive, motivational, affective, and somatic symptoms of depression. Past reports have indicated that test–retest reliability is adequate (Beck, Steer, & Carbin, 1988). The BDI–II has been found to be valid among psychiatric inpatient and outpatient samples (Beck et al., 1988).

Beck Anxiety Inventory (BAI)—The BAI (Beck & Steer, 1993) is a widely used self report questionnaire that assesses anxiety symptom severity. The BAI consists of 21 items and measures the presence and severity of a wide range of common anxiety symptoms. Past reports have indicated adequate internal consistency, test-retest reliability, and external validity (e.g., Beck, Epstein, Brown, & Steer, 1998).

Exogenous cuing task—The exogenous cuing task was developed by Posner (1980) and modified to incorporate emotional cues (e.g., Beevers, Wells, Ellis, & McGeary, 2009; Koster, De Raedt, Goeleven, Franck, & Crombez, 2005). Each trial sequence (see Figure 2) began by presenting a fixation cross in the center of the screen for 500 ms. Then, a face cue

was presented on either the left or the right side of the visual field for 1,500 ms. After cue offset, a probe (either * or **) appeared immediately on the left or right side of visual field and remained on the screen until the participant responded. The participant's task was to identify probe type as quickly and accurately as possible. Participants pressed a corresponding button on a response box to indicate the type of probe that appeared. Reaction time (RT) for the participant to respond with a button press following the probe onset was logged for each trial. After the participant responded, the screen was black for 500 ms before the next trail began. Seventy-five percent of probes appeared on the same side of visual field as the visual cue (a valid trial). Twenty-five percent of the probes appeared on the opposite side of visual field as the cue (an invalid trial). Both valid and invalid trials had a 50% chance of having either the single- or double-asterisk probe.

Cue stimuli were images of faces taken from the Pictures of Facial Affect (Ekman & Friesen, 1976) photo set. Human faces were selected because facial expressions receive special processing priority (Farah, Wilson, Drain, & Tanaka, 1998), and because human faces have been used extensively in behavioral and imaging studies, and are arguably more ecologically valid than written words. Twelve faces were selected from each of the following categories: happy, sad, fear, and neutral. All stimuli were presented on a black background on a 17-in. (43-cm) color monitor. Stimuli were approximately 10.5 × 17 cm when presented on the screen. Participants completed 10 practice trials using neutral faces as cues. Anyone failing to respond accurately to at least eight of the 10 trials repeated the practice trials until they had achieved 80% accuracy. Participants then completed a total of 96 trials. They viewed each of the 48 stimuli twice. Order of stimulus presentation was randomized for each participant, with the stipulation that each of the 48 stimuli was viewed once before stimuli were repeated.

As suggested by Mogg, Holmes, Garner, and Bradley (2008), a general measure of attentional bias can be derived from the exogenous cuing task using the following formula:

$$\text{Attentional bias score (ABS)} = (\text{mean RT invalid emotion cue} - \text{mean RT valid emotion cue}) - (\text{mean RT invalid neutral cue} - \text{mean RT valid neutral cue}).$$

Positive values reflect an attentional bias for emotional cues relative to neutral cues. Negative values reflect an attentional bias for neutral cues relative to emotional cues. Bias scores were calculated for each emotional valence: sad, fear, and happy.

Exogenous cuing task trials with incorrect responses (0.37%) were deleted and not used in analysis. Mean RTs were generated per individual, per condition (e.g., sad invalid, sad valid, happy valid, happy invalid, etc.). Trial-level RTs that were at least 2 standard deviations beyond the mean per individual, per condition were deleted (3.55% of total raw data) and a new mean RT was then calculated, per individual, per condition, and used in the analyses. Together, these procedures resulted in the exclusion of 3.92% of the raw data.

Profile of Mood States (POMS): Sad mood was measured at three time points: before, immediately after termination of the mood induction protocol, and twelve minutes after the mood induction, using four descriptors taken from the POMS (McNair, Lorr, & Droppleman, 1992). These included items with the best factor loadings for the depression mood scale: "sad," "worthless," "blue," and "hopeless." Participants rated how well each item described their current mood on a 5-point Likert scale ranging from *not at all* (0) to *very much* (4). Scores from these items were summed to create an index of sad mood at each time point.

Mood variables: We created two variables to represent mood reactivity and mood recovery. Mood reactivity represents the difference between baseline mood and mood immediately after the mood induction: Higher scores reflect greater mood reactivity. Mood recovery represents the difference between mood immediately after the mood induction and mood twelve minutes later: Lower scores reflect greater mood recovery. Biased attention moderates mood persistence in MDD 15 Procedure.

Participants completed the MINI screening interview over the phone with a trained interviewer. Participants who passed the screening assessment were scheduled for a laboratory appointment. Upon arrival, participants were oriented to the lab, provided informed consent, and completed a demographic survey. They then completed the SCID interview to confirm presence of inclusion criteria and absence of exclusion criteria. Qualified participants then completed several self-report questionnaires, including the BDI-II. Next, they completed the exogenous cuing task and mood induction in a counter balanced order (half mood induction and recovery before exogenous cueing and half after). Mood induction type was also counter balanced across participants (music and video). Sad mood was measured using items from the POMS before, immediately after termination of the mood induction procedure, and after a twelve-minute delay. Upon completion of study procedures, participants were debriefed and paid $15 per hour (up to a maximum of $50) for their participation. The Internal Review Board at the University of Texas at Austin approved all study procedures.

Mood induction: Participants were randomly assigned to receive one of two standardized mood inductions. One was a standardized film clip that has been shown to specifically elicit sadness (Gross & Levenson, 1995). The sad clip is 170 seconds and is taken from the film, *The Champ*, in which the father of a young boy dies after suffering a severe beating during a boxing match. A high-resolution digital version of the film clip was presented on a 20 inch LCD computer monitor. For the second mood induction participants listened to sad music while imagining a time in their life when they were very sad. The sad music (Samuel Barber's *Adagio for Strings*) effectively induced a sad mood in previous mood provocation Biased attention moderates mood persistence in MDD 14 research (Hunt & Forand, 2005). This type of sad mood induction in general is effective in eliciting a temporary sad mood (Van der Does, 2002). We used two mood inductions to ensure that results are not specific to a particular set of mood induction procedures.

Participants' mood was monitored throughout the study to ensure that it had returned to pre-experiment levels before being dismissed from the study. For those whose mood had not returned to baseline, a positive mood induction procedure was administered. An opportunity to talk with a doctoral level clinician was also offered to participants who continued to report sad mood following the positive mood induction. Treatment referrals were also offered to all participants in the study.

Statistical Analysis

All analyses were performed in R (http://www.r-project.org/) and STATA 11 (StataCorp, College Station, TX). The assumptions underlying repeated measures analysis of variance (ANOVA) and regression were tested and confirmed at each stage of analysis.

Results

Sample Characteristics

Descriptive statistics for the sample are presented in Table 1. The mean age of participants was 29.01 years (*SD* = 8.77), although the depressed group was significantly older than the

nondepressed group. Participants were predominantly women, and the distribution of gender did not differ across depression groups. The distribution of participants across racial groups approximated census estimates from the community (54.4% White, 8.5% African American, 19.5% Asian, and 17.6% other). The distribution of race across depression groups approached statistical significance. Across these racial groups, 21.4% of the sample was Hispanic. The number of Hispanic participants did not differ across depression groups. Given group differences, we controlled for age, gender, and race at each stage of the subsequent analyses.

Next, we examined depression group differences in depression severity, anxiety symptoms, and medication use (see Table 1). MDD and control groups differed in their levels of reported depression severity as indexed by the BDI–II. The average BDI–II score in the MDD group was in the moderate range (25.06), whereas the average score in the control group was in the clinically insignificant range (3.49). MDD and control groups also differed on reported levels of anxiety symptoms as measured by the Beck Anxiety Inventory (BAI). The average BAI score in the MDD group was in the mild range, whereas the average score in the control group was in the clinically insignificant range. Thus, we controlled for anxiety symptoms (BAI scores) at each stage of analysis. Finally, MDD and control groups differed in use of psychotropic medication (i.e., allowable medications as specified by exclusion criteria). Thirteen depressed individuals (27.1%) were taking a psychotropic medication, whereas none of the control individuals were taking psychotropic medications. For analyses limited to the MDD group, we also controlled for use of psychotropic medication.

Mood Induction

To confirm that the mood induction procedures successfully increased sad mood, we performed a repeated measures ANOVA with mood as the dependent variable and time (before and immediately after the mood induction) as the within-subject factor. This model revealed a significant main effect for time, $F(1, 271) = 160.30$, $p < .0001$, Cohen's $d = 1.54$, indicating that the mood inductions did increase sad mood as expected. Change in sad mood did not vary as a function of depression status, $F(1, 270) = 0.12$, $p = .72$. MDD and control participants reacted similarly to the mood induction procedure.

Next, we tested whether mood induction order (before or after exogenous cuing task) moderated these effects. The interaction term for Order × Time was significant, $F(1, 270) = 10.51$, $p = .0013$, Cohen's $d = 0.40$, suggesting that there were differences in mood reactivity based on the order in which the mood induction occurred. Simple effects testing indicated that although the mood induction produced significant changes in mood irrespective of the order in which it was administered, those who completed the attentional bias assessment before the mood induction reported greater reactivity to the mood induction, $F(1, 132) = 99.68$, $p < .0001$, Cohen's $d = 1.74$, compared with those who completed the mood induction prior to the bias assessment, $F(1, 138) = 66.39$, $p < .0001$, Cohen's $d = 1.39$. Therefore, we included mood induction order as a covariate in all subsequent analyses.

Finally, we tested whether mood induction type (music or movie) moderated the effect of time on mood. The interaction term for Type × Time was significant, $F(1, 270) = 17.39$, $p < .0001$, Cohen's $d = 0.51$, indicating that there were significant differences in mood reactivity based on type of mood induction. Simple effects testing indicates that although both induction types produced significant changes in mood, the music induction produced a stronger effect on mood, $F(1, 138) = 122.33$, $p < .0001$, Cohen's $d = 1.88$, than the movie induction, $F(1, 132) = 49.17$, $p < .0001$, Cohen's $d = 1.22$. Therefore, we included mood induction type as a covariate in all subsequent analyses.

Mood Variables

We then explored differences in baseline mood, mood reactivity, and mood recovery by depression group (see Table 2). As expected, depressed individuals reported significantly higher levels of baseline sad mood, $F(1, 259) = 53.24$, $p < .0001$, Cohen's $d = 0.91$. As reported in the repeated measures analysis, MDD and control groups did not show significant differences in mood reactivity, $F(1, 259) = 0.27$, $p = .60$. Similarly, MDD and control groups did not show significant differences in mood recovery, $F(1, 259) = 0.38$, $p = .54$. These results indicate that MDD and control participants had similar patterns of mood reactivity and mood recovery despite baseline differences in sad mood. It is important to note, however, that there was a wide range of mood reactivity and recovery within each group (see Table 2).

Next, we explored the relationship between mood reactivity and recovery. We hypothesized a strong negative relationship between mood reactivity and mood recovery. Indeed, the correlation between these outcomes was strong and in the anticipated direction, $r = -.80$, $p < .0001$; after controlling for covariates, $t(259) = -20.59$, $p < .001$. The nature of this relationship did not differ as a function of depression status, $t(257) = 0.87$, $p = .38$. Although individuals experienced differing levels of reactivity to a sad mood provocation, they, on average, were able to recover from these moods within a relatively short period of time (i.e., return to baseline within 12 min).

Finally, we examined whether our estimates of mood reactivity and recovery were related to depression severity in the MDD group. Reactivity in the MDD group was not associated with depression severity, $r = -.18$, $p = .22$; after controlling for covariates, $t(35) = -1.20$, $p = .24$. Thus, more severely depressed participants did not demonstrate reduced reactivity. In line with our predictions, mood recovery was positively associated with depression severity in the MDD group, $r = .32$, $p = .02$; after controlling for covariates, $t(35) = 2.74$, $p = .01$ (see Figure 3). Slower mood recovery was associated with increased depression severity. This finding indicates that impairments in mood recovery are associated with worse outcomes among depressed individuals. Mood reactivity and recovery were unrelated to depression severity in the control group (reactivity: $r = .04$, $p = .57$; recovery: $r = .01$, $p = .88$).

Attentional Bias

A detailed summary of ABSs by depression group is listed in Table 3. The reader will note substantial within-group variability among the three bias scores across depressed and nondepressed groups. We first examined whether there were differences in levels of attentional bias across MDD and control groups. Contrary to previous findings, we did not discover a difference in ABSs for sad stimuli, $F(1, 260) = 0.26$, $p = .61$, fear stimuli, $F(1, 260) = 1.44$, $p = .23$, or happy stimuli, $F(1, 260) = 0.27$, $p = .60$, based on depression group.

It is important to note that the only group difference we observed in ABS was a main effect for gender: Men and women differed in bias for sad, $F(1, 260) = 5.06$, $p = .03$, Cohen's $d = 0.28$, and fear stimuli, $F(1, 260) = 4.11$, $p = .04$, Cohen's $d = 0.25$, but not happy stimuli, $F(1, 260) = 1.67$, $p = .20$. Men showed significantly stronger bias for both sad ($M_{Men} = 25.71$ ms, $SD_{Men} = 145.27$ ms; $M_{Women} = -12.83$ ms, $SD_{Women} = 93.05$ ms) and fear stimuli ($M_{Men} = 30.02$ ms, $SD_{Men} = 116.66$ ms; $M_{Women} = 3.47$ ms, $SD_{Women} = 89.00$ ms).

It is also important to note that there was no main effect of assessment order (before or after the mood induction) on ABSs for sad, $F(1, 260) = 1.98$, $p = .16$, fear, $F(1, 260) = 0.16$, $p = .69$, and happy stimuli, $F(1, 260) = 0.06$, $p = .80$. Moreover, these results were not moderated by depression status across sad, $F(1, 259) = 0.44$, $p = .51$, fear, $F(1, 259) = 0.64$, $p = .42$, and happy stimuli, $F(1, 259) = 0.04$, $p = .84$. Engaging in the mood induction procedure either

before or after the exogenous cuing task did not appear to substantively influence ABSs for either depressed or nondepressed participants.

Next, we examined whether ABSs for sad, fear, and happy stimuli were associated with mood reactivity. Furthermore, we tested two-way interactions to determine whether these relationships varied as a function of depression status. ABS was unrelated to mood reactivity across sad, $t(259) = -0.98$, $p = .33$, fear, $t(259) = -1.10$, $p = .27$, and happy stimuli, $t(259) = -1.21$, $p = .23$. This did not differ across depression groups: ABS Sad × Depression Group, $t(257) = 0.09$, $p = .93$; ABS Fear × Depression Group, $t(257) = 0.72$, $p = .48$; ABS Happy × Depression Group, $t(257) = -0.79$, $p = .43$. These findings indicate that attentional biases are not associated with levels of mood reactivity.

Finally, we examined whether ABSs for sad, fear, and happy stimuli moderated the relationship between mood reactivity and mood recovery. Furthermore, we tested three-way interactions to determine whether these relationships varied as a function of depression status.

Bias for sad stimuli—The two-way interaction between sad bias and mood reactivity predicting mood recovery was significant, $t(257) = 4.00$, $p < .001$, effect size $r = .24$. The three-way interaction between sad bias, mood reactivity, and depression status predicting mood recovery was also significant, $t(253) = 2.65$, $p = .008$, effect size $r = .16$. Simple effects testing of this three-way interaction indicated that although the two-way interaction between sad bias and mood reactivity was significant for both MDD, $t(33) = 2.92$, $p = .006$, effect size $r = .45$, and control groups, $t(209) = 2.54$, $p = .012$, effect size $r = .17$, the effect was much stronger in the MDD group (see Figure 4). Individuals with stronger attentional bias for sad stimuli reported greater impairments to mood recovery when they reacted to a mood-inducing event. This relationship was stronger among individuals with current MDD compared with healthy controls.

To further examine the nature of this effect in the MDD group, we examined simple slopes for the relationship between mood reactivity and mood recovery among depressed individuals with higher and lower attentional bias for sad stimuli (i.e., ±1 *SD*). These simple slopes are plotted in the MDD panel of Figure 4. The simple slope for individuals with higher levels of sad bias was not significant, $t(44) = -1.48$, $p = .145$, indicating that as mood reactivity increased, these individuals did not demonstrate a corresponding level of mood recovery. By contrast, the simple slope for individuals with lower levels of attentional bias for sad stimuli was significant, $t(44) = -7.16$, $p < .001$, suggesting that higher levels of mood reactivity were associated with greater mood recovery among individuals with lower levels of bias for sad stimuli. This analysis further supports the idea that depressed individuals with higher levels of attentional bias for sad stimuli experience impaired mood recovery, particularly when they react to a mood-inducing event.

Bias for fear stimuli—The two-way interaction between fear bias and mood reactivity predicting mood recovery was significant, $t(257) = 3.36$, $p = .001$, effect size $r = .21$. The three-way interaction between fear bias, mood reactivity, and depression status was not significant, $t(253) = 0.45$, $p = .65$. Individuals with stronger attentional bias for fear stimuli reported greater impairments to mood recovery when they reacted to a mood-inducing event (see Figure 5). This effect was consistent across as the full sample, as this relationship was not moderated by depression status.

Bias for happy stimuli—The two-way interaction between happy bias and mood reactivity predicting mood recovery was not significant, $t(257) = 0.72$, $p = .47$. The three-way interaction between happy bias, mood reactivity, and depression status predicting mood

recovery was not significant, $t(253) = 0.81$, $p = .42$. Biased attention for happy stimuli was not associated with differences in mood recovery when individuals reacted to a mood-inducing event.

Discussion

This study examined whether attentional biases for emotional stimuli are associated with the persistence of sad mood among individuals with and without MDD. Cognitive theories of depression implicate information-processing biases, such as biased attention, in the maintenance of depressive symptoms. Previous research suggests that depressed and dysphoric individuals harbor biased attention for emotional information (e.g., Bradley et al., 1997; Eizenman et al., 2003; Gotlib et al., 2004; Kellough et al., 2008); however, the consequences of these biases have not been examined. This study sought to link these biases to mood persistence, a hallmark symptom of depression.

Our results support the idea that negative attentional biases facilitate the persistence of sad mood. Depressed and nondepressed individuals who demonstrated more pronounced biases for negative stimuli and experienced greater reactivity to a mood-inducing event showed greater difficulty recovering from that event after 12 min. Biases for sad stimuli were particularly important for depressed individuals, as this bias was more strongly associated with impairments in mood recovery for depressed versus nondepressed participants. Furthermore, impairments in mood recovery were positively associated with depression severity in the MDD group. Together, these findings provide evidence that more severely depressed individuals show impairments in mood recovery that are associated with attentional biases, particularly when they experience greater mood reactivity.

While these findings have important implications for depression, they also speak to theories of emotional experience more generally. Our findings suggest that attentional biases for negative information also interfere with mood recovery among nondepressed individuals when they react to mood-inducing events. Thus, these biases appear to broadly influence emotional experience and may reflect individual differences in one's ability to manage emotional reactions. These findings are in line with experimental evidence demonstrating that inducing a general negative bias (using both sad and fear stimuli) leads to higher levels of sad mood following a laboratory stress manipulation (MacLeod et al., 2002). Moreover, longitudinal studies suggest that negative processing biases predict future emotional and hormonal (i.e., cortisol) responses to stress (Fox, Cahill, & Zougkou, 2010) and vulnerability to negative mood states, such as depression and posttraumatic stress disorder symptoms (Beevers, Lee, Wells, Ellis, & Telch, 2011). Thus, individuals with negatively biased attention (for sad and/or fear stimuli) without current psychopathology may be at increased risk for the development of negative mood states. More longitudinal research is needed to address this important question.

Research on the nature of emotion regulation processes (Gross, 1998; Ochsner & Gross, 2005) suggests that cognitive control processes, including mechanisms underlying attentional control, may mediate adaptive emotion regulation strategies that help individuals manage distressing emotions (e.g., Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008). Therefore, one possibility is that the observed relationship between attentional bias and mood persistence is mediated by emotion regulation. Joormann (2004, 2006) has proposed that maladaptive emotion regulation strategies, including rumination, may mediate the relationship between inhibitory control deficits and depressive symptoms. Rumination represents the tendency to perseverate on the causes and consequences of depressed mood (Nolen-Hoeksema, 1991; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Depressed individuals believe that rumination is helpful (Papageorgiou & Wells, 2001, 2003; Watkins

& Baracaia, 2001), but evidence suggests that ruminative thinking amplifies and maintains depression (e.g., Just & Alloy, 1997). Future work must explore whether the relationship between attention and mood persistence observed in this study is mediated by broader, maladaptive emotion regulation strategies (see also Joormann & Gotlib, 2010).

Another possibility is that these biases reflect stable differences in underlying personality dimensions associated with negative affect. Indeed, attentional biases for both sad and fear stimuli were associated with impaired mood recovery among depressed and nondepressed individuals. These findings suggest that independent of current psychopathology, a general bias toward aversive stimuli is associated with prolonged sad mood. This conclusion is supported by evidence that attentional biases for sad and fear stimuli are associated with neuroticism and introversion (Chan et al., 2007; Derryberry & Reed, 1994), stable personality dimensions that are highly predictive of negative affect and, importantly, depression vulnerability (e.g., Clark et al., 1994). Therefore, biases for sad and fear stimuli may mediate the relationship between individual differences in stable personality dimensions (e.g., neuroticism) and prolonged episodes of negative affect. Future research is required to test this hypothesis.

Beside these theoretical implications, the current study highlights the value of taking an individual differences perspective when examining cognitive biases and mood in depression. This approach contrasts with previous research, which has primarily focused on comparing estimates of attentional bias between groups (e.g., depressed vs. nondepressed, dysphoric vs. nondysphoric). That approach is useful to infer differences between groups; however, it largely ignores within-group variability. For instance, some depressed individuals exhibit strong attentional biases for sad stimuli, whereas others do not. It is important to note that the current study indicates that these differences are meaningfully related to a hallmark symptom of depression. Thus, an individual differences approach may help identify mechanisms underlying variability (or stability) in symptom presentation both across individuals and within the same individual across time.

Our results point to two key areas of individual difference. First, attentional biases vary within a sample of MDD individuals: Many participants with MDD did not demonstrate negatively biased attention (see also Bradley et al., 1997; Gotlib et al., 2004). Importantly, these individuals showed less persistent mood following an acute mood induction. Second, mood reactivity varies between individuals with MDD. Whereas many individuals in our sample reacted strongly to the mood induction procedures, others did not. This finding is consistent with previous studies using mood induction procedures (e.g., Larsen & Ketelaar, 1989; Martin, 1990; Rottenberg, Kasch, Gross, & Gotlib, 2002). Identifying factors that explain individual differences in attentional bias and mood reactivity in MDD will be an important future direction for this area of research (cf. Rottenberg, Gross, & Gotlib, 2005).

Overall, this pattern of individual differences within MDD is consistent with extant literature. Neural models of MDD largely implicate the interaction between hypoactive regions underlying cognitive control (e.g., dorsal–lateral prefrontal cortex) and hyperactive regions underlying emotional reactivity (e.g., amygdala) (Disner, Beevers, Haigh, & Beck, 2011; Drevets, 2001; Mayberg, 2003). However, there is notable variability in this pattern of findings (e.g., Canli et al., 2004; Elliott, Rubinsztein, Sahakian, & Dolan, 2002; Lawrence et al., 2004) that has been linked to differences in affective symptoms (e.g., anhedonia, anger) as well as significant etiological considerations (e.g., history of childhood maltreatment) (Dougherty et al., 2004; Grant, Cannistraci, Hollon, Gore, & Shelton, 2011; Keedwell, Andrew, Williams, Brammer, & Phillips, 2005). Importantly, individual differences in pretreatment activity within these regions have also been shown to predict response to specific interventions (e.g., cognitive– behavioral therapy; Siegle, Carter, & Thase, 2006;

Siegle, Steinhauer, Friedman, Thompson, & Thase, 2011). This is an important avenue for future research and represents an exciting opportunity to integrate basic and applied research.

An important caveat is that this study was not designed to test the causal relationship between attentional bias and mood persistence. Although we manipulated mood, we did not manipulate attentional bias. Therefore, the observed associations between negative attentional bias and mood recovery are correlational and, thus, it remains unclear whether attentional biases cause mood persistence in depressed and nondepressed people.

Cognitive bias modification designs represent a growing class of experimental procedures aimed at testing causal predictions about putative cognitive biases associated with psychopathology (e.g., Mathews & Mackintosh, 1998; Wilson, MacLeod, Mathews, & Rutherford, 2006). These studies manipulate specific cognitive biases in a randomized, placebo-controlled design to test these predictions. Preliminary attention bias modification research with dysphoric people suggests that manipulating attentional biases may help ameliorate mood persistence (Wells & Beevers, 2010), although this hypothesis has not been tested directly. Conversely, inducing negative attentional biases in nondepressed individuals appears to exacerbate emotional responses to adverse events (MacLeod et al., 2002). Together, these findings lend preliminary support to the idea that attentional biases play a causal role in the persistence of sad mood. However, future work using these methods is required to directly test this hypothesis.

Another important limitation of this study is that it represents a laboratory study of mood persistence. Although this design provides experimental control of the mood manipulation, we were limited to examining mood persistence on the order of minutes, not weeks as the *DSM–IV* defines clinically significant persistent sad mood in MDD. Moreover, we examined mood persistence in response to a contrived laboratory-based mood induction procedure. This procedure represents a modest analogue to the types of real-life events that induce sad mood (Martin, 1990). Furthermore, outside the laboratory, multiple mood-inducing events likely interact in dynamic ways to predict mood reactivity and mood persistence. Future work is required to understand how cognitive biases influence the maintenance of mood over longer time intervals and during conditions of multiple, dynamic mood-inducing events (cf. Peeters, Nicolson, Berkhof, Delespaul, & deVries, 2003).

Moreover, we sampled mood only once during the recovery period. Thus, we have only one index of mood persistence across time. Research suggests that mood persistence (or emotional lability, more generally) is a dynamic, nonlinear process across time (e.g., Kuppens, Oravecz, & Tuerlinckx, 2010; Kuppens, Van Mechelen, Nezlek, Dossche, & Timmermans, 2007). Sampling mood with greater frequency during the recovery period would have allowed a more fine-grained analysis of mood persistence in MDD.

Finally, we used a brief assessment of attentional bias that was very similar to previously published work (Beevers et al., 2009). A brief assessment prevents fatigue for participants; however, it limits the number of trials included in indices of attentional bias, which may lower the reliability of these estimates. Future work should measure bias using more trials to address this potential limitation. Moreover, we relied on RT estimates to compute attentional bias scores; future efforts may benefit from greater precision by using eye registration technology to generate estimates of attentional bias.

Despite these limitations, this study is an important step toward understanding how attentional biases maintain depression. Results indicate that more severely depressed individuals show impairments in mood recovery that are associated with negative attentional

biases when they respond to mood-inducing stimuli. Furthermore, biases for sad stimuli may selectively impair efforts to regulate sad mood in MDD. These findings support cognitive theories of depression and provide a link between putative cognitive biases and a hallmark symptom of depression.

At the same time, these findings suggest that the adverse effects of negative attentional biases on mood recovery are not limited to MDD. Nondepressed individuals show similar, albeit less pronounced, impairments in mood recovery that are associated with biases for sad and fear stimuli. Therefore, these findings have implications for theories of emotion and emotion regulation more generally, and suggest that negative attentional biases interfere with efforts to resolve acute mood reactivity.

Taken together, these findings advance our understanding of how cognitive mechanisms maintain depressive symptoms. We believe this is an important step toward elucidating mechanisms that maintain MDD, a step that could ultimately help improve interventions aimed at preventing the onset of and promoting recovery from this common and debilitating psychiatric disorder.

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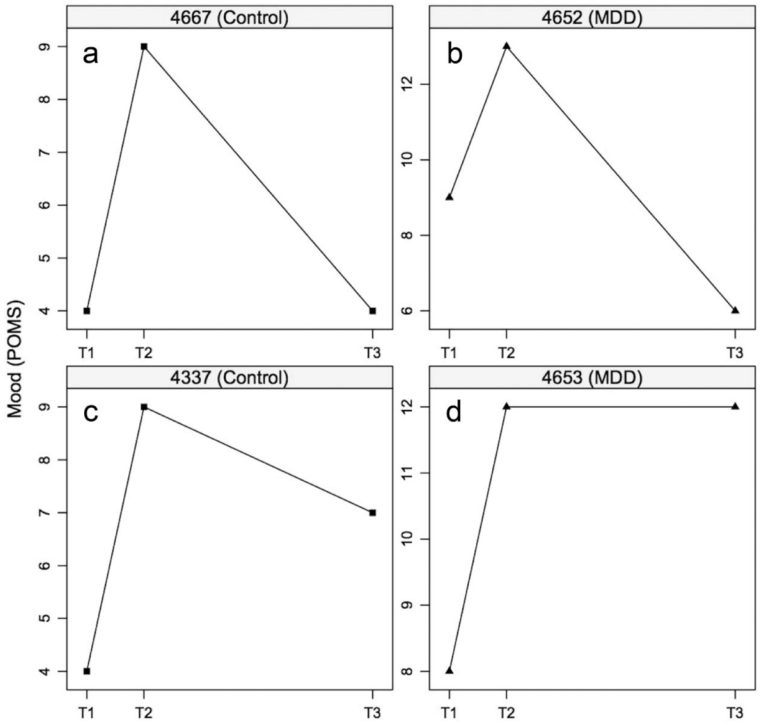

Figure 1.

Mood profiles. Profiles of mood reactivity and mood recovery for four participants. POMS = Profile of Mood States; MDD = major depressive disorder; T1 = POMS at baseline; T2 = POMS immediately after mood induction (~ T1 + 4 min); T3 = POMS after 12 min of recovery (= T2 + 12 min). Note the group (control vs. MDD) differences in baseline mood. Panels a and b reflect “successful” mood recovery; Panels c and d reflect “impaired” mood recovery.

Figure 2.

Exogenous cueing task sequence. Trial sequence for valid and invalid trials. Fixation cross, face stimulus, and probe are not to scale. Face images from Pictures of Facial Affect by P. Ekman and W. Friesen, 1976, Palo Alto, CA: Consulting Psychologists Press. Copyright 1976 by Consulting Psychologists Press. Reproduced with permission from Paul Ekman, PH.D./Paul Ekman Group, LLC.

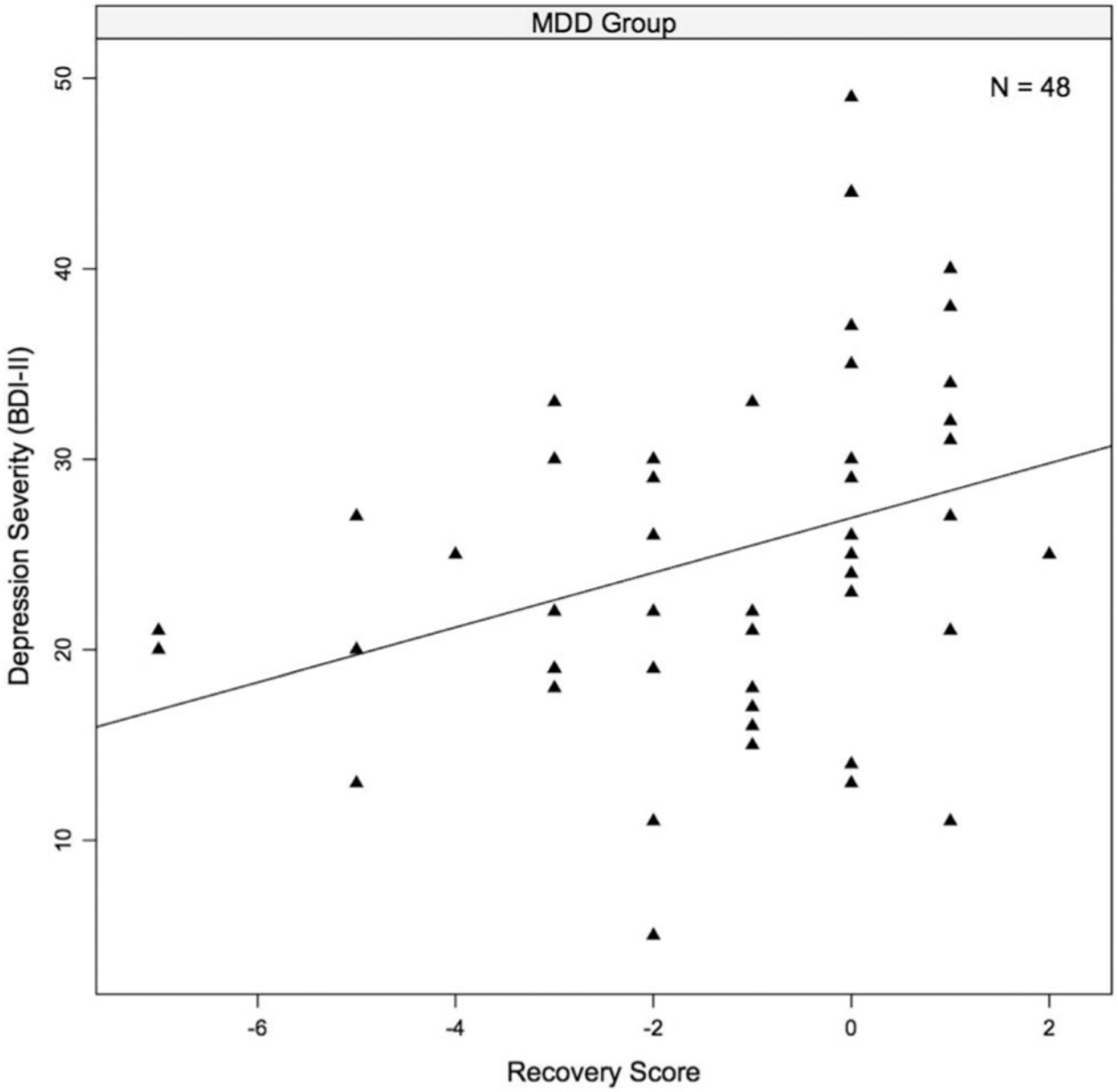

Figure 3.

Impaired mood recovery and depression severity. Relationship between mood recovery and depression severity in the group with major depressive disorder (MDD). Line represents fitted regression (r = .32, p = .02). Two observations overlap. BDI–II = Beck Depression Inventory—II.

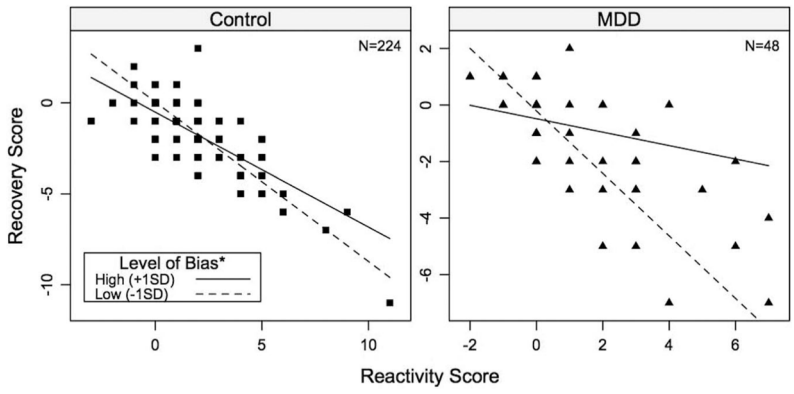

Figure 4.

Sad Bias. Mood recovery as a function of mood reactivity and bias for sad stimuli (by depression group). * Legend applies to both plots. There are multiple overlapping observations on each plot. MDD = major depressive disorder.

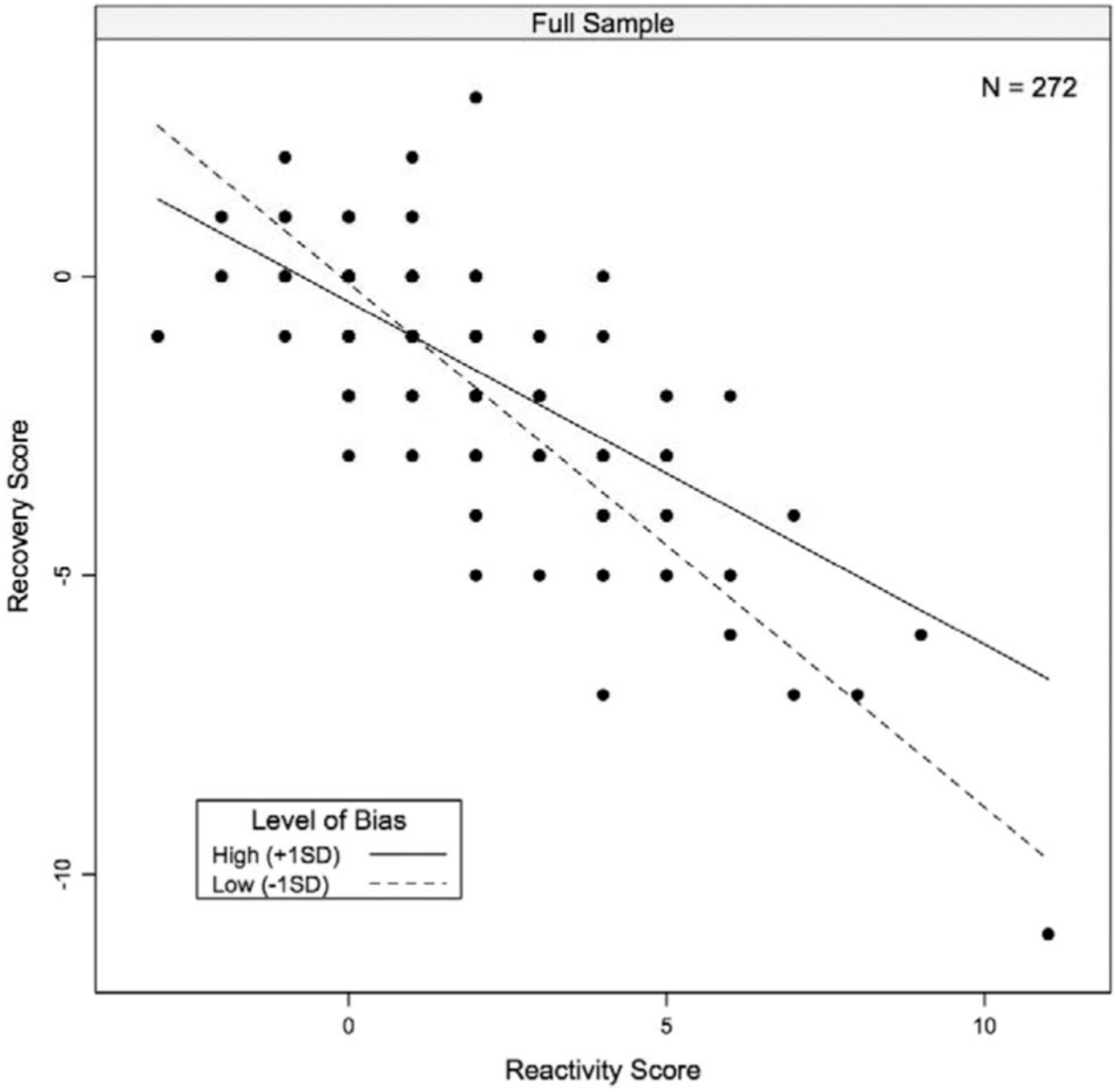

Figure 5.
Fear Bias. Mood recovery as a function of mood reactivity and bias for fear stimuli. There are multiple overlapping observations.

Table 1

Participant Demographics

Demographic	Control	MDD	Test
Mean (*SD*)			$F(1, 270) = 13.01$, $p = .0004$
Age (years)	28.14 (8.08)	33.06 (10.63)	
Gender, *n*			$\chi^2 = 2.53$, $p = .112$
Male	83	12	
Female	141	36	
Race, *n*			Fisher's exact, $p = .077$
African American	15	8	
Asian	49	4	
White	119	29	
Other[a]	51	7	
Hispanic, *n*			$\chi^2 = 1.43$, $p = .231$
Yes	51	40	
No	173	7	
Unknown	0	1	
Mean (*SD*)			$F(1, 270) = 555.85$, $p < .0001$
BDI–II score	3.49 (4.64)	25.06 (9.37)	
Mean (*SD*)			$F(1, 270) = 186.51$, $p < .0001$
BAI score	3.25 (4.03)	13.00 (6.23)	
Psychotropic medication, *n*			Fisher's exact, $p < .001$
Yes	0	14	
No	224	38	

Note. MDD = major depressive disorder; BDI–II = Beck Depression Inventory—II; BAI = Beck Anxiety Inventory.

[a]Includes American Indian, Native Hawaiian, multiple races, and "none" (i.e., did not endorse a race).

Table 2

Summary of Mood Variables (Baseline, Reactivity, Recovery) by Depression Group

	Control			MDD		
Mood	**Mean**	***SD***	**Range**	**Mean**	***SD***	**Range**
Baseline	4.71	1.35	(4, 11)	8.46	3.42	(4, 16)
Reactivity	1.46	1.79	(−3, 11)	1.35	2.26	(−2, 7)
Recovery	−1.33	1.64	(−11, 3)	−1.29	2.11	(−7, 2)

Note. MDD = major depressive disorder.

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Table 3

Summary of Attentional Bias Scores (Sad, Fear, Happy), by Depression Group

	Control			MDD		
Bias	**Mean**	***SD***	**Range**	**Mean**	***SD***	**Range**
Sad (ms)	0.15	115.69	(−320, 732)	2.87	114.36	(−208, 448)
Fear (ms)	7.15	97.96	(−390, 371)	38.81	106.95	(−202, 521)
Happy (ms)	8.53	116.79	(−505, 597)	13.08	77.35	(−127, 185)

Note. MDD = major depressive disorder.

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