

The Role of Emotion Regulation in Attentional Biases in Mood-Congruent Information
and Sustained Negative Affect in Depression

By

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Abstract

Objective: The present study examined the extent to which emotion regulation and distinct emotion regulation processes mediate the relationship between attentional bias and sustained negative affect in individuals at-risk for depression following a mood induction. **Method:** To assess this question, previously depressed ($n = 40$) and never depressed control ($n = 44$) participants underwent a sad mood induction and mood reactivity and recovery were measured. Sad mood was assessed at four different time points: immediately before and after mood induction, and six and 12 minutes after mood induction. Participants completed an exogenous cuing task to assess for attentional biases and answered questionnaires related to emotion regulation processes and depressive symptomatology. **Results:** Attentional bias did not significantly predict sustained negative affect after the mood induction and therefore mediational models could not be constructed. Further, there were no significant differences in attentional bias between previously depressed and never depressed individuals. However, cognitive reappraisal significantly predicted mood reactivity and mood recovery after 12 minutes, and executive suppression approached significance in predicting mood recovery after six minutes. Previously depressed and never depressed individuals significantly differed in their reported use of ruminative brooding and reflection. **Conclusions:** Results suggest that cognitive reappraisal may be particularly important in reducing sustained negative affect in depression and suggest there may be merit in examining the effects of emotion regulation strategies beyond the 12 minute time frame used in this study.

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Table of Contents

ABSTRACT	iii
ACKNOWLEDGEMENTS	iv
TABLE OF CONTENTS	v
INTRODUCTION	1
METHOD	9
Participants	
Measures	
Stimuli	
Procedure	
RESULTS	15
Mood Induction	
Mood Reactivity & Recovery	
Attentional Bias	
Bias for sad stimuli	
Bias for happy stimuli	
Mood reactivity as a moderator	
Bias for sad faces	
Bias for happy faces	
Emotion Regulation	
Emotion regulation as a mediator	
Emotion regulation processes and mood reactivity and recovery	
Emotion regulation processes and BDI-II scores	
DISCUSSION	22
REFERENCES	27
TABLES	
Table 1: Sample Characteristics	35
Table 2: Mood Induction Results, by measure	36
Table 3: VAS and PANAS Means	36
Table 4: Mood Reactivity and Recovery Scores, by depression history	36
Table 5: Attentional Bias Scores (Sad, Happy), by depression history	36
Table 6: Emotion Regulation Means, by depression history	37
Table 7: Prediction of Mood Recovery, by emotion regulation process	37

FIGURES

Figure 1: Mean VAS Scores, as a function of time	38
Figure 2: Mean NA Scores, as a function of time	39
Figure 3: Mean PA Scores, as a function of time	40
Figure 4: Mean Attentional Bias Scores for Sad Faces, by depression history	41
Figure 5: Mean Attentional Bias Scores for Happy Faces, by depression history	42

The Role of Emotion Regulation in Attentional Biases in Mood-Congruent Information and Sustained Negative Affect in Depression

Depressive disorders represent the leading cause of disability worldwide and are significant contributors to the global burden of disease (World Health Organization, 2012). In 2012, The World Health Organization estimated that depressive disorders affect 350 million people around the world. Suicide, which is often associated with depression and other mental illness, represented the tenth leading cause of death in the United States in 2010; an estimated 105 suicide completions per day occurred in the U.S. in 2010 (Center for Disease Control, 2012). Depressive disorders are therefore not only debilitating, but also potentially fatal. Individuals with depressive disorders experience disruptions in all domains of functioning. Further, occurrence of a major depressive episode is the best predictor of subsequent episodes and susceptibility to depression; increasing numbers of depressive episodes are predictors of the chronicity and severity of the disorder. Given the devastating impact of depression, it is important to evaluate theories of depression and understand the underlying mechanisms of the disorder so that efficacious prevention and intervention efforts may be realized.

Depression is characterized by a constellation of symptoms. To meet criteria for major depressive disorder, sad mood or anhedonia must be sustained for at least two weeks, along with at least four of the following symptoms: fatigue, concentration difficulties, indecisiveness, suicidal ideation, insomnia or hypersomnia, marked weight loss or gain, psychomotor retardation or agitation, and/or extreme feelings of guilt or worthlessness (American Psychiatric Association, 2013). Although the presentation of depression can be heterogeneous, sustained negative affect is a hallmark symptom of depression. Thus, understanding the processes that promote and maintain this negative mood state is essential.

Cognitive theories of depression posit that negative cognition contributes to and perpetuates depression (Beck, 1967). As compared to nonvulnerable individuals, those who are currently depressed or vulnerable to depression are thought to possess a depressogenic cognitive schema in which a triad of negative core beliefs about the self, the world, and the future are held (Beck, 1970). Depressogenic cognitive schemas include themes of loss, failure, worthlessness, rejection, and separation (Beck, 1976). Depressogenic cognitive schemas are maintained, strengthened, and expanded by recurrent processing of negative information that furthers interconnected networks of depressotypic thoughts and images (Bower, 1981; Ingram, 1984).

Additionally, cognitive theories suggest that depressed individuals have systematic emotional information processing and attentional biases that cause them to selectively process stimuli congruent with their depressogenic schemas (Beck, 1976). The content specificity hypothesis proposes that biases in depression should be directed towards depressotypic information with themes of sadness and loss as opposed to other types of valenced information (e.g., positive, neutral; Beck, 1976). Thus, those with depression should exhibit biases towards schema-congruent information, such as sad faces. This enduring bias to processing schema-congruent information is theorized to lead to sustained negative affect (Beck, 1967).

Further, those with susceptibility to depression may exhibit biases towards schema-congruent information and subsequently experience sustained negative affect. Vulnerability to depression has been operationalized by past research as having parental history of depression, a previous depressive episode, and/or as experiencing subthreshold levels of depression (Ingram & Hamilton, 1999). In vulnerable individuals, depressogenic schemas can be active or latent; when latent, schemas become activated by stress, and are thus described as latent but reactive (Segal & Shaw, 19886). Cognitive diathesis-stress models postulate that latent depressogenic schemas are

activated by environmental stressors or mood induction in a laboratory setting (see Scher, Ingram, & Segal, 2005 for a review). Activation of depressogenic schemas in vulnerable individuals activates depressotypic cognitive processes and products, such as attentional biases to mood-congruent information and negative automatic thoughts regarding the self (Bistricky, Ingram, & Atchley, 2011). Thus, active depressogenic schemas and resulting enduring bias to mood congruent information may result in sustained negative affect and perpetuation of depression.

Empirical evidence has provided support for theoretical models of mood-congruent biased attention and information processing in depression and dysphoria. Specifically, researchers have suggested that depressed and depression-vulnerable individuals exhibit preferential attention to and difficulty disengaging from mood congruent information once it has entered awareness. In order for mood-congruent information to enter awareness, studies have shown that the information must be presented for at least 1000 milliseconds (ms) or one second (Bradley, Mogg, & Lee, 1997). Nondepressed individuals, however, do not experience difficulties disengaging from negative information; instead, they are more likely to shift attention away from negative information. For example, Gotlib, Krasnoperova, Yue, and Joormann (2004) investigated attentional differences to interpersonal information between a clinically depressed group, a clinically anxious group, and a control group. Gotlib et al. (2004) found that the depressed group exhibited an attentional bias to, and difficulty disengaging from, sad or mood-congruent faces once the stimulus was presented for at least one second. The anxious group and control group did not exhibit the same biases. Additionally, Koster et al. (2005) studied attention to positively valenced, negatively valenced, and neutral words in dysphoric and nondysphoric participants. Koster et al. found that dysphoric participants exhibited increased

attention to negatively valenced words, while nondysphoric participants maintained attention to positive words.

Attentional biases have also been shown to persist despite recovery from a depressive episode. Joormann and Gotlib (2007) found that currently depressed and previously depressed participants exhibited biases to sad faces in a dot-probe task; never depressed controls did not exhibit this bias and avoided sad faces. In an eye-tracking study, Sears, Newman, Ference, and Thomas (2011) found that previously depressed and currently dysphoric participants oriented to images with themes of depression more frequently than never depressed controls. Sears et al. (2011) also found that previously depressed and dysphoric individuals oriented to positive images less frequently than controls. Thus, evidence has shown that depressed, dysphoric, and depression-vulnerable individuals have been shown to exhibit preferential attention, or attentional bias, to mood-congruent information.

Researchers have hypothesized that difficulty disengaging from mood-congruent information may preclude depressed and depression-vulnerable individuals from restoring positive affect. Attentional bias to mood-congruent information may contribute to sustained negative affect and therefore the maintenance of depression. Clasen, Wells, Ellis, and Beevers (2012) examined the association between attentional bias and persistence of a negative mood state following sad mood induction in individuals diagnosed with major depressive disorder and never depressed controls. Contrary to previous findings, Clasen et al. (2012) did not find differences in attentional bias between currently depressed and never depressed groups as ascertained by an exogenous cuing task. However, they found that attentional bias for sad stimuli was associated with greater impairments in recovery to baseline mood for the depressed group than for the control group. Thus, an association between depression, depression maintenance,

and attentional bias towards mood-congruent information has been established; however, explanations for this association have not been investigated.

Theorists have posited that vulnerable and nonvulnerable individuals do not differ in their reactivity to a negative event, but in their trajectory of emotional recovery from a negative event (Teasdale, 1988). Thus, the difference lies in their ability to restore their emotional baseline. Accordingly, researchers have proposed emotion regulation as an explanation for the relationship between attentional bias towards mood-congruent information and negative affect in depression (Joormann, 2004, 2006; Joormann & D'Avanzato, 2010). Emotion regulation has been conceptualized as the conscious or unconscious process of evaluating and modulating emotions in response to environmental stimuli at any point in the emotion generative process (Gross, 1998). Emotion regulation is a cognitive process that modifies the type, intensity, physiological experience, and behavioral expression of emotion, as well as when the emotion is expressed. The fundamental goal of emotion regulation is to increase positive affect and decrease negative affect (Gross, 1998). Several distinct cognitive emotion regulation processes have been identified. At the most basic level, these processes are differentiated by when they occur in the process of emotion generation (Gross, 2001). Processes are either *antecedent-focused* or *response-focused*. Antecedent-focused processes are those that occur before the emotional response and subsequent behavioral and physiological responses are generated. Response-focused processes are those that modulate current emotional responses and their behavioral and physiological correlates.

One antecedent-focused emotion regulation process is *cognitive reappraisal*. Cognitive reappraisal involves modifying a potentially emotion-eliciting stimulus so that its emotional impact will be altered (Gross, 1998). Those who deploy cognitive reappraisal actively attempt to reframe negative stimuli and situations and repair bad moods. Consequently, those who employ

cognitive reappraisal report increased positive affect and psychological well-being, and experience fewer depressive symptoms than those who do not regularly use cognitive reappraisal (Gross & John, 2003). Depressed individuals report less frequent use of cognitive reappraisal than remitted and never depressed individuals (Joormann & Gotlib, 2010). Cognitive reappraisal is therefore considered to be an adaptive emotion regulation process.

Executive suppression is a response-focused emotion regulation process. Executive suppression involves inhibiting the expression and experience of emotion (Gross, 2001). Although suppression is effective in reducing short-term emotional expression and experience, it is not effective in reducing long-term emotional experience and results in increased physiological arousal (Gross, 1998; Gross 2001). Interestingly, when an individual attempts to regulate negative emotion by means of suppression, paradoxical increases in negative emotion result (Gross, 1998). The use of executive suppression has been found to be associated with various forms of psychopathology and is a risk factor for depression (Aldao, Nolen-Hoeksema, & Schweizer, 2009). Thus, executive suppression is considered to be a maladaptive emotion regulation process.

Another response-focused emotion regulation process is *ruminatio*n, which is characterized by the repetitive recycling of thoughts. Rumination can be further differentiated into *ruminative brooding* and *reflection* (Trey

nor, Gonzalez, & Nolen-Hoekshema, 2003). Ruminative brooding is characterized by repetitive processing and focusing on the causes, feelings, and consequences of negative emotions in an effort to problem solve (Aldao et al., 2009; Trey

nor et al., 2003). Paradoxically, however, rumination inhibits, rather than promotes, problem solving. Further, brooding over depressotypic emotions promotes sustained negative affect (Gross, 1998). Rumination is thus considered to be a maladaptive emotion regulation

process. In contrast, reflection is a response-focused process characterized by engaging in problem solving (Treyner et al., 2003). Research has found that use of reflection predicts remission from a depressive episode and is longitudinally associated with less depression (Arditte & Joormann, 2011; Treyner et al., 2003). Thus, reflection is considered to be an adaptive emotion regulation process.

In sum, the goal of emotion regulation is to increase positive emotions and decrease negative emotions; however, certain emotion regulation processes increase negative emotions and reduce positive emotions. Those processes that increase negative emotions (e.g., suppression and brooding) are associated with decreased psychological well-being and psychopathology. Indeed, empirical evidence has shown that depressed individuals are more likely to use certain processes (brooding, suppression) than others (cognitive reappraisal). Further, maladaptive emotional regulation processes are present not only in depressed individuals but also in those who have remitted from a depressive episode (Ehring, Fischer, Schünlle, Bösterling, & Tuschen-Caffier, 2008). Despite this work, surprisingly little research has examined the role of emotion regulation in the maintenance of depression.

Empirical evidence has shown that certain emotion regulation processes are more frequently used in depressed individuals than others, suggesting that these processes may help to perpetuate negative emotions and subsequent depression. Not surprisingly, researchers have posited that emotion regulation may play a key role in the relationship between attentional bias to mood-congruent information in depressed and depression-vulnerable individuals; however, to our knowledge, no study has investigated the role of emotion regulation in the association of attentional bias and maintenance of sustained negative emotionality and depression.

In line with these ideas, the goal of the present study was to examine the extent to which emotion regulation and distinct emotion regulation processes may mediate the relationship between attentional bias and sustained negative affect in individuals at-risk for depression. To assess this question, previously depressed and never depressed control participants completed a modified version of the Clasen et al. (2012) exogenous cuing task to assess for attentional biases and answered questionnaires related to emotion regulation processes and depressive symptomatology. In the present study, depression vulnerability was operationalized as history of a clinically significant major depressive episode. As per the diathesis-stress model, participants underwent a sad mood induction intended to activate latent depressogenic schemas. The activation of such schemas should result in the emergence of dysfunctional cognitive processes (e.g., attentional bias). Thus, the mood induction was used to elicit depressotypic cognitions in the depression-vulnerable and allow for a comparison of attentional bias between groups.

I expected previously depressed participants to exhibit greater attentional bias towards sad information as compared to controls based on cognitive theories of depression and empirical support of these theories (e.g., Beck, 1976; Joormann & Gotlib, 2007; Ingram, 1984; Sears et al., 2007). Further, based on Clasen et al.'s (2012) findings, I expected participants who exhibit biases towards sad stimuli to have impaired mood recovery, regardless of depression history. Regarding emotion regulation, I hypothesized that emotion regulation strategies would mediate the relationship between attentional bias and sustained negative affect, such that rumination and ruminative brooding would prolong negative affect and reflection, executive suppression, and cognitive reappraisal would shorten the duration of negative affect in both groups. Further, I anticipated that previously depressed and never depressed participants would differentially use some emotional regulation processes based on evidence that maladaptive emotion regulation

strategies persist past remission from depression (Ehring et al., 2008); specifically, I predicted group differences in use of executive suppression, overall rumination, and ruminative brooding. Finally, I predicted that, regardless of depression history, participants who reported high levels of rumination or ruminative brooding would show greater mood reactivity and impaired mood recovery.

Method

Participants

The sample consisted of 84 students from the University of Kansas who participated in partial fulfillment of course requirements. Participants met the following inclusion criteria: (a) Beck Depression Inventory II (BDI-II) total less than or equal to nine and no previous history of depression as ascertained by the self-report version of the major depressive episode module of the Structured Clinical Interview for the Diagnostic and Statistical Manual IV-TR (SCID-SR; control); or (b) BDI-II total less than or equal to nine and endorsement of a previous, but not current, depressive episode within the past three years on the SCID-SR (previously depressed). The never depressed control group consisted of 44 participants and the previously depressed group consisted of 40 participants. Sample characteristics are presented in Table 1.

Measures

Self-Report Structured Clinical Interview for DSM-IV-TR Axis I Disorders (SCID-SR). A modified self-report version of the SCID (First et al., 2002) was used to evaluate the presence of a current or past major depressive episode. For the purposes of this study, only the major depressive episode (MDE) section of the mood module will be administered in a self-report form. The SCID-SR was used instead of the SCID-I-NP because undergraduate research

assistants ran the majority of participant sessions. Given the amount of training and experience necessary to administer the SCID-I-NP, the SCID-SR was best suited for this study.

Beck Depression Inventory (BDI-II). The BDI-II is a 21-item self-report questionnaire developed to assess depression severity (Beck, 1996). The BDI-II is a widely used measure of depression. Participants were asked to respond to each question based on the past two weeks, including the day of administration. BDI-II scores may range from 0 to 63, with higher scores indicating more symptoms of depression (Beck, Steer, & Brown, 1996). The BDI-II has shown adequate reliability and validity (Beck, et al., 1996).

Emotion Regulation Questionnaire (ERQ). The ERQ (Gross & John, 2003) is a 10-item self-report questionnaire designed to measure trait-level emotion regulation responses to positive and negative emotions. Specifically, the ERQ examines cognitive reappraisal (e.g., *I control my emotions by changing the way I think about the situation I'm in*) and executive suppression (e.g., *I control my emotions by not expressing them*) through two subscales. Responses are rated on a 7-point Likert scale, where 1=*strongly disagree*, 4=*neutral*, and 7=*strongly agree* are item anchors. The ERQ has been shown to be valid and reliable, with good internal consistency ($\alpha = .77$), test-retest reliability ($r = .69$), and convergent and discriminant validity (Gross & John, 2003).

Ruminative Responses Scale (RRS). The RRS (Treynor, Gonzales, and Nolen-Hoeksema, 2003) is a 22-item self-report questionnaire that examines rumination. The RRS has two subscales for brooding and reflection that were identified through factor analysis. Each subscale is comprised of six items. Each item is framed with the prompt, "*When you are feeling sad, or depressed, how often do you...*". An example item from the brooding scale is ...*Think "What am I doing to deserve this?"*, and an example item from the reflection scale is ...*Analyze*

recent events to try to understand why you are depressed. Item responses have anchors 1=*Almost never* to 4=*Almost always*. Adequate internal consistency and test-retest reliability for the RRS were established in a large community sample for brooding ($\alpha=.77$, $r=.62$) and reflection ($\alpha=.72$, $r=.60$) subscales (Treyner et al., 2003).

Visual Analogue Scale (VAS). The VAS (Luria, 1975) is a 100 millimeter line used to assess current mood state, in this case sad mood. Participants were asked to indicate their current level of sadness by drawing a mark at the point on the line that correlated with their current level of sadness. The line is anchored with extremes of the mood state of interest; with *not sad at all* at 0 mm and *extremely sad* at 100 mm. Scores range from zero to 100, with higher scores denoting higher reports of current sadness. Scores are derived by measuring the distance from the least extreme anchor, *not sad at all*, to the point marked by the participant.

The Positive and Negative Affect Schedule (PANAS). Both the positive (PA) and negative (NA) affect scales of the PANAS (Watson, Clark, & Tellegen, 1988) were used to measure mood immediately before and after the mood induction. The PA and NA scales represent the degree to which individuals are experiencing positive and negative affect within the environment; thus, lower scores on either denote minimal positive or negative emotionality, whereas high scores would denote distress on the NA or pleasurable engagement on the PA (Watson et al., 1988). The PANAS is a 20-item scale consisting of 10 positive words and 10 negative words. Participants were asked to rate how much each word described how they were currently feeling on a 5-point Likert scale, where 1=*not at all* and 5=*extremely*. Positive items were summed to create a positive affect (PA) score, and negative items were summed to create a negative affect (NA) score. The PANAS has shown adequate reliability and validity on both scales, with good internal consistency on the PA ($\alpha=0.89$) and NA ($\alpha=0.85$; Crawford & Henry,

2004). Crawford and Henry (2004) established normative means for the PA and NA scales based on a large clinical sample. The mean for the PA scale is 31.31 ($SD = 7.65$) and the mean for the NA scale is 16.00 ($SD = 5.90$). Additionally, Crawford and Henry (2004) found that the PA scale explained a significant proportion of variance unique to depression versus anxiety, and also found that the PA scale explained significantly more of the proportion of variance unique to depression than the NA scale. Crawford and Henry (2004) posited that this may be due to the negative relationship between anhedonia and high PA.

Stimuli

The exogenous cuing task. The exogenous cuing task (Posner, 1980) was used to measure attentional bias and difficulty disengaging from emotional stimuli before, immediately after, and twelve minutes after mood induction. The length of reaction time to a probe quantitatively assesses attentional bias and difficulty disengaging from emotional stimuli after viewing stimuli. These stimuli can be emotional faces or words. Increasing length of reaction time corresponds to greater attentional bias and increased difficulty in disengaging from the previous stimulus. A modified version of the exogenous cuing task that incorporates emotional faces was used (Clasen et al., 2012). Each session incorporated emotional images of happy, sad, and neutral faces taken from the set of Karolinska Directed Emotional Faces (KDEF; Lundqvist, Flykt, & Öhman, 1998). Thirty-two faces were selected from each emotional category so that 96 total faces were shown in each session. Words are traditionally used in the task, but Clasen et al. (2012) posited that human faces are more appropriate because facial expressions receive processing priority and are widely used in research. Further, processing of human facial affect influences emotion regulation, in that human facial expressions communicate and elicit emotions (Bistricky, Ingram, & Atchley, 2011; Ruys & Stapel, 2008). Human facial expressions elicit

immediate emotion, and because of this, are thought to be more effective in eliciting emotions than words (Bistricky, Ingram, & Atchley, 2011; Vanderploeg, Brown, & Marsh, 1987).

The modified exogenous cuing trial began with a white fixation cross in the center of the screen for 500 ms. A face then appeared in either the left or right side of the visual field for 1500 ms. When the face disappeared, a probe—an O or a Q—appeared in the same or opposite location of the face. Participants were asked to press the computer key corresponding to the probe type as quickly and accurately as possible. The probe did not disappear until the participant responded. Reaction times (RTs) and accuracy were recorded. After each response, a black screen appeared for 500 ms until the next trial began. Probes appearing on the same side of the visual field are valid and probes appearing in the opposite side of the visual field are invalid. In this task, 50% of the probes were valid and 50% of probes were invalid. Within valid and invalid trials, there was a 50% chance of the probe being an O or a Q. The task began with 16 practice trials using neutral faces; participants had to correctly respond to 80% of the faces before proceeding with the task.

Mood induction. Participants were provided a CD player that instructed them to recall a negative event in their life along with the event’s emotional, physiological, and interpersonal correlates. They were instructed to think about this event while they listened to music from the “Field of Dreams” soundtrack for approximately eight minutes. This mood induction has been successfully used to elicit negative mood in never depressed and previously depressed individuals (Ingram & Ritter, 2000). After listening to the soundtrack, participants were asked to write about the negative event they had thought about. A positive mood induction of thinking and writing about a happy time was presented at the end of the study. Participants were offered the opportunity to talk to a clinician and were informed about available treatment services.

Procedure

The duration of each session was approximately 60 minutes; participation was completed in one session. Participants who completed a pre-screening questionnaire and had a BDI-II total of nine or less were invited to participate in the study. After consent, participants were asked to complete the SCID-SR and BDI-II in order to reaffirm eligibility. Responses to the SCID-SR and BDI-II were evaluated as soon as these measures were completed. If participants no longer met inclusion criteria, they were thanked for their time, awarded appropriate credit, and excused. If inclusion criteria were met, the session proceeded as follows.

Participants were first seated in front of a stimulus computer to complete the exogenous cuing task. Instructions for the task appeared on the computer screen. Participants were asked to focus on the fixation cross at the center of the screen between trials. They were informed that a face would appear on either side of the screen after the fixation cross disappeared. Participants were told that either an “O” or a “Q” would appear on either side of the screen after the face disappeared. They were asked to press the “O” key if they saw an O and the “Q” key if they saw a Q. Participants were instructed to press the O or Q as quickly as possible without making mistakes. Participants were instructed to place their middle finger on the computer’s “O” button and their index finger on the computer’s “Q” button. The “4” key and the “5” key on the computer’s number pad were programmed as “O” and “Q,” respectively. After reading the instructions, participants performed a practice trial while the researcher was in the room. The researcher ensured participant understanding and exited the room while participants completed the task. Upon completion of the task, participants completed the ERQ, RRS, and a demographics questionnaire.

Participants then underwent the sad mood induction. Immediately before and after the sad mood induction, participants completed the VAS and PANAS to assess current mood state and ensure the mood induction had the intended effect. Participants completed the exogenous cuing task again immediately after and 12 minutes after the mood induction. At six and 12 minutes after the mood induction, the VAS was administered to assess current level of sadness and track mood recovery. Thus, sad mood was assessed at four time benchmarks: immediately before and after mood induction, six minutes after mood induction, and 12 minutes after mood induction. The exogenous cuing task was administered after the final administration of the VAS.

After participants completed the final exogenous cuing task, they were debriefed and given a positive mood induction to cultivate more positive emotions. They were asked to think and write about a happy time in their life. Participants were offered the opportunity to talk to a clinician and were informed about available treatment services. At the end of the session, participants were thanked and awarded commensurate credit.

Results

Mood Induction

A repeated measures ANVOA was performed to examine the efficacy of the mood induction in increasing sad mood. VAS scores from pre-mood induction, immediately after mood induction, six minutes after mood induction, and 12 minutes after mood induction were entered as dependent variables and within-subjects factors; depression history (previously depressed or never depressed control) was entered as a between-subjects factor. Mauchly's test indicated that the assumption of sphericity was violated, $\chi^2(5) = 58.08, p < .001$, and thus degrees of freedom were corrected using the Hunyh-Feldt estimates of sphericity ($\epsilon = .77$). The interaction of VAS scores and depression history was not significant. Further, the main effect of group was not

significant, indicating that VAS scores did not significantly differ as a function of depression history. There was a significant main effect for VAS across time of administration. Contrasts between pre-mood induction VAS scores and VAS scores obtained immediately after mood induction revealed a significant increase in sad mood. Contrasts between VAS scores obtained immediately after mood induction and six minutes after mood induction revealed a significant decrease in sad mood. Sad mood significantly decreased between six and 12 minutes after mood induction. Thus, sad mood increased significantly from baseline after the mood induction. Sad mood then decreased such that baseline sad mood was nearly restored after six minutes and fully restored after 12 minutes. See Figure 1 for a graphical representation of these results.

The PANAS was also used to assess reactivity to the mood induction. Pre- and post-mood induction scores from the Negative Affect (NA) scale of the PANAS were entered as the dependent variable and within-subjects factor, and group was entered as the between-subjects factor. The interaction of depression history and NA approached significance. There was a significant main effect for time, indicating that participants experienced a significant increase in negative affect following the mood induction. There was not a significant main effect for depression history, indicating that groups did not significantly differ in increase of negative affect after mood induction. Please see Figure 2 for a graphical representation of these results.

Additionally, pre- and post-mood induction scores from the Positive Affect (PA) scale of the PANAS were examined in a repeated measures ANOVA. The interaction of depression history and time of administration was not significant. The mood induction significantly decreased positive affect. Positive affect did not differentially decrease as a function of depression history. Please see Figure 3 for a graphical representation of these results. Further,

please see Table 2 for a summary of mood induction results and Table 3 for a summary of VAS and PANAS means.

Mood Reactivity and Recovery

Similar to Clasen et al. (2012), three mood variables were created to represent reactivity to and recovery from the mood induction. Subtracting the baseline VAS score from the time two VAS score measured reactivity, with larger, positive difference scores denoting greater mood reactivity. Two mood recovery variables were created to represent the recovery from mood induction at six minutes after mood induction and 12 minutes after mood induction. Subtracting the time two VAS score from the time three and time four VAS scores, respectively, created these variables. For these recovery variables, increasingly negative values denote greater mood recovery (e.g., VAS2 = 70; recovery1 = 30-70 = -40; recovery2 = 10-70 = -60). A summary of mood reactivity and recovery after six and 12 minutes is provided in Table 4.

Mood reactivity, $F(1, 82) = 0.33, p = .57$, recovery after six minutes, $F(1, 82) = 0.47, p = .49$, and recovery after 12 minutes, $F(1, 82) = 0.84, p = .36$, did not significantly differ as a function of depression history, indicating that groups reacted and recovered similarly to the mood induction. A significant negative correlation was found between mood reactivity and mood recovery after six minutes, $r = -0.62, p < .001$. Similarly, a significant negative correlation was found between mood reactivity and mood recovery after 12 minutes, $r = -0.72, p < .001$. Thus, larger increases in reported sad mood were associated with larger decreases in sad mood after six minutes and 12 minutes. Mood recovery after six minutes and mood recovery after 12 minutes were significantly positively correlated, $r = .84, p < .001$, indicating that decreases in sad mood after six minutes were associated with decreases in sad mood after 12 minutes.

BDI-II scores significantly predicted reactivity to the mood induction, $\beta = -.23$, $t(82) = -2.13$, $p = .04$, adjusted $R^2 = .041$, such that lower BDI-II scores predicted higher rates of reactivity. BDI-II scores did not significantly predict mood recovery after six minutes, or mood recovery after 12 minutes.

Attentional Bias

A quantitative attentional bias score for each participant was derived from observed reaction times. Mogg, Holmes, Garner, and Bradley (2008) suggested the following formula as a measure of general attentional bias derived from the exogenous cuing task:

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

Emotion cues are happy or sad faces, and neutral cues are neutral faces. Attentional bias scores were calculated separately for happy and sad cues. Positive difference values denote a bias for emotional cues relative to neutral cues, whereas negative difference values denote a bias for neutral cues relative to emotional cues. Median was substituted for mean in this study, as median is also a measure of central tendency.

Incorrect responses to task trials were not included analyses. Omitting incorrect responses resulted in excluding 1.87% of raw RT data. Median RTs were calculated for each trial type (e.g., invalid sad, valid sad) and session number (one, two, or three) for each participant. Median was selected instead of mean based on individual differences in skew and distribution of trial type. Median best captured individual differences without eliminating outliers. A summary of attentional bias scores is provided in Table 5. It is important to note that the large standard deviations in attentional bias scores found in the present study are congruent with those reported in other studies (e.g., Clasen et al., 2012).

Bias for sad stimuli. A repeated measures ANOVA with attentional bias administration time as the dependent variable and within-subjects factor, and depression history as the between-subjects factor did not reveal a significant interaction of depression history and attentional bias scores for sad faces or for main effects for time of administration and depression history. Please see Figure 4.

Bias for happy stimuli. A repeated measures ANOVA with attentional bias score administration time as the dependent variable and within-subjects factor, and depression history as the between-subjects factor showed a nonsignificant interaction of depression history and attentional bias scores for happy faces. The main effects for time of administration and depression history were also not significant. Please see Figure 5.

Mood reactivity as a moderator. The relationship between attentional bias for sad and happy faces, mood reactivity, and depression history was examined. Because attentional bias scores did not significantly differ as a function of time, attentional bias from the first administration were used in all of the following analyses.

Bias for sad faces. The three-way interaction for depression history, attentional bias for sad faces, and mood reactivity was not significant in predicting mood recovery after six minutes, or after 12 minutes. The two-way interaction of mood reactivity and attentional bias towards sad faces did not significantly predict mood reactivity after six minutes or 12 minutes.

Bias for happy faces. The three-way interaction for depression history, attentional bias towards happy faces, and mood reactivity did not significantly predict recovery after six minutes, or 12 minutes. The two-way interaction for attentional bias for happy faces and mood reactivity did not significantly predict mood recovery after six minutes or 12 minutes.

Emotion Regulation

The use of each emotion regulation strategy was examined as a function of depression history. Use of cognitive reappraisal and executive suppression did not significantly differ as a function of depression history. Level of overall rumination as assessed by the RRS differentially varied as a function of depression history, $F(1, 82) = 9.09, p = .003, \eta^2_p = .10$. The differential employment of ruminative brooding approached significance, $F(1, 82) = 2.89, p = .093$, such that those with a history of depression reported higher levels of ruminative brooding. Use of reflection significantly varied as a function of depression history, $F(1, 82) = 6.19, p = .015, \eta^2_p = .07$, and those with a history of depression reported greater levels of reflection than never depressed controls. Again, participants with a history of depression reported greater levels of overall rumination than previously depressed controls. Means of emotion regulation by depression history are presented in Table 6.

Emotion regulation as a mediator. As previously reported, bias for sad faces did not significantly differ as an effect of mood induction or time of administration. Similarly, bias for happy faces did not significantly differ as a result of time or mood induction. Since attentional bias scores did not significantly differ as a function of time, mood induction, and group, attentional bias scores from the first administration were used in mediation analysis.

In order to examine whether mediation has occurred, three relationships must be established: (1) the independent variable predicts the mediator; (2) the independent variable predicts the dependent variable; and (3) the mediating variable predicts the dependent variable (Baron & Kenney, 1986). The second relationship was first examined, as it was present in all mediation models. Regarding the second relationship, bias for happy faces did not significantly predict recovery after six minutes or after 12 minutes. Further, bias for sad faces did not significantly predict mood recovery after six minutes or after 12 minutes. Thus, mediation

models could not be tested because the relationships between attentional bias and sustained negative affect (recovery after six and 12 minutes) were not significant.

Emotion regulation processes and mood reactivity and recovery. The relationship between each emotion regulation strategy and reactivity to the mood induction was assessed. Overall rumination, ruminative brooding, reflection, and executive suppression did not significantly predict mood recovery. However, cognitive reappraisal significantly predicted mood reactivity, $\beta = .27$, $t(82) = -2.50$, $p = .015$, adjusted $R^2 = .059$. Thus, those who endorsed greater levels of cognitive reappraisal experienced greater reactivity to the mood induction. The relationship between each emotion regulation strategy and mood recovery at six minutes and 12 minutes after mood induction was also assessed. Ruminative brooding, or simply brooding, did not significantly predict mood recovery after six minutes or 12 minutes. Reflection also did not significantly predict mood recovery after 6 minutes or after 12 minutes. The overall rumination score derived by the RRS did not significantly predict mood recovery after six minutes or after 12 minutes. Use of cognitive reappraisal did not significantly predict mood recovery after six minutes; however, cognitive reappraisal did significantly predict mood recovery after 12 minutes, such that those with higher reported levels of reappraisal had negative, and therefore greater, recovery after 12 minutes. Use of executive suppression approached significance in predicted mood recovery after six minutes, but did not significantly predict recovery after 12 minutes. Please see Table 7 for a summary of these results.

Emotion regulation processes and BDI-II scores. The association between BDI-II scores and emotion regulation processes were examined. The correlation between BDI-II scores and overall rumination, reflection, ruminative brooding, and cognitive reappraisal were not significant. However, the correlation between BDI-II scores and executive suppression was

significant, $r(82) = .219, p = .023$, suggesting that higher BDI-II scores correspond to more frequent use of executive suppression.

Discussion

This study examined the role of emotion regulation in the association between attentional bias for emotional faces and sustained negative affect following a mood induction in previously depressed and never depressed participants. Contrary to the main hypothesis, none of the examined emotion regulation processes mediated the relationship between attentional bias towards sad faces and sustained negative affect. Further, incongruent with cognitive theories and previous research (Joorman & Gotlib, 2007; Sears et al., 2011), the present study did not find significant differences in attentional bias scores as a function of depression history. The relationship between emotion regulation processes and sustained negative affect, operationalized as mood recovery in the present study, was examined. Contrary to emotion regulation theory, ruminative brooding, reflection, and overall rumination did not significantly predict mood recovery. However, in line with theory, cognitive reappraisal significantly predicted mood recovery after 12 minutes, such that those with higher reported levels of reappraisal experienced greater mood recovery. Interestingly, higher levels of cognitive reappraisal coincided with greater reactivity to the mood induction. Consistent with emotion regulation theory and research that executive suppression only temporarily reduces negative emotionality (Gross 1998, 2001), use of suppression approached significance in predicting mood recovery after six minutes, such that those with higher reported levels of suppression experienced less mood recovery.

Regarding differences in use of emotion regulation processes between previously depressed and never depressed individuals, reported use of ruminative brooding, reflection, and overall rumination significantly differed as a function of depression history, such that previously

depressed individuals reported higher levels of these variables. Contrary to previous findings (e.g., Aldao et al., 2009), there were no differences in reported use of executive suppression between groups; however, use of executive suppression increased as BDI-II scores increased, in line with research that suggests suppression is associated with increased risk of depression (Aldao et al., 2009). Reported frequency of use of cognitive reappraisal did not significantly differ as a function of depression history, which is congruent with findings that previously depressed and never depressed individuals use cognitive reappraisal more frequently than do currently depressed individuals (e.g., Joorman & Gotlib, 2010).

There are several potential reasons why emotion regulation did not mediate the relationship between attentional bias and sustained negative affect. Perhaps the short duration of the experiment in a lab did not permit enough time to assess the effects of emotion regulation on sustained negative affect. In particular, previous literature and research has posited that use of executive suppression promotes temporary relief from negative emotionality, but that it also predicts negative emotionality later. Thus, mood could be sampled hours or days in a short-term longitudinal fashion after such an experiment to better capture the effects of some emotion regulation processes. Additionally, while the emotion regulation constructs examined in the present study have theoretical and empirical support for affecting mood, other constructs beyond those examined might be explored. Several emotion regulation processes and coping styles have been identified, including acceptance, mindfulness-based emotion regulation, behavioral and experiential avoidance, and emotion-focused and problem-focused coping (Folkman & Lazarus, 1980; Folkman & Lazarus, 1986). Further, different personality dimensions, such as trait neuroticism and extroversion, have been linked to differential use of emotion regulation processes (Wang, Shi, & Li, 2009). Thus, factors such as trait neuroticism and extraversion may

explain the relationship between attentional bias and negative affect. The respective relationships between neuroticism and negative affect and extraversion and positive affect have been well established in the literature (e.g., Costa & McCrae, 1980), and experimental manipulation of neuroticism and extraversion increase negative and positive affect, respectively (McNiel & Fleeson, 2006). Thus, an examination of trait neuroticism and extraversion and their corresponding coping and emotion regulation processes and their relationships to attentional biases and sustained negative affect could be a worthwhile endeavor.

Another important issue that may have contributed to the nonsignificant findings is the mood induction procedure. In the present study, participants were asked to think about a negative event in their life while they listened to sad music. After the music ended, they were asked to write about the event they had thought about. Research suggests that writing about a negative event may paradoxically result in increases in positive affect, depending on the individual's coping style and the nature of the event (Baker & Berenbaum, 2007). Thus, instructing participants to write about the negative event may have inadvertently counteracted the negative mood state caused by thinking about a negative event while listening to sad music. Indeed, while the mood induction produced a significant increase in sad mood, participants went from a mean VAS score of 20.18 to 42.08 before and after the mood induction, respectively. The VAS is a 100mm line with zero corresponding to no sadness, 50 corresponding to moderate sadness, and 100 corresponding to extreme sadness. Thus, in the present study, the average participant felt slightly sad before mood induction and slightly less than moderately sad after mood induction. The effects of the induction dissipated quickly after the induction, as mean VAS scores were 28.13 and 22.40, after six and 12 minutes, respectively. Further, while negative affect as assessed by the PANAS significantly increased, mean negative affect before and after mood induction

remained in the normative range established by Crawford and Henry (2004; $M = 16.00$ $SD = 5.90$). Positive affect significantly decreased after mood induction, and positive affect scores after the mood induction fell one standard deviation below the normative range for positive affect; however, the mean baseline positive affect score was slightly below Crawford and Henry's norms but still within the average range. The positive affect scale of the PANAS corresponds to degree of pleasurable engagement in the environment. Thus, significant decreases suggest a decrease in pleasurable feelings, but not an increase in sad mood. Overall, the mood induction produced a brief, short-lived effect that may have contributed to the null findings.

One important limitation of this study is that a brief assessment of attentional bias was used at three different time points in an effort to prevent participant fatigue and diminish practice effects. However, the small number of trials used in the present study could have impacted reliability of the generated attentional bias scores and may have contributed to nonsignificant findings. In addition, because mood induction did not have a significant effect on attentional bias, separate sessions should be collapsed into one session. Thus, a single session with a greater number of trials could be employed in future research. Additionally, more accurate means of assessing attentional bias, such as eye-tracking technology, could be used in place of RT assessment.

In sum, emotion regulation did not mediate the relationship between attentional bias and negative affect. Further, contrary to previous findings and cognitive theories, no differences were found in attentional biases between previously depressed and never depressed individuals and how they reacted to a mood induction, and attentional bias did not predict sustained negative affect. However, some differences in how emotions are regulated emerged and were somewhat consistent with previous research. The results of the present study suggest that cognitive

reappraisal may be particularly important in reducing sustained negative affect. Future work might sample mood a few hours or days after mood induction to better capture the effects of some emotion regulation processes, such as executive suppression. Future studies could employ lengthier cognitive tasks without repetition to assess attentional bias and more precise measures, such as eye-tracking, could be utilized in place of RT assessment. Additionally, future studies could examine the extent to which personality variables, coping strategies, and emotion regulation strategies beyond those examined in the present study may influence the association between attentional bias and sustained negative affect in depression. Uncovering factors that cultivate and perpetuate sustained negative affect is important not only for the prevention and treatment of depression, but also for general psychological well-being.

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Table 1
Sample Characteristics

Characteristic	Control	PD	Statistic
Mean age (years; <i>SD</i>)	19.20 (2.20)	19.07 (1.44)	$t(82) = ns$
Mean BDI-II Score (<i>SD</i>)	4.82 (2.61)	5.00 (2.65)	$t(82) = ns$
Mean depression length (weeks)		12.02 (1.87)	
Gender, <i>n</i>			$\chi^2(1) = ns$
Female	27	30	
Male	17	10	
Race, <i>n</i>			Fisher's exact test = <i>ns</i>
White	35	33	
African American	2	0	
Asian	1	2	
Hispanic	4	2	
American Indian	0	0	
Other	2	3	
Income, <i>n</i>			Fisher's exact test = <i>ns</i>
Under \$15,000	7	5	
\$15,000 – 50,000	9	5	
\$50,000 – 100,000	7	14	
\$100,000 – 200,000	15	11	
Over \$200,000	6	5	
Year in school, <i>n</i>			Fisher's exact test = <i>ns</i>
Freshman	29	26	
Sophomore	8	10	
Junior	3	1	
Senior	8	10	

Table 2

Mood Induction Results, by measure

Measure	Statistic
VAS	
Main effect of time	$F(2.32, 82) = 67.70, p < .001, \eta^2_p = .45$
Contrast of VAS1 and VAS2	$F(1, 82) = 112.02, p < .001, \eta^2_p = .58$
Contrast of VAS2 and VAS3	$F(1, 82) = 91.89, p < .001, \eta^2_p = .53$
Contrast of VAS3 and VAS4	$F(1, 82) = 23.99, p < .001, \eta^2_p = .23$
NA	
Main effect of depression history	$F(1, 82) = 3.351, p = .07$
Main effect of time	$F(1, 82) = 21.90, p < .001, \eta^2_p = .21$
PA	
Main effect of time	$F(1, 82) = 64.45, p < .001, \eta^2_p = 0.44$

Table 3

VAS and PANAS Means

Measure	Time 1 ^d	Time 2 ^e	Time 3 ^f	Time 4 ^g
Mean VAS ^a score (SD)	20.18 (20.33)	42.08 (22.74)	28.13 (19.50)	22.40 (19.04)
Mean NA ^b score (SD)	14.67 (4.89)	16.73 (4.96)	-	-
Mean PA ^c score (SD)	28.25 (7.86)	22.92 (9.23)	-	-

Notes: ^aVAS is the Visual Analog Scale. ^bNA is the Negative Affect Scale of the PANAS. ^cPA is the Positive Affect Scale of the PANAS. ^dTime 1 occurred immediately before the mood induction. ^eTime 2 occurred immediately after the mood induction. ^fTime 3 occurred 6 minutes after mood induction. ^gTime 4 occurred 12 minutes after mood induction.

Table 4

Mood Reactivity and Recovery Scores, by depression history

	Control			PD		
	Mean (SD)	Median	Range	Mean (SD)	Median	Range
Reactivity	20.77 (19.30)	18.00	(-13, 63)	23.15 (18.66)	17.00	(-3, 62)
Recovery1 ^a	-13.00 (12.45)	-12.50	(-44, 9)	-15.00 (14.32)	-17.50	(-38, 9)
Recovery 2 ^b	-17.86 (18.45)	-13.50	(-68, 17)	-21.67 (19.68)	-18.50	(-62, 13)

Notes: ^aRecovery1 denotes mood recovery six minutes after mood induction. ^bRecovery 2 denotes mood recovery 12 minutes after mood induction.

Table 5

Attentional Bias Scores (Sad, Happy), by depression history

	Control			PD		
	Mean (SD)	Median	Range	Mean (SD)	Median	Range
Valence, Trial						
Sad (ms), 1	2.40 (61.48)	0.75	(-185, 142.5)	13.79 (45.98)	21.00	(-81, 100)
Happy (ms), 1	5.84 (60.74)	4.75	(-192.5, 135)	7.12 (51.07)	6.75	(-118, 150.5)
Sad (ms), 2	8.28 (68.21)	4.25	(-93.5, 268)	10.05 (52.51)	8.75	(-93, 194)
Happy (ms), 2	.81 (50.03)	1.75	(-156, 105)	8.84 (68.44)	10.00	(-94, 256)
Sad (ms), 3	-7.90 (56.84)	-6.75	(-165, 123)	1.83 (58.88)	5	(-153.5, 151)
Happy (ms), 3	-12.75 (48.07)	-14.5	(-110.5, 89)	12.92 (59.70)	14	(-106, 158)

Table 6

Emotion Regulation Means, by depression history

	Control Mean (SD)	PD Mean (SD)
Overall rumination ^a	43.68 (10.66)	50.68 (10.58)
Ruminative brooding	10.55 (2.61)	11.68 (3.46)
Reflection	9.84 (3.54)	11.63 (2.97)
Cognitive reappraisal	31.02 (6.71)	30.83 (5.95)
Executive suppression	14.16 (5.36)	14.60 (4.61)

Note: ^aOverall rumination describes the total score derived from the RRS.

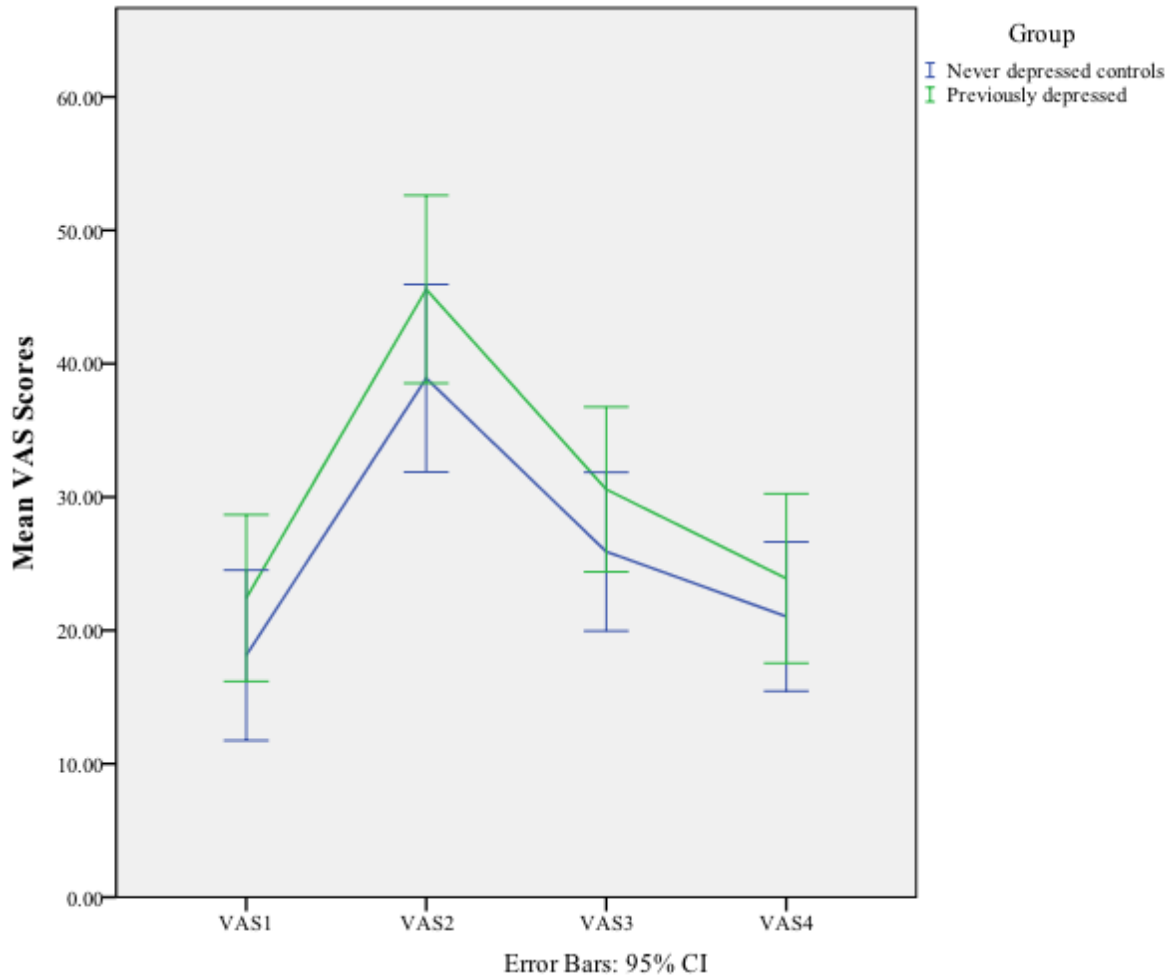
Table 7

Prediction of Mood Recovery, by emotion regulation process

Process	Six minutes ^a	12 minutes ^b
Overall rumination ^c	$\beta = -0.078, t(82) = -0.71, p = .48$	$\beta = -0.13, t(82) = -1.18, p = .24$
Ruminative brooding	$\beta = -0.31, t(82) = -0.65, p = .52$	$\beta = -0.54, t(82) = -0.79, p = .43$
Reflection	$\beta = 0.00, t(82) = 0.001, p = 1.00$	$\beta = -0.091, t(82) = -0.83, p = .41$
Cognitive reappraisal	$\beta = -0.10, t(82) = -0.93, p = .36$	$\beta = -0.28, t(82) = -2.61, p = .011$
Executive suppression	$\beta = 0.18, t(82) = 1.67, p = .099$	$\beta = 0.13, t(82) = 1.15, p = .25$

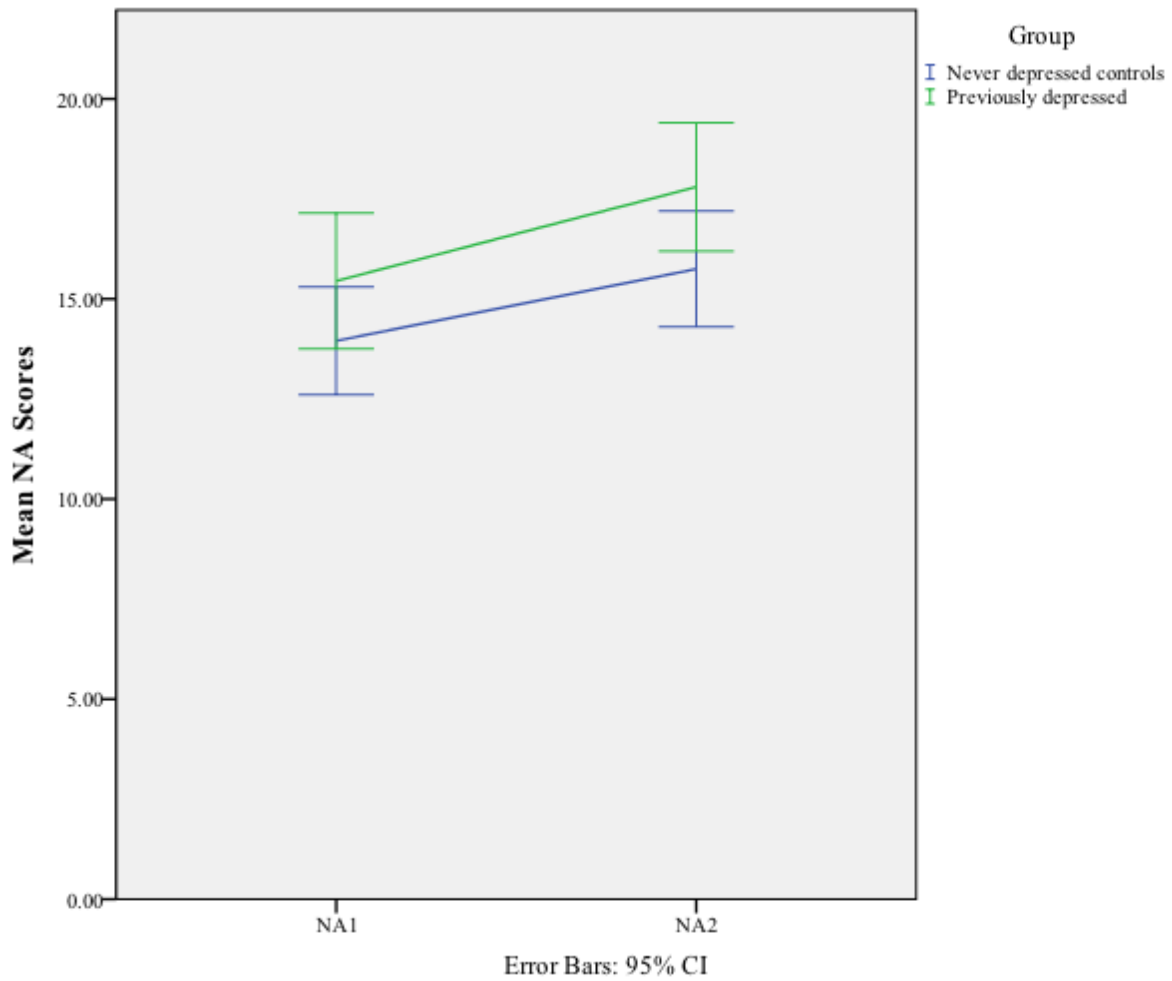
Notes: ^aSix minutes denotes mood recovery observed six minutes after mood induction. ^bTwelve minutes denotes mood recovery observed 12 minutes post mood induction. ^cOverall rumination describes the total score derived from the RRS.

Figure 1
Mean VAS Scores, as a function of time



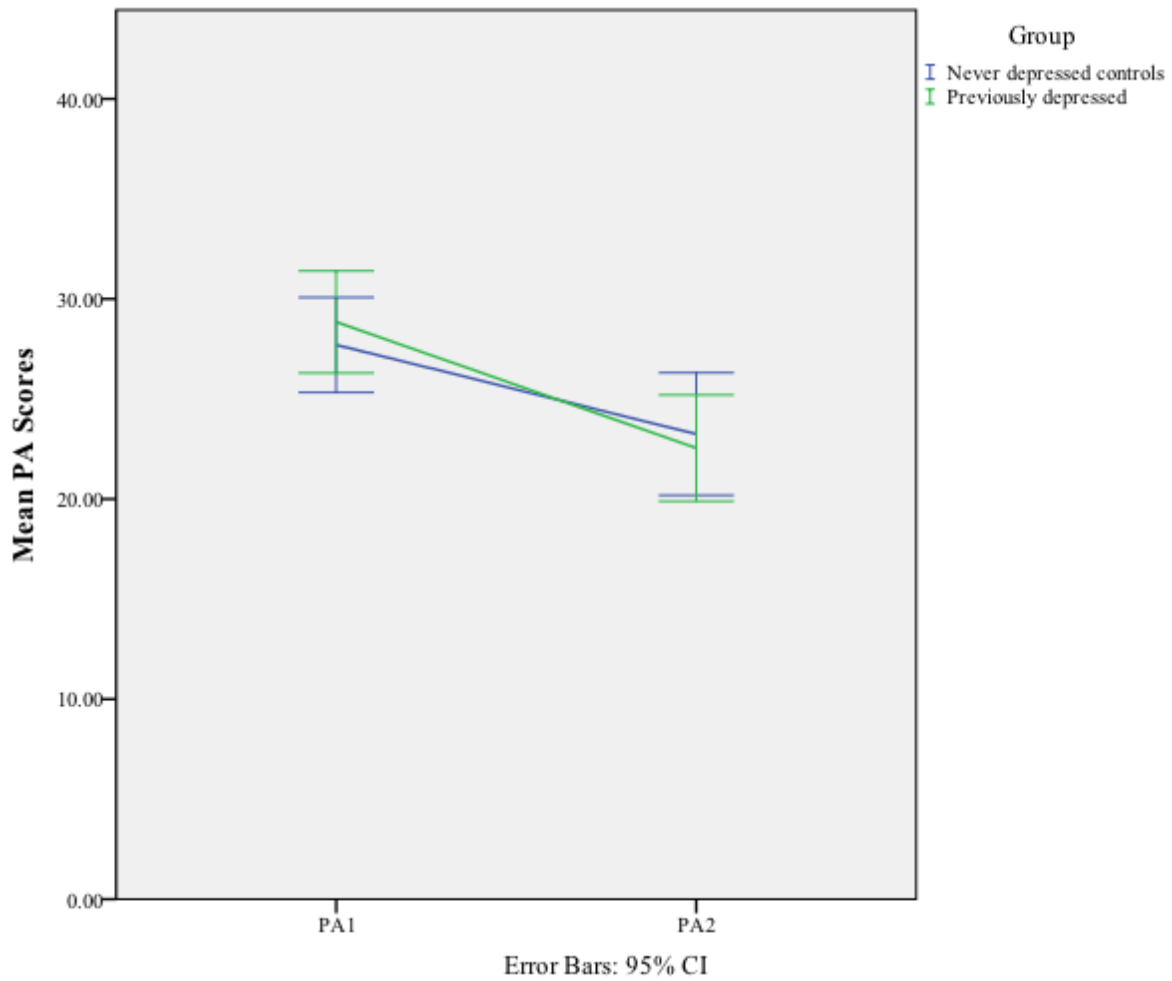
Notes: VAS1 denotes VAS score before mood induction. VAS2 denotes VAS score immediately after mood induction. VAS3 denotes VAS score six minutes after mood induction, and VAS4 denotes VAS score 12 minutes after mood induction.

Figure 2
Mean NA Scores, as a function of time



Notes: NA1 denotes NA score before mood induction. NA2 denotes NA score immediately after mood induction.

Figure 3
Mean PA Scores, as a function of time



Notes: PA1 denotes PA score before mood induction. PA2 denotes PA score immediately after mood induction.

Figure 4
Mean Attentional Bias Scores for Sad Faces, by depression history

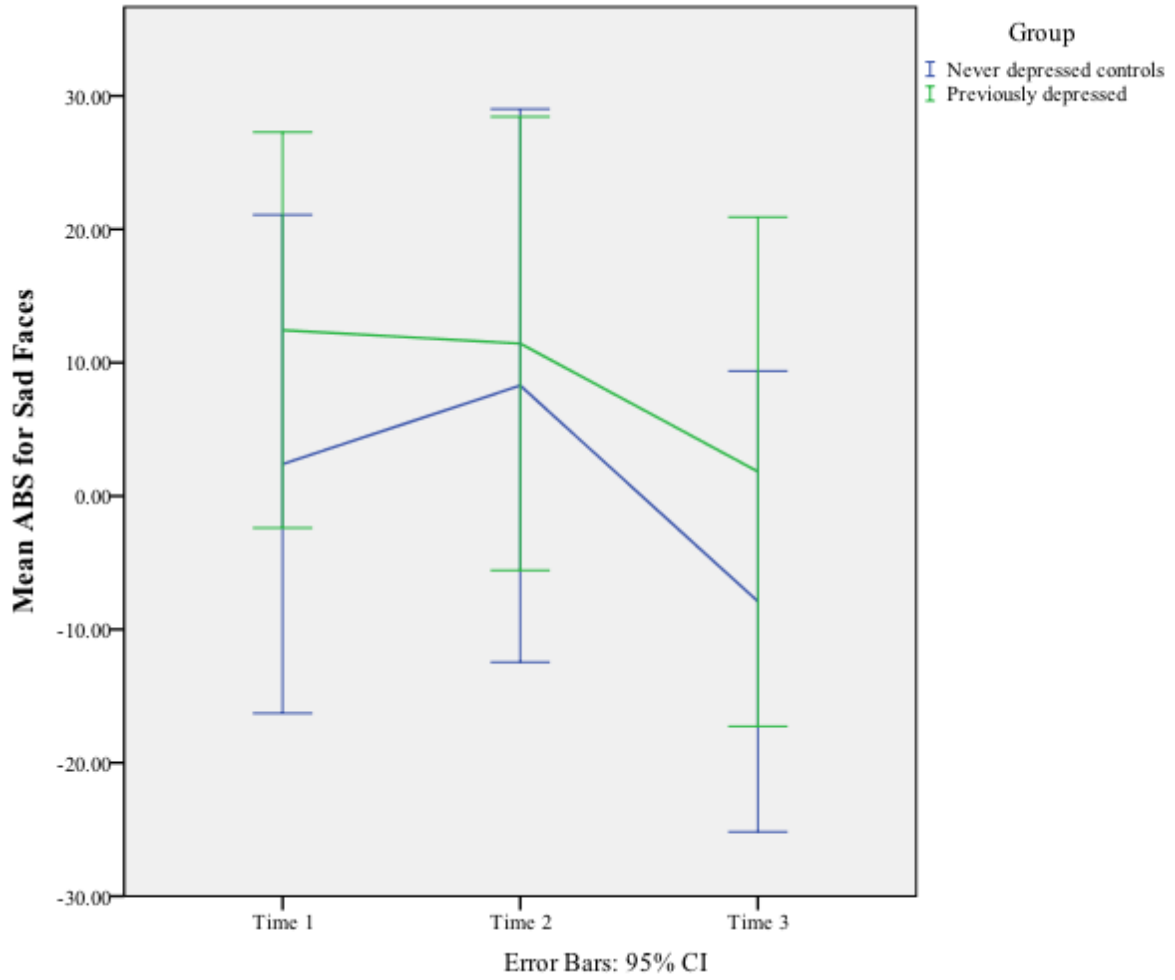


Figure 5
Mean Attentional Bias Scores for Happy Faces, by depression history

