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Emotion Regulation in Depression: Relation to Cognitive Inhibition

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Abstract

Depression is a disorder of impaired emotion regulation. Consequently, examining individual differences in the habitual use of emotion regulation strategies has considerable potential to inform models of this debilitating disorder. The aim of the current study was to identify cognitive processes that may be associated with the use of emotion regulation strategies and to elucidate their relation to depression. Depression has been found to be associated with difficulties in cognitive control and, more specifically, with difficulties inhibiting the processing of negative material. We used a negative affective priming task to assess the relations among inhibition and individual differences in the habitual use of rumination, reappraisal, and expressive suppression in clinically depressed, formerly depressed, and never-depressed participants. We found that depressed participants exhibited the predicted lack of inhibition when processing negative material. Moreover, within the group of depressed participants, reduced inhibition of negative material was associated with greater rumination. Across the entire sample, reduced inhibition of negative material was related to less use of reappraisal and more use of expressive suppression. Finally, within the formerly depressed group, less use of reappraisal, more use of rumination, and greater expressive suppression were related to higher levels of depressive symptoms. These findings suggest that individual differences in the use of emotion regulation strategies play an important role in depression, and that deficits in cognitive control are related to the use of maladaptive emotion regulation strategies in this disorder.

Keywords

Depression; Rumination; Inhibition; Emotion Regulation

Depression is a disorder of impaired emotion regulation. Sustained negative affect and a persistent reduction in positive affect are the hallmark features of a diagnosis of a major depressive episode. Indeed, theorists have suggested that depression vulnerable and non-vulnerable people do not differ primarily in their initial response to a negative event, but in their ability to recover from the ensuing negative affect (e.g., Teasdale, 1988). From this perspective, we need to examine factors that impair or facilitate this recovery. Thus, individual differences in the habitual use of specific emotion regulation strategies may play an important role in the onset and maintenance of depression. It is likely that there are a number of factors that affect emotion regulation in depression, including knowledge of effective strategies and motivation to implement them (Campbell-Sills & Barlow, 2007; Kring & Werner, 2004). In the present study, we focus on one specific factor that may be associated with individual

differences in the habitual use of specific emotion regulation strategies: individual differences in the cognition inhibition of emotional material.

Surprisingly few studies have examined the use and effectiveness of emotion regulation strategies in depression. Moreover, most of these studies have included participants who obtain high scores on depression inventories instead of diagnosed samples. Nevertheless, the findings of these studies support the claim that more frequent use of certain strategies (e.g., expressive suppression, thought suppression, rumination, catastrophizing) and less frequent use of other strategies (e.g., reappraisal, self-disclosure) are related to levels of symptoms of depression and anxiety (e.g., Campbell-Sills, Barlow, Brown, & Hofman, 2006; Garnefski & Kraaij, 2006; Garnefski & Kraaij, 2007; Gross & John, 2003). In addition, recent studies suggest that impaired emotion regulation not only characterizes currently depressed people, but is also evident following recovery from this disorder (Ehring, Fischer, Schueller, Boesterling, & Tuschen-Caffier, 2008). Although it is difficult to categorize a specific regulation strategy as maladaptive without taking into account the context in which the strategy is used, numerous studies have demonstrated that the habitual use of particular strategies, primarily reappraisal and expressive suppression, is associated with positive and negative outcomes, respectively. Whereas cognitive reappraisal involves the re-interpretation of the emotion-eliciting situation in a way that changes the emotional response, expressive suppression involves inhibiting the behavioral expression of the emotion experience. Indeed, recent studies demonstrate that voluntary changes of the interpretation of a situation can change the intensity of an emotional reaction (Gross, 1998; Ochsner, Bunge, Gross, & Gabrieli, 2002; Ochsner et al., 2004). Moreover, it is noteworthy that emotion regulation strategies that rely less on changes in cognition, such as inhibition of emotion expression, have been consistently found to be less effective than are strategies that target cognition (Gross, 1998; Gross & Levenson, 1997). Importantly, habitual use of reappraisal vs. expressive suppression has been shown to be associated with the experience and expression of greater positive affect and lesser negative affect, better interpersonal functioning, and increased well-being (Gross & John, 2003).

The majority of studies investigating emotion regulation in depression have focused on the use of rumination. According to Nolen-Hoeksema and her collaborators, rumination is a particularly detrimental response to negative affect that hinders recovery from negative mood and prolongs depressive episodes (Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008). Rumination is a *style* of thought rather than just negative *content* (Nolen-Hoeksema, 1991; Nolen-Hoeksema et al., 2008); consequently, rumination is defined by the process of recurring thoughts and ideas often described as a “recycling” of thoughts, and not necessarily by the content of these recurring thoughts. Recent studies have identified two subcomponents of rumination. Reflective pondering is proposed to be an adaptive response to negative events and mood states; in contrast, brooding is proposed to be maladaptive and most closely related to depression risk (Treynor, Gonzalez, & Nolen-Hoeksema, 2003). In an extensive program of experimental and correlational studies, Nolen-Hoeksema and colleagues investigated rumination in depression and dysphoria and analyzed how this response style exacerbates sad moods and predicts future depressive episodes (e.g., Morrow & Nolen-Hoeksema, 1990). Not only are these ruminative thoughts a debilitating symptom of depression, but they have also been associated with vulnerability to the onset of depression, the recurrence of depressive episodes, and the maintenance of negative affect (Nolen-Hoeksema, 2000; Nolen-Hoeksema et al., 2008; Roberts, Gilboa, & Gotlib, 1998). Little is known, however, about cognitive processes that are associated with individual differences in the frequency of use of rumination or other emotion regulation strategies.

The present study was designed to examine whether individual differences in inhibiting the processing of emotional material are related to the use of specific emotion regulation strategies. Investigators examining the interaction of cognition and emotion have proposed that the

experience of negative mood is generally associated with the activation of mood-congruent representations in working memory (Isen 1984; Siemer, 2005). Thus, negative mood has been found to be related to more frequent negative thoughts, to selective attention to negative stimuli, and to greater accessibility of negative memories (Blaney, 1986; Matthews & MacLeod, 2005; Rusting, 1998). This research has also demonstrated, however, that changes in cognition due to negative mood are usually transient, and mood-congruent cognitions are often replaced quite quickly by thoughts and memories that serve to regulate and repair the mood state (Erber & Erber, 1994; Parrott & Sabini, 1990; Rusting & DeHart, 2000). The critical question, therefore, is why, in response to negative mood, some people fail to regulate and instead initiate a self-defeating cycle of increasingly negative ruminative thinking and intensifying negative affect. If changes in mood are, in fact, associated with activations of mood-congruent material in working memory, individual differences in the ability to control the contents of working memory might play an important role in the use of certain emotion regulation strategies. Specifically, difficulties controlling the access of mood-congruent material to working memory may be associated with increased ruminative thinking, with greater use of other forms of maladaptive emotion regulation and, therefore, with difficulties recovering from negative mood (Joormann, Yoon, & Siemer, in press).

Working memory is a limited-capacity system that provides temporary access to a select set of representations in the service of current cognitive processes (Cowan, 1999; Miyake & Shah, 1999). Given the capacity limitation of this system, it is important that the contents of working memory be updated efficiently, a task controlled by executive processes and, more specifically, by inhibition (e.g., Friedman & Miyake, 2004; Hasher, Zacks, & May, 1999; Tipper, 2001). In fact, Hasher and Zacks (1988) proposed that the efficient functioning of working memory depends on inhibitory processes that limit the access of information and that update working memory by removing information that is no longer relevant. It is noteworthy, therefore, that several researchers have suggested that rumination and depression are associated with deficits in executive function, particularly with deficits in inhibition (Hertel, 1997, 2004; Joormann, 2005; Linville, 1996). Importantly, inhibitory dysfunction, and more specifically, reduced control of access of negative cognitions and memories that were activated by a negative mood state to working memory, could be associated with rumination. In addition, given the capacity limitations of working memory, inhibitory dysfunction could be associated with difficulties attending to and processing new information. Difficulties ignoring mood-congruent material and processing mood-incongruent material may hinder individuals ability to attend to novel aspects of the emotion-eliciting situation and to re-interpret the emotion-eliciting cues. Thus, reduced cognitive inhibition is likely to be associated with less use of cognitive reappraisal.

A small number of investigators have examined associations among inhibition of emotional material, depression, and habitual use of rumination by using a modified negative priming task (Joormann, 2004; Goeleven, De Raedt, Baert, & Koster, 2006; Frings, Wentura, & Holtz, 2007). In this task, participants are instructed to respond to a target stimulus while ignoring a simultaneously presented emotional stimulus that is clearly marked as to-be-ignored and irrelevant to the task; on the subsequent trial, the to-be-ignored emotional stimulus may become the target. Inhibition is operationalized as the differential delay between responding to a previously ignored stimulus and responding to a novel stimulus (Hasher et al., 1999; Wentura, 1999). Joormann (2006) found that participants who scored high on a self-report measure of rumination exhibited reduced negative priming when processing emotional distractors, a finding that remained significant after controlling for level of depressive symptoms. Joormann (2004) further demonstrated that dysphoric participants and participants with a history of depressive episodes exhibit reduced negative priming of negative material. Frings et al. (2007) recently replicated these findings using a slightly modified negative priming task in a group of dysphoric participants. Finally, Goeleven et al. (2006) replicated these findings using a negative priming task with emotional faces. These investigators demonstrated that, compared

to nondepressed controls, currently and formerly depressed participants show reduced negative priming when responding to sad facial expressions but did not differ from controls when processing happy expressions.

The goals of the present study were to assess cognitive inhibition in depression and to examine whether individual differences in inhibition are related to the habitual use of specific emotion regulation strategies. This study extends the scope of previous research that has examined the association between inhibition and rumination by incorporating two emotion regulation strategies that likely play a role in increasing vulnerability to depression: reappraisal and expressive suppression. We predict that reduced cognitive inhibition when processing negative material is related not only to more frequent use of rumination, but also to less frequent use of reappraisal. Inhibition allows people to stop the processing of the activated mood-congruent material in working memory and to reorient their attention to other aspects of the situation which should be associated with increased reinterpretation of the emotion-eliciting situation to change the ensuing affect. Given that expressive suppression depends less on changes in cognition than do rumination or reappraisal, we expected to find no relation between inhibition and the use of expressive suppression. In addition, the current study assesses trait and state effects by comparing samples of currently depressed and remitted depressed participants. We predict that both currently and remitted depressed participants are characterized by reduced cognitive inhibition when processing negative material, and that individual differences in cognitive inhibition are related to a more frequent use of maladaptive emotion regulation strategies, specifically rumination, and to a less frequent use of reappraisal.

Method

Participants

Three groups of participants took part in the study: participants diagnosed with a current Major Depressive Disorder (MDD); participants who had experienced at least one diagnosable depressive episode in their lives but were currently remitted (RMD); and never-disordered controls (CTL). Participants were solicited through advertisements posted in numerous locations within the local community (e.g., internet bulletin boards, university kiosks, supermarkets). Participants' responses to a telephone interview provided initial selection information. This phone screen established that participants were fluent in English and were between 18 and 60 years of age. Participants were excluded for severe head trauma and learning disabilities, as well as for psychotic symptoms, bipolar disorder, and alcohol or substance abuse within the past six months. This telephone interview was also used to identify individuals who were likely to meet criteria for inclusion in one of the three groups. Those individuals were invited to come to the laboratory for a more extensive interview.

Trained interviewers administered the Structured Clinical Interview for the DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1995) to these individuals during their first session in the study. This interview schedule assesses DSM-IV current and lifetime diagnoses for anxiety, mood, psychotic, alcohol and substance use, somatoform, and eating disorders. The SCID has demonstrated good reliability for the majority of the disorders covered in the interview (e.g., J. B. Williams et al., 1992). SCID-I interviewers had previous experience with administering structured clinical interviews and were trained specifically to administer the SCID-I interview. Our team of interviewers achieved excellent interrater reliability for a major depressive episode ($\kappa = 1.00$) and nonpsychiatric controls ($\kappa = 0.92$; Gotlib et al., 2004a).

Participants were included in the depressed group if they met the Diagnostic and Statistical Manual of Mental Disorders, 4th ed. (DSM-IV; American Psychiatric Association, 1994) criteria for MDD. Participants were included in the RMD group if they met DSM-IV criteria for a past major depressive episode. In addition, a slightly modified version of this interview

was used to determine whether each participant in the RMD group had fully recovered from depression, using guidelines recommended by the NIMH Collaborative Program on the Psychobiology of Depression (e.g., Keller et al., 1992): 8 consecutive weeks with no more than 2 symptoms of no more than a mild degree (i.e., ratings of 1 [no symptoms] or 2 [minimal symptoms, no impairment]). The never-disordered control group consisted of individuals with no current diagnosis and no history of any Axis I disorder. A total of 101 individuals (22 MDD, 47 RMD, and 32 HC) participated in this study.

Questionnaires

Depressive symptoms and general functioning—Participants completed the Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996), a 21-item, self-report measure of the severity of depressive symptoms. The reliability and validity of the BDI-II have been well documented (Beck et al., 1996). In addition, the SCID interviewers provided ratings of general functioning on the General Assessment of Functioning Scale (GAF; range 0–100) that is part of the DSM. We also asked participants to estimate the number of previous depressive episodes that they had experienced and assessed whether they were currently receiving psychological or pharmacological treatment.

Rumination scales—We used the 22-item Ruminative Responses Scale (RRS, Treynor et al., 2003) to assess how participants tend to respond to sad feelings and symptoms of dysphoria. The RRS assesses responses to dysphoric mood that are focused on the self (*think about all your shortcomings, failings, faults, mistakes*), on symptoms (*think about how hard it is to concentrate*), or on possible consequences and causes of moods (*analyze recent events to try to understand why you are depressed*) using a 4-point scale (almost never to almost always). Previous studies have shown good test-retest reliability and acceptable convergent and predictive validity (Nolen-Hoeksema & Morrow, 1991; Nolen-Hoeksema, Parker, & Larsen, 1994; Treynor et al., 2003). The division of the RRS into brooding and reflective pondering subscales was modeled after Treynor et al. (2003), who found both subscales to have acceptable internal consistencies and retest-reliabilities.

Reappraisal and Suppression—Finally, we used the Emotion Regulation Questionnaire (ERQ; Gross & John, 2002) to assess the frequency with which people use cognitive reappraisal or expressive suppression to regulate affect. The ERQ consists of two subscales that measure habitual use of reappraisal or suppression using a 7-point scale. The reappraisal subscale (*I control my emotions by changing the way I think about the situation I'm in*) consists of 6 items and the suppression subscale (*I keep my emotions to myself*) consists of 4 items. Previous studies have shown acceptable internal consistencies that are slightly higher for the reappraisal than for the suppression subscale (Gross & John, 2003).

Negative Affective Priming Task (NAP)

Stimuli—Words from the Affective Norms of English words (ANEW; Bradley & Lang, 1999), which lists valence and arousal ratings for over 1000 English adjectives, verbs, and nouns on 9-point scales, were used as stimuli. Adjectives with a rating of 4 or less were examined for possible inclusion in the negative valence category, adjectives with a rating between 4 and 6 were examined for inclusion in the neutral category and adjectives with a rating over 7 were examined for inclusion in the positive valence category. From these lists we selected words taking care to ensure that the positive and negative words did not differ in arousal ratings or word length. The final set of 56 positive words had an average valence rating of $M = 7.55$ ($SD = 0.47$) and an arousal rating of $M = 5.37$ ($SD = 1.03$), while the final set of 56 negative words had an average valence rating of $M = 2.41$ ($SD = 0.45$) and an average arousal rating of $M = 5.46$ ($SD = 1.03$). The final set of 16 neutral words had an average valence rating of $M = 5.27$ ($SD = 0.60$) and an average arousal rating of $M = 4.07$ ($SD = 1.07$). Neutral,

positive, and negative words did not differ in average word length, $F(2, 125) < 1$, *ns*. Group differences were found for arousal, $F(2, 125) = 11.90$, $p < .01$ and valence, $F(2, 125) = 1566.78$, $p < .01$. Whereas positive and negative words did not differ in arousal ratings, $t(110) < 1$, *ns*, positive and negative words were higher in arousal compared to the neutral words (positive: $t(70) = 4.39$, $p < .01$; negative: $t(70) = 4.72$, $p < .01$).

Design and Procedure—Consistent with standard negative priming designs, the NAP-design consists of consecutive pairs of trials, a prime trial and a test trial (Joormann, 2004). In each trial two adjectives are presented, a target and a distractor, along with the instruction to ignore the distractor and to respond to the target. It is important to note that participants are not aware of the distinction between prime trials and test trials. In the negative priming condition, distractors presented in the prime trial (prime-trial distractors) and targets presented in the test trial (test-trial targets) are related by shared valence. In the control condition, the prime-trial distractor and test-trial target words are unrelated. Prime and test trials in the negative priming condition each consist of one positive and one negative word, and either the positive or the negative word is the target. Negative priming and control conditions do not differ in the valence of the distractor or target in the test trials; in the control condition, however, the prime-trial distractor is a neutral word.¹ To the extent that the inhibition of the (valence of) the distractor of the prime trial is still activated, responses to same-valence targets in the test trial should be delayed. Thus, the stronger the inhibition, the longer the delay in the priming condition compared to the control condition.

Participants were told that two words were going to be presented simultaneously in the upper and lower half of the computer screen, one word in blue and one word in red; they were asked to attend only to the blue word and to ignore the red word. Participants were told further to decide whether the blue word was positive or negative as quickly and as accurately as possible by pressing an assigned key on the computer keyboard. Each trial proceeded as follows: first, a fixation cross was presented for 500 ms. Immediately following the disappearance of the fixation cross, the prime trial (a distractor word in red and a target word in blue) were presented simultaneously on the computer screen until the participants responded. The letters were 1 cm in size. There was a gap of 1 cm between the words and the spatial position of the target and distractor word was randomly assigned on each trial. Following the response to the prime trial, the fixation cross was presented and, after a delay of 500 ms, the next trial (test trial) began. Again, two words were presented and participants were asked to respond to the word presented in blue and to ignore the word presented in red. Either the test-trial target was related to the prime-trial distractor (negative priming condition, NP) or the test-trial target and the prime-trial distractor were unrelated (control condition, C). Reaction times and responses were recorded. After the instructions were presented on the computer screen, participants completed 10 practice trials. Following the practice trials, participants completed 320 trials, arranged in 5 blocks of 64 prime and test trials in addition to filler trials. The sequence of trials within blocks was newly randomized for each participant. For each participant a random sample of words was selected from the word lists. The responses to the prime and test trials were recorded, but only the responses to the test trials were analyzed.

¹Given previous criticism of the NAP design (e.g., Frings et al., 2007), in the present study we included several filler trials that were used to control for alternative explanations of the negative priming effect. Specifically, we compared control conditions in which the distractor in the prime trial was neutral to control conditions in which the distractor in the prime trial was of the opposite valence of the target in the prime trial. We compared both control conditions and found that they did not differ significantly from each other. In addition, we obtained negative priming effects and group differences both when examining each of the control conditions separately and when collapsing across the different control conditions. Details regarding these additional analyses are available upon request.

Results

Participant Characteristics and Group Differences in Emotion Regulation Strategies

Demographic characteristics of the three groups of participants and scores on the emotion regulation scales are presented in Table 1. Demographic and questionnaire data were missing for 1 MDD participant, 1 CTL participant and 3 RMD participants. As is evident from the Table, the three groups did not differ significantly in age, $F(2, 94) < 1$, or education, $\chi^2(2, 94) = 1.23, p > .05$. As expected, the groups did differ in their BDI scores, $F(2, 94) = 130.64, p < .01$: the MDD group had significantly higher BDI scores than did both the CTL ($d = 3.76$) and the RMD participants ($d = 3.41$), both $ps < .05$, who did not differ significantly from each other, $t(74) = 1.38, p > .05$. Seven participants in the MDD group reported current comorbid disorders, including eating disorders and anxiety disorders.

As can be seen in Table 1, the three groups of participants differed in their tendency to respond to negative events with overall rumination, $F(2, 94) = 17.82, p < .01$, and with brooding, $F(2, 94) = 27.02, p < .01$, and reflection, $F(2, 94) = 5.28, p < .01$. MDD participants reported being more prone to ruminate than did both CTL (rumination: $t(50) = 7.45, p < .01, d = 2.06$; brooding: $t(50) = 7.44, p < .01, d = 2.07$; reflection: $t(50) = 2.40, p < .02, d = 0.67$) and RMD participants (rumination: $t(64) = 2.30, p < .03, d = 0.60$; brooding: $t(64) = 5.43, p < .01, d = 1.44$). MDD and RMD participants did not differ, however, in their tendency to use reflection, $t(64) < 1, ns$. Finally, RMD participants were more likely to ruminate than were CTL participants (rumination: $t(74) = 3.95, p < .01, d = 0.91$; brooding: $t(74) = 2.05, p < .05, d = 0.47$; reflection: $t(74) = 3.09, p < .01, d = 0.72$). The three groups of participants also differed in their reported use of reappraisal, $F(2, 94) = 9.76, p < .01$, but not of expressive suppression, $F(2, 94) = 1.34, p > .05$. MDD participants endorsed less use of reappraisal than did CTL, $t(50) = 4.03, p < .01, d = 1.15$, and RMD, $t(64) = 4.10, p < .01, d = 1.26$, participants, who did not differ significantly from each other $t(74) < 1, ns$. In sum, whereas MDD and RMD participants differ from CTL participants in their use of rumination, only MDD participants were less prone to use reappraisal than were RMD and CTL participants, and no group differences were obtained for expressive suppression.

Negative Priming Task

Because performance on the negative priming task is generally associated with low error rates, we did not expect to find group differences in error rates in the present study. The mean percentages of correct responses are presented in Table 1. As expected, the overall number of correct responses was high (CTL: 97%; MDD: 97%; RMD: 97%). We conducted a mixed effects analyses of variance (ANOVA) to examine differences in the number of correct responses as a function of group and experimental condition. A three-way (group [CTL, MDD, RMD] by condition [NP, C] by valence [positive, negative]) ANOVA yielded only a significant main effect of valence, $F(1, 98) = 8.81, p < .01$; no other main effects or interactions were significant.

We restricted our analyses of reaction times to trials in which participants made correct responses. Extreme RT scores (below 300 and above 2000 ms) were considered as outliers and eliminated from the analyses (less than 10% of all RTs, no difference among groups; $F(1, 98) < 1, ns$). Mean response latencies for participants in the three groups are presented in Table 1. To examine group differences in negative priming for positive and negative words, we conducted a three-way (group [CTL, MDD, RMD] by condition [NP, C] by valence [positive, negative]) ANOVA. This analysis yielded a significant main effect of condition, $F(1, 98) = 42.38, p < .01, \eta_p^2 = .30$, a significant interaction of group and valence, $F(2, 98) = 3.50, p < .03, \eta_p^2 = .07$, and the predicted significant three-way interaction of group, condition, and valence, $F(2, 98) = 6.21, p < .01, \eta_p^2 = .11$. To examine the three-way interaction, separate ANOVAs were

conducted for each group. For the CTL participants, the two-way ANOVA yielded significant main effects of valence, $F(1,31) = 10.55$, $\eta_p^2 = .25$, and condition, $F(1,31) = 35.30$, $\eta_p^2 = .53$, both $ps < .01$. CTL participants were faster to respond to positive than to negative stimuli and were slower to respond to the words presented in the negative priming condition than in the control condition. The ANOVA conducted with the MDD participants yielded a significant main effect of condition, $F(1,21) = 9.85$, $p < .01$, $\eta_p^2 = .32$, and the predicted significant interaction of valence and condition, $F(1,21) = 7.81$, $p < .01$, $\eta_p^2 = .27$. Follow-up *t*-tests indicated that the MDD participants were slower to respond to negative priming than to control trials only when positive, $t(21) = 3.72$, $p < .01$, but not when negative words were presented, $t(21) < 1$, *ns*. Thus, MDD participants showed the expected lack of negative priming for negative words. Finally, the ANOVA with the RMD participants yielded a significant main effect for condition, $F(1,46) = 12.39$, $p < .01$, $\eta_p^2 = .21$, and a significant interaction of valence and condition, $F(1,46) = 4.72$, $p < .04$, $\eta_p^2 = .09$. Follow-up *t*-tests indicated that RMD participants showed negative priming for negative words, $t(46) = 4.26$, $p < .01$, but not for positive words, $t(46) = 1.02$, $p > .05$.

Finally, to evaluate group differences in negative priming for positive and negative words, individual bias scores were calculated by subtracting the reaction times in the control conditions from the reaction times in the corresponding negative priming conditions (Joormann, 2004). A two-way (group repeated over valence) ANOVA yielded a significant interaction of group and valence, $F(2,98) = 6.21$, $p < .01$, $\eta_p^2 = .11$; no other main effects or interactions were significant, all $F_s < 1$. These results are presented in Figure 1. A positive value represents a slowing in the negative priming condition compared to the control condition and represents negative priming. Figure 1 shows that all groups exhibit negative priming with the critical exceptions of MDD participants when responding to negative words and RMD participants when responding to positive words.

Negative Priming and Emotion Regulation

An important aim of this study was to investigate the relation between individual differences in cognitive inhibition and the habitual use of discrete emotion regulation strategies. To this end, we correlated negative priming scores with measures of emotion regulation, first, across the full sample, and then within each participant group. Interestingly, we obtained no significant correlations among the use of suppression, reappraisal, and rumination either in the full sample or in the subsamples with two exceptions; within the full sample suppression and rumination were correlated, $r(97) = .23$, $p < .05$, and for RMD participants less reappraisal was related to more expressive suppression, $r(45) = -.32$, $p < .05$. Also, no significant correlations among negative priming scores and BDI scores were found with the exception of a significant correlation between BDI scores and NAP scores for negative material in the full sample, $r(97) = -.22$, $p < .05$.

For the full sample, individual differences in the inhibition of negative material were significantly correlated with the tendency to respond to negative events with reflection, with increased use of reappraisal, and with decreased use of expressive suppression (see Table 2). In addition, rumination, and specifically the brooding subcomponent of rumination, decreased use of reappraisal, and increased use of expressive suppression all were correlated with increased levels of depressive symptoms. To take individual differences in depression severity into account, we further conducted hierarchical linear regression analyses entering BDI scores in step 1 and negative priming scores for negative material in step 2. As we described above, we found no significant correlations among the different emotion regulation strategies, with the exception of a negative correlation between suppression and rumination. Therefore, we did not control for overlap with other emotion regulation measures, with the exception of controlling for suppression when predicting rumination and vice versa. Including BDI scores

in step 1 did not affect the association between negative priming for negative material (NP-neg) and reappraisal ($R^2 = .30, p < .01$; Step 2: $\Delta R^2 = 0.4, p < .03$; $\beta(\text{BDI}) = -.47, p < .01$; $\beta(\text{NP-neg}) = .20, p < .03$). In addition, the association between reflection and NP-neg remained significant when BDI scores and suppression scores were entered in step 1 ($R^2 = .11, p < .02$; Step 2: $\Delta R^2 = .06, p < .02$; $\beta(\text{BDI}) = .14, n.s.$; $\beta(\text{suppression}) = .21, p < .05$; $\beta(\text{NP-neg}) = .26, p < .01$). Finally, the association between suppression and NP-neg remained significant when BDI and rumination scores were entered in step 1 ($R^2 = .11, p < .02$; Step 2: $\Delta R^2 = .05, p < .03$; $\beta(\text{BDI}) = .06, n.s.$, $\beta(\text{rumination}) = .19, n.s.$, $\beta(\text{NP-neg}) = -.24, p < .03$). In sum, the relation of inhibition with reflection, reappraisal, and suppression was not due to individual differences in depression severity or due to overlap with individual differences in the use of other emotion regulation strategies.

In the CTL group, the ability to inhibit the processing of negative material was related to less expressive suppression. As we described above, we found no significant correlations among the emotion regulation measures in the control group; these measures, therefore, were not included in the regression analysis. The relation between NP-neg and suppression remained significant when BDI scores were entered in step 1 ($R^2 = .21, p < .04$; Step 2: $\Delta R^2 = .21, p < .01$; $\beta(\text{BDI}) = .01, n.s.$; $\beta(\text{NP-neg}) = -.46, p < .01$). In the MDD group, inhibition of negative material was associated with decreased rumination and, specifically, with brooding (see Table 3); inhibition of positive material within the MDD group was associated with increased reflection. In addition, increased levels of rumination and brooding and decreased use of reappraisal were associated with increased levels of depressive symptoms. No significant correlations among emotion regulation measures were found in the MDD group. To control for depression severity, we again conducted hierarchical regression analyses entering BDI scores in step 1. The relation between negative priming for negative material and rumination remained significant ($R^2 = .49, p < .01$; Step 2: $\Delta R^2 = .07, p < .05$; $\beta(\text{BDI}) = .60, p < .01$; $\beta(\text{NP-neg}) = -.29, p < .05$). In addition, the relation between brooding and negative priming for negative material remained significant when BDI scores were entered in step 1 ($R^2 = .30, p < .04$; Step 2: $\Delta R^2 = .12, p < .05$; $\beta(\text{BDI}) = .37, p < .05$; $\beta(\text{NP-neg}) = -.35, p < .05$). Finally, in the RMD group there were no significant correlations among indices of inhibition and emotion regulation. Those RMD participants who were more likely to ruminate and to reflect, however, reported higher BDI scores. Finally, less use of reappraisal and greater use of expressive suppression were significantly correlated with higher BDI scores in this group².

Discussion

Sustained negative affect is a hallmark feature of depressive episodes. Consequently, examining individual differences in the habitual use of emotion regulation strategies and investigating the association between the use of specific strategies and individual differences in cognitive processes that may underlie difficulties in emotion regulation has considerable potential to improve our understanding of depression. Previous studies have reported that rumination in response to negative events and negative mood states increases the risk for the onset of a depressive episode (see Nolen-Hoeksema et al., 2008 for a recent review). In this study we extended this work by focusing on the relation between cognitive processes, specifically inhibition of negative material, and the use of specific emotion regulation strategies and by assessing the use of reappraisal and expressive suppression in addition to rumination.

²We correlated our main constructs (inhibition, emotion regulation) with a number of potential third variables that may affect the relation between inhibition and emotion regulation. Specifically, we computed correlations with number of previous depressive episodes in the MDD and RMD groups, as well as with current psychological and pharmacological treatment in these two groups. Finally, we investigated correlations with GAF ratings for all of our participants. Only two significant correlations were obtained. GAF was correlated with reappraisal in the MDD group ($r = -.49$) and with suppression in the RMD group ($r = -.30$). Given that we found no significant correlation between reappraisal/suppression and inhibition in either the MDD or the RMD groups, we did not include GAF scores as a control variable in our analyses.

Reduced inhibition of negative irrelevant material has been proposed to underlie rumination and may also be associated with the use of other emotion regulation strategies such as reappraisal (Joormann, Yoon, & Siemer, in press). Our results replicate previous work that has found depression to be associated with reduced inhibition (Joormann, 2004; Goeleven et al., 2006). Currently depressed participants were the only group in this study to show a lack of negative priming when responding to negative material in the NAP task. Contrary to our predictions and to previous studies, remitted depressed participants did not differ from control participants in their inhibition of negative material. These participants did differ from the control group, however, in their inhibition of positive material. These findings suggest that reduced inhibition when processing negative material is present only during acute depressive episodes and remits along with other symptoms of depression following recovery. Research on cognitive biases in previously depressed participants, however, has demonstrated that a stressor or some other form of priming strategy such as a negative mood induction or a self-focus manipulation may be necessary to observe biases in cognitive processes in these participants (Segal & Ingram, 1994; see Scher, Ingram, & Segal, 2005, for a recent review). Indeed, in previous work investigators have found attentional biases in remitted depressed participants (Ingram et al., 1994); moreover, recent studies have found that RMD participants exhibit increased amygdala activation when recalling mood-congruent memories after a mood induction (Ramel et al., 2007). These findings suggest that remitted participants do differ from control participants in their processing of emotional material. Our RMD group, however, also differed from remitted depressed participants examined in most previous studies. In contrast to studies that include in their remitted group individuals who are not currently diagnostically depressed but who have a history of depression, we conducted a clinical interview to establish full recovery at the symptom level. Our findings suggest that recovery from depression involves changes in the processing of both negative and positive material. Indeed, the RMD participants inhibited negative material but not positive material, indicating the operation of a positive bias in this group. Because these results were unexpected, replication is required. In addition, we did not include a negative mood induction. Future studies, therefore, should compare a neutral and negative mood group or use some other form of priming to investigate the possibility that when under stress, remitted depressed participants exhibit inhibition deficits similar to those found to characterize the currently depressed group.

We also replicated previous findings, obtained mostly with non-diagnosed samples, of differences between depressed and nondepressed participants in the habitual use of emotion regulation strategies (Campbell-Sills et al., 2006; Garnefski & Kraaij, 2006; Garnefski & Kraaij, 2007; Gross & John, 2003). In the present study MDD participants were more likely than were CTL and RMD participants to endorse the habitual use of rumination and less likely to use reappraisal. Interestingly, previously and currently depressed participants did not differ in their use of reflective pondering. In addition, whereas RMD participants were less likely to use rumination than MDD participants, they were still more likely than were CTL participants to report using rumination. Previous studies have also shown that RMD participants are prone to use maladaptive strategies such rumination (Ehring et al., 2008; Joormann, Dkane, & Gotlib, 2006). In addition, rumination, and specifically, reflective pondering, was correlated with BDI scores in the RMD participants. Thus, reflective pondering may not be a more “adaptive” form of rumination, at least in people who have experienced previous episodes of depression. Interestingly, only MDD participants differed from the CTL participants in their use of reappraisal, and no group differences were obtained for the use of expressive suppression.

Our results provide further support for the formulation that some emotion regulation strategies are more adaptive than others (Gross & John, 2002), and this may be particularly true in clinical samples. Specifically, looking at the full sample and at the group of MDD participants, increased levels of rumination, brooding, and expressive suppression were related to higher BDI scores, whereas increased use of reappraisal was associated with lower BDI scores; no

significant correlations between emotion regulation and BDI scores were obtained within the CTL group. Importantly, within the RMD group, greater use of rumination and expressive suppression, and less use of reappraisal, were highly correlated with BDI scores. These findings suggest that individual differences in the use of emotion regulation strategies may play an important role in recovery from depression and may increase risk for relapse. Consistent with these findings, Kuehner and Weber (2001) demonstrated that level of rumination at termination of treatment for depressed inpatients predicted relapse six months later. Similarly, Singer and Dobson (2007) found that remitted depressed patients who were instructed to ruminate during a negative mood induction had higher levels of depressed mood than did participants who were instructed to use distraction.

Finally, consistent with our predictions, individual differences in the inhibition of negative material were related to the use of specific emotion regulation strategies. Negative priming for negative material was associated with more reflection, more reappraisal, and less expressive suppression in the full sample. Importantly, these associations remained significant when controlling for BDI scores and overlap among emotion regulation measures. The association between negative priming and reappraisal is particularly noteworthy as it suggests that deficits in cognitive control make it less likely that people habitually re-interpret an emotion-eliciting situation in a way that allows them to modify emotional responding. Interestingly, Johnstone, van Reekum, Urry, Kalin, and Davidson (2007) recently reported that during reappraisal of emotional pictures, there was a correlation within nondepressed individuals (but not within depressed persons) between increased dorso-lateral prefrontal cortex (DLPFC) activation and decreased amygdala response mediated by the ventro-medial prefrontal cortex, suggesting depression-associated difficulties in recruiting brain regions involved in cognitive control to regulate emotions. Future research examining cognitive processes that support the use of this adaptive emotion regulation strategy could have important treatment implications. For example, Siegle, Thompson, Carter, Steinhauer, and Thase (2007) presented preliminary data demonstrating that a brief intervention targeted at increasing cognitive control in severely depressed outpatients led to significant decreases in both depressive symptoms and rumination. Indeed, recent work by this group suggests that training in attentional control may be an effective treatment component for depression (Siegle, Ghinassi, & Thase, 2007). Leyman, deRaedt, Vanderhasselt, and Baeken (in press) recently reported that a 10-day repetitive transcranial magnetic stimulation of the DLPFC led to significant mood improvements and to increased inhibitory control in the processing of negative information (assessed with a NAP task) in patients with MDD.

An unexpected correlation between negative priming for negative material and less use of expressive suppression was found in the control group. Expressive suppression, compared with reappraisal or rumination, does not depend as much on changes in cognition; it is surprising, therefore, that individual differences in cognitive control should be related to the use of suppression. In contrast to more cognitive strategies, expressive suppression is used late in the emotion generation sequence, likely after other strategies have failed and negative affect is experienced (Gross, 2002; Gross & John, 2003). Thus, people who exhibit reduced inhibition of negative material may be more likely to experience high levels of negative affect, which leads them to use expressive suppression more frequently, presumably because they could not use other strategies as effectively. Future studies are clearly needed to replicate this finding and to examine mechanisms underlying this association.

This study adds to a small but growing literature linking depression and rumination with deficits in executive control. Within the MDD group, reduced inhibition of negative material was related to increased levels of rumination and, specifically, brooding. These results replicate previous studies that have found that rumination is associated with reduced inhibition of negative material (Joormann, 2006; Joormann & Gotlib, 2008). Although investigators have

suggested that reduced inhibition plays an important role in rumination (Hertel, 1997, 2004; Linville, 1996), the current study is among the first to demonstrate such an association empirically. Because previous studies assessed executive functions and inhibition while participants were processing neutral stimuli, they do not address the important question of why rumination typically involves negatively valenced material. In addition, most of these studies have employed tasks that rely on a variety of executive processes, making it difficult to specify exactly what mechanisms are impaired in depression and rumination. For example, Davis and Nolen-Hoeksema (2000) used the Wisconsin Card Sorting Task and found that, compared to non-ruminators, ruminators made more perseverative errors, regardless of their level of depressive symptomatology. Watkins and Brown (2002) induced rumination in depressed participants and demonstrated that these individuals showed stereotyped counting responses in a random number generating task, reflecting their difficulty inhibiting prepotent responses. Recently, Whitmer and Banich (2007) employed a task-switching design and demonstrated that in a student sample, self-reported rumination was associated with difficulties inhibiting prior mental sets, but not with difficulties in switching to a new set. In contrast to these results, Goeleven et al. (2006) found that self-reported level of rumination was not related to inhibition of sad faces in a negative affective priming task. Goeleven et al. suggested that this finding might be due to the use of facial expressions, underscoring the potentially important association between rumination and semantic material, such as that found in the present study.

In closing, we should note several limitations of the current study. First, the current study relied on correlational data to investigate the association between individual differences in inhibition and the habitual use of emotion regulation strategies, which precludes us from making causal claims concerning these constructs. It is an important first step to demonstrate that specific cognitive processes are related to the use of emotion regulation strategies, but there are several third variables that may explain the observed associations. We included depression severity in our analyses and investigated the overlap among the different emotion regulation strategies as well as the correlations among our main constructs and variables such as current treatment, number of depressive episodes, and general level of functioning. We should point out here that this approach resulted in a large number of tests which may have increased type I error. We should also note that there are potentially important variables that we did not assess, such as treatment history and length of depressive episodes. Future studies are needed to examine more explicitly causal relations among the observed variables. In this context, it would be interesting to manipulate inhibitory control to examine whether this affects the use of emotion regulation strategies, or to conduct longitudinal studies that assess inhibitory functioning and emotion regulation prior to the onset of a depressive episode. Second, we utilized self reports to assess use of emotion regulation strategies; such reports may be subject to demand effects and provide little information about whether these strategies are used successfully. Thus, it is possible that cognitive processes such as inhibition are not only associated with the frequency of use of specific strategies but play a critical role in influencing whether the strategies (like reappraisal) are successful. As a related point, although emotion regulation is often strategically initiated, it can also occur without an individual's knowledge or intent (Mauss, Cook, & Gross, 2007). Automatic emotion regulation cannot be assessed via self-report; nevertheless, cognitive processes may play an important role in how frequently automatic emotion regulation occurs and how successful it is. Future research is needed to examine these aspect of emotion regulation and their relation to cognitive processes such as inhibition. Third, it is important to point out that some of our constructs (such as rumination) are complex, and different ways of defining and assessing them have been proposed. We used the RRS and the ERQ because they are the most frequently used measures of these constructs and because previous work has established relations among these measures and depression and inhibition. Future studies should compare different ways of assessing these and related constructs. Fourth, the negative priming task assesses only one aspect of inhibitory control in working memory: access of irrelevant emotional material. Other aspects of cognitive control, such as the ability to discard irrelevant

material from working memory or the ability to manipulate and prioritize information in working memory, may have detrimental effects on emotion regulation that warrant further study. Indeed, most contemporary theories postulate that executive control is not a unitary construct but, instead, involves several components such as the ability to focus attention, shift attention between stimuli, manage multiple tasks, and inhibit irrelevant processes or responses (e.g., Jonides & Nee, 2005; Nee & Jonides, 2008). It is important in future research that we systematically investigate these different processes and their relation to emotion regulation and depression (e.g., Joormann & Gotlib, 2008; Joormann, Nee, Berman, Jonides, & Gotlib, in press). Finally, we should point out that the concept of inhibition has been criticized in research on attention and memory (e.g., Friedman & Miyake, 2004; MacLeod et al., 2003; but see Anderson, 2003; Tipper, 2001). We believe, however, that our finding of a lack of negative priming in the MDD group, which is correlated with the use of specific emotion regulation strategies, represents an important finding even if the precise mechanisms underlying this association are not known.

This study represents a critical first step in examining the relations among inhibition, emotion regulation, and depression. Because the experience of negative mood states and negative life events is associated with the activation of mood-congruent cognitions in working memory, the ability to control access to working memory could be crucial in differentiating people who recover easily from negative affect from those who initiate a vicious cycle of maladaptive emotion regulation and deepening sad mood. Investigating individual differences in executive functions and, specifically, in the inhibitory control of the contents of working memory, has the potential to provide important insights into the maintenance of negative affect and vulnerability to experience depressive episodes.

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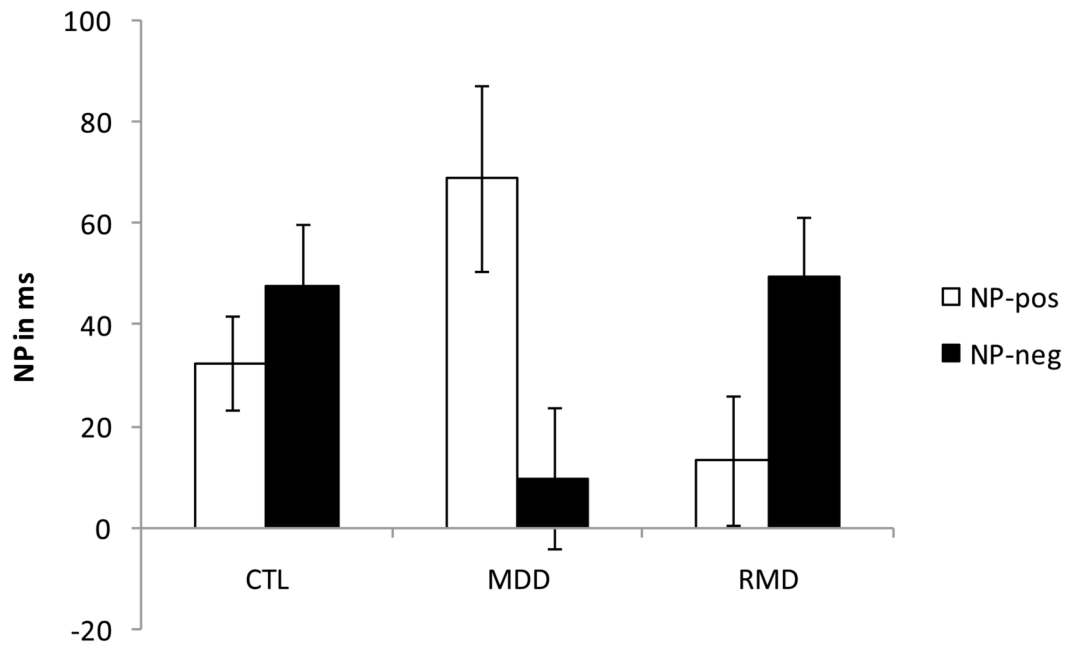


Figure 1. Negative priming (NP: Difference in ms negative priming - control condition) for positive (pos) and negative (neg) words in currently depressed (MDD), formerly depressed (RMD) and never-depressed control participants (CTL). Error bars represent one standard error.

Table 1

Sample characteristics and mean error rates (in %) and reaction times (in ms) in the negative priming task

	CTL	MDD	RMD
<i>N/N</i> female	32/15	22/16	47/30
Age (SD)	38.85 (12.02)	36 (9.27)	35.88 (9.68)
% Caucasian	67%	75%	86%
GAF	82.66 (6.31) _a	50.48 (6.26) _b	77.07 (11.07) _c
College degree	86%	85%	77%
Number of episodes	0 _a	5.79 (3.85) _b	3.62 (3.32) _c
Psychotherapy	0	55%	25%
Medication	0	48%	22%
BDI-II	3.06 (3.86) _a	30.81 (10.62) _b	4.71 (5.63) _b
RRS	31.38 (10.04) _a	52.83 (10.38) _b	44.09 (15.85) _c
RRS-Brooding	7.17 (2.24) _a	13.04 (3.46) _b	8.48 (3.04) _c
RRS-Reflection	8.37 (3.09) _a	10.42 (2.94) _b	11.13 (4.30) _b
ERQ-Reappraisal	4.94 (1.00) _a	3.81 (0.96) _b	5.05 (1.21) _a
ERQ-Suppression	3.20 (1.14)	3.61 (1.10)	3.63 (1.26)
NP-pos	972 (194)	986 (200)	965 (209)
	96.6% (0.04)	97.3% (0.04)	96.5% (0.04)
NP-neg	1013 (204)	956 (210)	977 (208)
	96.8% (0.04)	96.7% (0.04)	95.7% (0.04)
C-pos	965 (212)	917 (160)	952 (206)
	97.5% (0.03)	97.5% (0.03)	97.4% (0.03)
C-neg	965 (212)	947 (199)	927 (189)
	97.5% (0.04)	97.1% (0.04)	97.3% (0.03)

Note. CTL = Participants in the control group, RMD = Remitted depressed participants, MDD = Currently depressed participants; GAF = Global Assessment of Functioning Rating Scale, BDI-II = Beck Depression Inventory-II, RRS = Ruminative Response Scale, ERQ = Emotion Regulation Questionnaire; NP-pos = Negative Priming for positive words, NP-neg = Negative Priming for negative words; C-pos = Control condition for positive words, C-neg = Control condition for negative words; Means with different subscripts within rows differ significant at at least $p < .05$

Table 2

Correlations of negative priming and emotion regulation scales in full sample and in CTLs

	Full Sample (N = 97)			CTL (N = 31)		
	NP-pos	NP-neg	BDI	NP-pos	NP-neg	BDI
RRS	.09	.05	.54*	.06	.10	.09
RRS-Brooding	.09	-.02	.65*	.14	.14	.28
RRS-Reflection	.07	.20*	.15	.01	.28	-.01
ERQ-Reappraisal	.10	.25*	-.51*	.26	.15	.09
ERQ-Suppression	.01	-.24*	.21*	.21	-.46*	-.05

Note. CTL = Participants in the control group; BDI = Beck Depression Inventory. RRS = Ruminative Response Scale, ERQ = Emotion Regulation Questionnaire; NP-pos = Negative Priming for positive words, NP-neg = Negative Priming for negative words.

* $p < .05$.

Table 3
 Correlations of negative priming and emotion regulation scales in MDD and RMD

	MDD (N = 21)			RMD (N = 45)		
	NP-pos	NP-neg	BDI	NP-pos	NP-neg	BDI
RRS	.28	-.38*	.65*	-.04	.13	.43*
RRS-Brooding	.05	-.41*	.43*	-.17	-.01	.22
RRS-Reflection	.47*	.29	.27	.02	.17	.29*
ERQ-Reappraisal	.30	.13	-.43*	.23	.22	-.45*
ERQ-Suppression	-.01	-.15	.28	-.06	-.15	.60*

Note. RMD = Remitted depressed participants, MDD = currently depressed participants; BDI = Beck Depression Inventory, RRS = Ruminative Response Scale, ERQ = Emotion Regulation Questionnaire; NP-pos = Negative Priming for positive words, NP-neg = Negative Priming for negative words; C-pos = Control condition for positive words, C-neg = Control condition for negative words.

* $p < .05$.