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Cognitive inhibition in depression☆

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Abstract

Cognitive inhibition is a key mechanism in the regulation of emotion. There is emerging evidence that depression is characterized by deficits in the inhibition of mood-congruent material. These deficits could result in prolonged processing of negative, goal-irrelevant aspects of presented information thereby hindering recovery from negative mood and leading to the sustained negative affect that characterizes depressive episodes. Indeed, it has been suggested that deficits in cognitive inhibition lie at the heart of memory and attention biases in depression, and set the stage for ruminative responses to negative events and negative mood states. A ruminative response style results in a heightened vulnerability to experience episodes of major depression. Recent research has demonstrated that deficient inhibition of negative material is associated with heightened rumination. In this article, we review the depression literature with a focus on studies that investigate cognitive inhibition in depressed participants and in participants who report a history of major depressive episodes. In addition, we summarize neurobiological findings that indicate a strong relation between depression and deficits in inhibition and we take a closer look at the relation of inhibition, rumination and mood regulation.

Keywords: Depression; Inhibition; Attention; Memory; Bias; Cognition

Studies investigating the interaction of cognition and emotion have a long tradition in depression research. While previous investigators have primarily focused on examining the content of depressive cognition, recent studies have begun to investigate cognitive processes that may underlie and enhance the negative thoughts that characterize depressive disorders (see Mathews & MacLeod, 2005, for a recent review). Most of these studies, however, have narrowly focused on demonstrating the existence of cognitive deficits and biased processing in depression using a variety of self-report measures and experimental tasks. Very few studies have examined how deficits in free recall, attentional biases towards negative material, and mood-congruent memory are related to each other and, more importantly, are related to the hallmark feature of depression which is sustained negative affect.

Negative mood is generally associated with, or consists in part of, 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; Mathews & MacLeod, 2005; Rusting, 1998). This research has also demonstrated, however, that experiencing negative mood does not necessarily lead to prolonged negative affect. Indeed, 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 initiate a self-defeating cycle of increasingly negative thinking and intensifying negative affect. If changes in mood are, in fact, associated with activations of mood-congruent material in working memory, the ability to control the contents of working memory might play an important role in recovery from negative mood. Indeed, researchers have argued that such deficits in cognitive control are related to individual differences in the tendency to ruminate (Davis & Nolen-Hoeksema, 2000). Rumination consists of persistent and recurring thoughts that unintentionally enter consciousness (Nolen-Hoesema, 1987), and has been defined as "behaviors and thoughts that focus one's attention on one's depressive symptoms and on the implications of those symptoms" (Nolen-Hoeksema, 1991, p. 569). It has been conceptualized as a trait-like response style that perpetuates depressive symptoms (Nolen-Hoeksema, 1991). Given that sustained negative affect and anhedonia are the defining symptoms of a major depressive episode, it is important that we

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identify the processes that hinder mood regulation and lead into rumination.

We propose that a closer investigation of individual differences in cognitive inhibition helps us to better understand rumination and dysfunctions in mood regulation in depression. Specifically, we propose that depressed persons experience difficulty disengaging from, and inhibiting elaborative processing of, negative stimuli or information. We posit further that this inhibitory deficit is related to the high level of rumination that has been found to characterize depressed individuals when they experience negative mood states and events. Depressed individuals cannot prevent negative material from entering and remaining in working memory, leading them to rehearse, or to ruminate about, negative content, which leads to better long-term memory for negative material and serves to exacerbate their negative affect.

1. Cognitive inhibition

Inhibition, working memory, and cognitive control are important concepts in understanding dysfunctional cognitive processes that underlie sustained processing of negative information and rumination in depression. Working memory is commonly described as a system for the active maintenance and manipulation of information and for the control of attention (Baddeley, 1986). An important characteristic of working memory, and one that differentiates it from long-term memory, is its capacity-limited focus of attention (see Cowan, 1995). Given this capacity-limitation, inhibitory control is critical for updating working memory content efficiently and is therefore essential for engaging in goal-directed planning and for maintaining a coherent stream of thought. Thus, Hasher and Zacks (Hasher, Zacks, & May, 1999; Hasher and Zacks, 1988) proposed that the efficient functioning of working memory depends on inhibitory processes that both limit the access of information into working memory and update the contents of working memory by removing information that is no longer relevant.

As outlined by Dillon and Pizzagalli (this issue) in the introductory article, most contemporary theories postulate that inhibition is not a unitary construct but, instead, involves several components such as response inhibition, cognitive inhibition, and emotional inhibition (e.g., Friedman & Miyake, 2004; Harnishfeger, 1995; Nigg, 2000). In addition, cognitive inhibition operates at different stages of the processing of information (Hasher & Zacks, 1988; Hasher et al., 1999) for example, by preventing off-goal information from having access to working memory or by reducing the activation of information that was once relevant, but now is irrelevant because of a change in goals.

What happens when these processes malfunction? As Hasher and Zacks (1988) have pointed out, too much irrelevant information gets into working memory. As a consequence, links between relevant and irrelevant information are created and stored in long-term memory, setting the stage for slow and less accurate retrieval of relevant information and enhanced retrieval of irrelevant information. In addition, irrelevant information in working memory is sustained longer. Thus, individuals who exhibit an inhibitory deficit are easily distracted by irrelevant information and thoughts which may result in cognitive deficits and disrupt a coherent stream of thought. Indeed, a reduced ability to inhibit irrelevant representations has been proposed as a source of low working memory capacity (Engle, Kane, & Tuholski, 1999). As outlined by Dillon and Pizzagalli (this issue), weakened inhibitory processes have been proposed and found in a range of populations, including older adults (Hasher, Stoltzfus, Zacks, & Rypma, 1991), children with Attention Deficit Disorder (Bjorklund & Harnishfeger, 1990), patients with Obsessive-Compulsive Disorder (Enright & Beech, 1990), and patients with schizophrenia (Frith, 1979).

As the central function of working memory, malfunctioning inhibitory processes might have severe cognitive and emotional consequences, and rumination may be one of them. In the following sections we will summarize evidence for deficits in cognitive inhibition that are associated with depression, starting with research on cognitive deficits and cognitive biases. We will finish with a discussion of evidence for the link between cognitive inhibition and dysfunctional mood regulation and with a short discussion of possible neurobiological correlates of inhibitory deficits in depression.

2. Cognitive deficits and biases in depression

While depressed people report concentration difficulties and impairments in the recall of neutral stimuli (Burt, Zembar, & Niederehe, 1995), they easily concentrate on negative self-focused thoughts and exhibit enhanced recall of mood-congruent memories (Gotlib, Roberts, & Gilboa, 1996; Rusting, 1998). Thus, while some researchers focus on examining "cognitive symptoms" of depression, such as concentration difficulties, distractibility, attention deficits, and impaired recall of information independent of valence, other researchers have focused on examining biased processing of emotional information. Very few attempts have been made to try to integrate the findings obtained in these separate lines of research (Ellis & Ashbrook, 1988; Hertel, 1997; Williams, Watts, MacLeod, & Mathews, 1997).

2.1. Cognitive deficits

What is the evidence for a generalized cognitive deficit in depression? Depressed people often complain about concentration difficulties (Watts & Sharrock, 1985) and "difficulty concentrating" has been included as a symptom of a major depressive episode in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; APA, 1994). There is a large amount of literature that strongly suggests depression-related impairment in the recall of non-emotional information (for reviews see Burt et al., 1995; Johnson & Magaro, 1987; Mathews & MacLeod, 1994). In the frequently cited meta-analysis by Burt et al. (1995), however, memory impairments were seen most consistently for inpatients relative to outpatients. Moreover, these kinds of memory impairments were also reported

in other psychological disorders (e.g. schizophrenia, comorbid substance abuse, and anxiety disorders). Burt et al. (1995), therefore, proposed that memory deficits are associated with psychopathology in general rather than with disorder-specific factors. Moreover, in a series of studies, Hertel and her collaborators (Hertel, 1998; Hertel & Rude, 1991; Hertel & Milan, 1994) have gathered evidence that depression-related impairments are not observed in all components of memory but are primarily observed in free recall tasks and in controlled aspects of recognition. Recognition tests combine controlled and automatic retrieval processes in that either the controlled recollection of prior exposure to the test items, or the automatic experience of familiarity, are used for judgments about test items. Hertel found the automatic components of recognition to be intact in depressed individuals (Hertel, 1998; Hertel & Milan, 1994; Hertel & Hardin, 1990).

Overall, studies conducted so far provide evidence that depression is associated with greater memory impairment in contexts in which (1) attention is not constrained by the task (Hertel & Hardin, 1990; Hertel & Rude, 1991), (2) increased cognitive effort is required (Hasher & Zacks, 1979, see review by Hartlage, Alloy, Vazquez, & Dykman, 1993), and (3) attention is easily allocated to personal concerns and other thoughts irrelevant to the task (Ellis, Thomas, & Rodriguez, 1984; Seibert & Ellis, 1991). In a study that required participants to judge whether a target word fit into a corresponding sentence frame and then tested recall of the target words, Hertel and Rude (1991) were able to eliminate a depressive deficit by providing instructions that focused participants on the task and did not allow task irrelevant thoughts. In their "focused" condition, participants were required to keep each word in mind for the duration of the trial. In their "unfocused" condition, however, participants could respond right away and could choose to sustain attention on the task or to think about other concerns. Following-up on these studies, Hertel (1998) also reported that dysphoric students who had to wait in an unconstrained situation (without being given any instructions regarding what to do during the waiting period) and dysphoric students who were instructed to rate self-focused material designed to induce rumination, showed comparable recall deficits. No deficit was found for dysphoric students who were told what to do during the waiting period (rating self-irrelevant and task-irrelevant material).

These results suggest that, at least with respect to memory deficits, depressed people might have the ability to perform at the level of non-depressed people in structured situations but have problems doing this on their own initiative in unconstrained situations (Hertel, 2004). Moreover, these results suggest that the elimination of the opportunity to ruminate also eliminated the impairment in the memory task, a result that might explain why unconstrained tasks lead to impaired performance in the depressed group. Unconstrained situations call for cognitive flexibility and goal-oriented behavior and require cognitive control, that is, focal attention to relevant stimuli as well as inhibition of irrelevant material (Hertel, 2000, 2004). Thus, these performance deficits in the recall of neutral information do not seem to reflect a generalized deficit or a lack of resources on the part of depressed individuals but might instead be due to depression-related inhibitory dysfunctions in the processing of irrelevant information.

Studies that have investigated depression-related deficits in attention tasks and in tasks that assess executive functions have also reported inconclusive findings. Breslow, Kocsis, and Belkin (1980) found differences between depressed and nondepressed participants on the digit span while other studies could not replicate this effect (Colby & Gotlib, 1988; Gray, Dean, Rattan, & Cramer, 1987; Harvey et al., 2004). Channon, Baker, and Robertson (1993) compared depressed participants with controls on a variety of working memory tasks and found very few differences (i.e., only on the backward digit span; see also Barch, Sheline, Csernansky, & Snyder, 2003; Fossati, Amar, Raoux, Ergis, & Allilaire, 1999; Purcell, Maruff, Kyrios, & Pantelis, 1997). Recently, Rose and Ebmeier (2006) reported that depressed patients were slower and less accurate on an n-back task but that task difficulty did not influence this effect. These findings replicate results reported by Harvey et al. (2004) who also reported that performance deficit on the n-back task was correlated with number of hospitalizations and longitudinal course of the disorder. Harvey et al. (2004) did not find differences between their control and depressed group on a number of other tasks assessing working memory functioning, including digit span. In line with these finding, Egeland et al. (2003) concluded from their study that reduced performance on working memory tasks in depression is due to a non-specific speed reduction and to a loss of vigilance that is consistent with a lack of effort but not with a specific deficit in executive functioning.

Most of the tasks that involve working memory, e.g., the digit span, involve relatively short retention intervals and thus seem to allow a more direct assessment of attentional processes irrespective of retrieval from long-term memory. The tasks have been criticized, however, because the relatively slow paced presentations that are used to ensure perception might allow for chunking and active rehearsal of material and might thus reflect memory deficits instead of deficits in attention (Rokke, Arnell, Koch, & Andrews, 2002). In a more recent study using an attentional blink paradigm that involves rapid serial presentations, significant group differences in performance between moderately to severely dysphoric (BDI over 21) and nondysphoric participants were found but only under demanding dual-tasking conditions (Rokke et al., 2002). Moreover, only nine moderately to severely dysphoric participants were included in this study.

Grant, Thase, and Sweeney (2001) recently administered a battery of cognitive tasks to 123 depressed outpatients and noted the surprising absence of cognitive deficits in their sample. The only indications of deficits were less completed categories, increased perseveration, and impaired maintenance of set on the Wisconsin Card Sorting task (WCST), a widely used measure of executive control and cognitive flexibility. These results suggest depression-related deficits in the generation and maintenance of problem solving strategies and difficulties in set switching (see also Harvey et al., 2004; Merriam, Thase, Haas, Keshavan, & Sweeney, 1999). There was no evidence, however, for deficits in executive functioning on any of the other tasks the authors employed. In line with these results, Davis and Nolen-Hoeksema

(2000) administered the WCST to participants who scored high on a self-report measure of ruminative style and reported that ruminators made more errors than non-ruminators.

Thus, so far, limited evidence is available to suggest that depressed participants are characterized by deficits in attention tasks and by deficits in tasks that assess executive control when processing neutral information. Grant et al. (2001) conclude from their study that cognitive deficits more likely characterize elderly depressed people and severely depressed inpatients that present with psychotic features (see also Harvey et al., 2004; Rose & Ebmeier, 2006, for similar conclusions). Indeed, most studies that have found differences examined depressed inpatients but even in these studies depressed patients did not exhibit cognitive deficits across all tasks. Several investigators have also pointed out that the tasks employed in these studies, e.g. the WCST, assess many different processes, making it difficult to separate primary functional deficits (e.g. Harvey et al., 2004; see also Dillon & Pizzagalli, this issue). More studies are clearly needed to clarify these results.

To summarize, surprisingly little empirical support has been found so far for depressive deficits in the processing of neutral information. Most promising are results obtained in studies that investigate recall and recognition. While the literature strongly suggests a depression-related impairment in the recall of neutral information, it seems safe to say that the bulk of evidence suggests deficits in the control of attention rather than limited processing capacities. When their attention is well controlled by the demands of the task, no depressive deficits are found. Focusing attention requires the inhibition of task-irrelevant thoughts. As discussed earlier, Hertel (1998) has shown that an experimental context that eliminates the opportunity to ruminate eliminates the impairments in a memory task. Hertel (2004) has further proposed that rumination and negative self-focused thoughts are prepotent responses for depressed individuals. Overriding prepotent responses and focusing attention on the demands of the current task is the role of inhibitory processes in attention and memory. Inhibitory dysfunction in depression might thus be responsible for this lack of self-controlled attention to the task at hand. Deficits in the processing of neutral information may therefore be a consequence of biases in the processing of emotional material.

2.2. Cognitive biases

Depression is characterized by negative, automatic thoughts about the self, the future, and the world. Indeed, cognitive theories of depression propose that automatic thoughts and preferential processing of negative material play an important role in the onset, maintenance, and recurrence of depressive episodes. In addition, one of the most successful interventions for depression, cognitive-behavioral therapy, focuses on dysfunctional automatic thoughts (Beck, 1976) while other models highlight cognitive biases as possible vulnerability markers (Gotlib & Krasnoperova, 1998; Gotlib & Neubauer, 2000; Ingram, Miranda, & Segal, 1998).

Cognitive models of depression predict cognitive biases in all aspects of information processing, including attention, memory, and interpretation (Beck, 1976). Overall, there is strong evidence for biased memory processes, at least in explicit memory tasks (Mathews & MacLeod, 2005; Williams et al., 1997). Biased memory for negative, relative to positive, information in explicit memory tasks represents perhaps the most robust cognitive finding associated with major depression (Blaney, 1986; Matt, Vazquez, & Campbell, 1992). In a meta-analysis of studies assessing recall performance of persons with major depression, Matt and colleagues found that this group remembers 10% more negative words than positive words. Non-depressed controls demonstrated a memory bias for positive information in 20 out of 25 studies.

In contrast, a number of studies have failed to find attentional biases in depression (e.g., MacLeod, Tata, & Mathews, 1987; Mogg, Bradley, Williams, & Mathews, 1993). Furthermore, no attentional biases were found in participants who had previously been depressed as compared to participants who had never been depressed (e.g., Gilboa & Gotlib, 1997; Hedlund & Rude, 1995). Consequently, Williams, Watts, MacLeod, and Mathews (1988) and Williams et al. (1997) proposed an alternative model, stating that depressed persons are not characterized by biases in attentional functioning, but rather, by biases in post-attentional elaboration. These authors suggested that anxiety-congruent biases are observed in tasks, which assess the early, orienting stage of processing, prior to awareness, e.g. selective attention and priming tasks. In contrast, depressive biases are observed in strategic elaboration, and therefore would be found in recall tasks, but not in selective attention tasks.

Although this formulation seems plausible, it may be premature to conclude that depressed persons are not characterized by an attentional bias. Recent studies using the dot probe task, for example, reported selective attention in depression. These biases were only found under conditions of long stimuli exposures (Bradley, Mogg, & Lee, 1997; Gotlib, Krasnoperova, Yue, & Joormann, 2004; Joormann & Gotlib, 2007a, 2007b; Joormann, Talbot, & Gotlib, 2007; Mogg, Bradley, & Williams, 1995). In the dot probe task, a pair of stimuli (words or faces) is presented simultaneously, one stimulus is neutral and the other is emotional. Participants are asked to respond to a probe that replaces the neutral or the emotional stimulus. Allocation of attention to the spatial position of the stimuli is determined from response latencies to the probes. Mogg et al. (1995) reported a mood-congruent bias in depressed participants, but only under supraliminal conditions. Likewise, Bradley et al. (1997) reported a mood-congruent bias in the dot probe task for both induced and naturally occurring dysphoria when stimuli were presented for 500 or 1000 ms, but not for briefly presented stimuli (14 ms). Using a dot probe task with emotional faces instead of words, Gotlib et al. (2004) found an attentional bias for negative faces that were presented for 1000 ms in clinically diagnosed depressed participants. In two recent studies, Joormann et al. (2007) and Joormann and Gotlib (2007a) replicated these findings in a sample of remitted depressed participants and in a sample of non-disordered girls who are at high risk for depression onset due to their mother's psychopathology. According to Bradley et al. (1997), these results suggest that a selective bias for negative information in depression exists, but does not

operate throughout all aspects of selective attention: depressed individuals may not automatically orient their attention towards negative information in the environment, but once such information has come into the focus of their attention, they may have greater difficulty in disengaging from it.

Similar difficulties in disengaging attention from negative material have been demonstrated in an exogenous cueing task (Koster, DeRaedt, Goeleven, Franck, & Crombez, 2005; Koster, Leyman, DeRaedt, & Crombey, 2006). These studies were conducted to examine the nature of attentional bias in dysphoria which can be done by using the spatial cueing task. In this task, participants are instructed to detect a visual target that is presented either at the left or right side of a fixation cross. In half of the trials, the visual target is preceded by a cue word on the same side (valid trials) and in the remaining trials the cue word is presented at the opposite side (invalid trials). To assess the effect of valence on attentional disengagement, positive, negative, or neutral cue words are presented. Response times (RTs) to targets are usually faster in valid compared to invalid trials (i.e., cue validity effect). This effect, however, disappears or is even reversed at longer intervals between cue onset and target onset (SOA > 300 ms) because the previously attended location is inhibited in favor of a new location. Thus, a prolonged cue validity effect indicates enhanced attentional engagement and difficulty in inhibiting the previously presented cue. Attentional engagement can further be calculated by subtracting RTs to valid emotional cues from RTs to valid neutral cues. Difficulty in attentional disengagement is assessed by subtracting RTs to invalid neutral trials from RTs to invalid emotional trials. Dysphoric participants exhibited an enhanced cue validity effect for and impaired attentional disengagement from negative words for the long SOA (i.e., 1500 ms), but no effects were observed at 250 ms SOA (Koster et al., 2005). Furthermore, the control group demonstrated an enhanced cue validity effect for positive words at 1500 ms presentation time. These results provide further evidence that dysphoria is associated with greater difficulty in disengaging attention from negative information. A more recent study using pictures of neutral, happy, sad, and angry faces, however, failed to find any group differences in the cue validity effect (Koster et al., 2006).

Response latencies as a measure of attentional bias have been criticized because they do not allow the distinction between attentional and non-attentional factors such as deficits in motor response and response selection. In a recent study, Eizenman et al. (2003), therefore, employed eye tracking technology to continuously monitor point of gaze. Participants were presented with slides with four different pictures. Each slide consisted of either four neutral pictures or four pictures displaying an object associated with one of four different themes: loss and sadness, threat and anxiety, interpersonal attachment and social contact. Depressed individuals compared to controls spent significantly more time looking at pictures featuring sadness and loss (longer total fixation time) and had significantly longer average glance durations for these pictures. Fixation frequency, however, did not differ between the depressed and the control group. This suggests that depressed individuals do not direct their attention to negative information more frequently than control participants, but once it captures their attention they exhibit difficulties disengaging from it.

These results are consistent with recent research into selective attention which suggests that selective attention is not a unitary concept and that different components (e.g., orienting vs. maintenance/disengagement) and underlying mechanisms of selective attention have to be separated (LaBerge, 1995; Posner, 1995). Selective attention involves at least two mechanisms: (a) activation of selected, relevant information and (b) active inhibition of unselected, irrelevant information (Hasher & Zacks, 1988; Milliken & Tipper, 1998; Neill, Valdes, & Terry, 1995; Tipper, 1985). Our review of the literature suggests that depression is not associated with differential initial activation levels of negative, compared to neutral, stimulus representations. Instead, malfunctioning inhibitory mechanisms in the processing of negative stimuli might explain the observed difficulties in disengaging attention from negative material and, consequently, the increased elaboration of negative material that is associated with this disorder.

3. Evidence from inhibition tasks

Although it is very straightforward to draw a theoretical distinction between pure activation and activation/inhibition positions, it is nevertheless very difficult to generate unequivocal evidence for the role of inhibition in a given task (Anderson & Bjork, 1994; Anderson & Spellman, 1995). In the previous sections we mainly summarized indirect evidence for inhibitory deficits using findings from general research on memory and attention in depression. While we believe that the pattern of results clearly suggests that inhibitory deficits are related to impairments in memory and to cognitive biases in depression, all of the tasks that we discussed assess a multitude of processes and therefore do not directly speak to the question of whether depression is characterized by inhibitory deficits. Over the last 15–20 years, however, a number of experimental methodologies have emerged that have the potential to test inhibition models in that they provide data that cannot be easily explained without a concept like cognitive inhibition. Some of these designs, such as negative priming (Tipper, 1985) and directed forgetting (Bjork, 1972) are discussed below.

3.1. Selective attention

Cognitive inhibition of irrelevant information is crucial in a range of tasks that require selective attention. Negative priming is an experimental task that aims to distinguish activation from inhibition accounts of selective attention (Neill et al., 1995; Tipper, 1985). In this task, participants are asked to respond to a target in the presence of a distractor. Participants can be asked, for example, to name a word written in red while ignoring a word written in blue that is presented at the same time. The *negative priming effect* is defined as a delayed target response latency when the distractor from a previous trial becomes the target on the present trial. Thus, negative priming occurs when, in the following trial, the presented target is identical or related to the previously presented to-be-ignored distractor. The inhibi-

tion of the distractor that is activated on the first trial remains activated on the following trial, delaying the response to a target that is identical to or related to the ignored distractor. The delay in responding, therefore, assesses the strength of inhibition of the distractor that was presented on the previous trial.

Negative priming has been observed in a variety of selective attention tasks, including semantically related distractor-target pairs (e.g., a picture of a dog following an ignored picture of a cat; Tipper, 1985). One of the most prolific areas of research in negative priming has been the identification of individual differences that are correlated with the effect. Deficits in negative priming have been observed in various samples, such as older adults (Hasher et al., 1991), young children (Tipper, Bourque, Anderson, & Brehaut, 1989) and in patients with schizophrenia (Frith, 1979). Such individual differences in negative priming have been linked to a reduced ability of these subgroups to inhibit the intrusion of irrelevant information into working memory (Neill et al., 1995). Linville (1996) was the first to investigate negative priming in depression. She reported that depressed individuals were less likely to inhibit distracting information. Specifically, participants were asked to complete a modified lexical decision task that required them to inhibit the presence of a distractor (i.e., letter string) while identifying whether a second string is a word. Control participants were slower in responding to letter strings that they had been asked to ignore on an earlier trial. Depressed individuals, however, failed to show this effect. Similarly, MacQueen, Tipper, Young, Joffe, and Levitt (2000) used a negative priming task in which color and location of prime and target were varied systematically and reported reduced inhibition of distractors in depressed participants. While these results support the proposition that depression is related to inhibitory dysfunction, inhibition deficits might be even more prevalent in the processing of emotional information. In particular, the observation of negative automatic thoughts and ruminations on negative information in depression leads to the hypothesis that there is a valence-specific inhibitory deficit in depression. Thus, inhibition should be selectively reduced for negative stimuli.

Negative affective priming is a task that was designed to assess inhibition in the processing of emotional information (NAP; Gotlib, Yue, & Joormann, 2005; Joormann, 2004; see also Goeleven, DeRaedt, Baert, & Koster, 2006). In accordance with standard negative priming designs, the NAP-design consists of consecutive pairs of trials, a prime-trial and a test-trial (see Fig. 1). In each trial, two adjectives are presented, i.e., a target and a distractor, along with an instruction to ignore the distractor and to respond to the target. 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, prime trial distractor and test trial targets are unrelated. Negative priming is analyzed using a within-subject comparison of negative priming and corresponding control conditions. Several authors have pointed out that it is important to introduce a self-focus or self-reference in order to find pronounced depressive biases

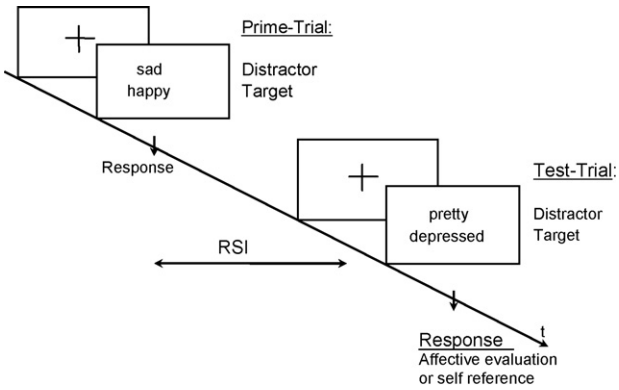

Fig. 1. Design: negative affective priming. Two consecutive trials, a prime and a test trial, are presented. In each trial, the subject is shown a distracter and a target word and is instructed to respond to the target and to ignore the distracter. The time interval between the participant's response and the presentation of the next pair of words is the so-called Response-Stimulus Interval (RSI). Adapted from Joormann (2004, p. 128). Copyright © 2004 by Psychology Press Ltd., http://www.psypress.co.uk/journals.asp. Adapted with permission.

in cognitive tasks (e.g., Segal, 1988; Segal & Vella, 1990). In the NAP task, participants are asked to evaluate the valence of the target word (positive vs. negative) or to evaluate whether the presented target word is self-descriptive.

In a series of studies using this task, Joormann (2004) demonstrated that dysphoric participants and participants with a history of depressive episodes exhibit reduced inhibition of negative material that they were instructed to ignore. Thus, these participants responded faster when a negative target was presented after a to-be-ignored negative distractor on the previous trial. As predicted, no group difference was found for the positive adjectives. In a related study, participants who scored high on a self-report measure of rumination exhibited a reduced ability to inhibit the processing of emotional distractors, a finding that remained significant even after controlling for level of depressive symptoms (Joormann, 2006). These findings were replicated using a negative priming task with emotional faces (Goeleven et al., 2006). Compared to nondepressed controls, depressed participants showed impaired inhibition of sad facial expressions, but intact inhibition of happy expressions. Never depressed individuals exhibited a stronger NAP effect for both sad and happy faces compared to neutral faces, indicating successful inhibition of emotional information in general. The performance of formerly depressed participants, however, was not different from controls. NAP effects for sad or happy faces were also not related to a self-report measure of rumination. Furthermore, NAP effects for sad faces predicted depression scores even after controlling for rumination scores. According to the authors, the lack of correlation between inhibitory deficits and rumination in their study might be explained by the fact that rumination is a rather verbal process whereas the stimuli in this study were images of facial expressions. These results support the hypothesis that elevated depression scores are related to inhibitory dysfunctions in the processing of negative stimuli. Further studies are needed, though, to examine negative priming in remitted depressed participants as well as the relation of negative priming to individual differences in rumination.

It is important to note, however, that negative priming tasks assess only one aspect of inhibition, that is, the ability to control the access of relevant and irrelevant material to working memory. While these studies suggest that depression, and probably also rumination, involve difficulties keeping irrelevant emotional information from *entering* working memory, no studies have examined whether depression and rumination are also associated with difficulties *removing* previously relevant negative material from working memory. Difficulties inhibiting the processing of negative material that was, but is no longer, relevant might explain why people respond to negative mood states and negative life events with recurring, uncontrollable, and unintentional negative thoughts.

To test this hypothesis, Joormann and Gotlib (2007b) adapted a modified Sternberg task developed by Oberauer (2001, 2005a, 2005b) that combines a short-term recognition task with instructions to ignore a previously memorized list of words to assess inhibition of irrelevant positive and negative stimuli. In this task, two lists of emotional words are presented simultaneously. After the lists are memorized, a cue indicates which of the two lists is relevant for the recognition task on the next display, in which participants indicate whether the probe that is presented came from the relevant list; probes from the no-longer-relevant list must be rejected, as must new probes. The difference in reaction times to an intrusion probe (i.e., a probe from the irrelevant list) and reaction times to a new probe (i.e., a completely new word) reflects the strength of the residual activation of the contents of working memory that were declared to be no longer relevant and, therefore, assesses a person's ability to remove irrelevant information from working memory. The results of this study indicate that participants diagnosed with major depression exhibit difficulties removing irrelevant negative material from working memory. Compared to never-depressed controls, depressed individuals exhibited longer decision latencies to an intrusion probe (i.e., a probe from the irrelevant list) than to a new probe (i.e., a completely new word), reflecting the strength of the residual activation of the contents of working memory that were declared to be no longer relevant (see Oberauer, 2001, 2005a, 2005b). This pattern was not found for positive material. To examine whether these difficulties were due simply to elevated levels of sad mood, we compared the performance of depressed participants to that of never-depressed participants who completed the task after receiving a sad mood induction. The depressed participants exhibited greater difficulty expelling irrelevant negative material from working memory than did the control participants who were in a sad mood, indicating that a negative mood state alone is not sufficient to explain this effect. We also found that difficulty removing negative irrelevant words from working memory was highly correlated with self-reported rumination, even after controlling for level of depressive symptoms: the higher the participants' scores on a self-report measure of rumination, the more difficulty they exhibited removing task-irrelevant negative material from working memory. In sum, therefore, these findings indicate that depression and rumination are associated with inhibitory impairments in the processing of emotional material, specifically, with difficulties removing irrelevant negative material from working memory.

3.2. Intentional forgetting

As outlined above, memory biases are consistently found in depression research. It has been suggested that analogous processes might underlie selective retrieval of target items from memory and selective attention to objects in the external environment (Anderson & Spellman, 1995). Consequently, if inhibitory dysfunction in depression is found in selective attention tasks, it may also be detectable in memory tasks. There is a consistent thread of studies within memory research that has addressed the idea of inhibitory dysfunctions. This work is concerned with the phenomenon of directed forgetting. The general idea of directed forgetting studies is to instruct participants at some point during the presentation of to-be-remembered (TBR) items that the items already presented and encoded are now to be forgotten and will not be tested later, that is, subjects are directed to forget certain stimuli (Bjork, 1972; Epstein, 1971). At some point free-recall and recognition of to-be-remembered and to-be forgotten (TBF) items are tested. In the majority of these studies the recall of TBF items is usually very low while their recognition level is usually very high. To our knowledge, no study so far has examined directed forgetting effects for neutral material in depressed subjects. In a directed forgetting task with positive and negative words, Power, Dalgleish, Claudio, Tata, and Kentish (2000), reported differential directed forgetting effects. When control participants and dysphoric participants were given the instruction to make valence judgments about the presented words and to remember them at the same time, both groups showed comparable directed forgetting effects for positive and negative words. When the participants were asked, however, to rate the presented words in terms of self-descriptiveness, the control participants recalled more positive than negative words under the "forget" instruction. This positive bias was not found for dysphoric participants. Moreover, in a third study using self-reference judgments and clinically diagnosed depressed participants, the depressed group exhibited a facilitation effect for negative words after the "forget" instruction while the control subjects, again, showed a positive bias.

Joormann and Tran (submitted for publication) examined the effects of self-reported levels of rumination on directed forgetting. All participants were asked to make valence judgments about the presented words and to try to remember the words at the same time. Participants were subsequently instructed to forget half of the words and remember the other half. We did not include a naturalistic condition but we found very clear group differences in the forget condition while no group differences emerged for the remember condition. Specifically, compared to participants who scored low on the Ruminative Responses Scale (RRS), participants who scored high exhibited reduced forgetting of positive and negative words in the forget condition. These participants also exhibited increased recall of negative words that were never presented during the learning phase. These results remained stable when depression scores were included as a covariate.

In a recent study, Hertel and Gerstle (2003) found additional evidence for reduced inhibition of negative words in dysphoric students. These authors employed a design that was

originally proposed by Anderson and Green (2001). Dysphoric and nondysphoric students learned word pairs, each consisting of a positive or negative adjective and a neutral noun. In subsequent practice trials participants practiced to either recall the target word or to suppress (i.e. make an active effort not to think about) the target word when given the adjective as a cue. On the final test, recall for all words was tested. Hertel and Gerstle (2003) found that recall from sets assigned for suppression practice was greater in the dysphoric group, with a tendency towards increased recall of to-be-suppressed negative words. Moreover, the degree of forgetting was significantly correlated with self-report measures of rumination and unwanted thoughts. Again, these results suggest a close relation between self-reported rumination and inhibitory dysfunctions. Using a slightly modified version of the Anderson and Green task in which participants were instructed to remember or forget positive and negative nouns, Joormann, Hertel, Brozovich, and Gotlib (2005) investigated intentional forgetting of positive and negative adjectives that depressed and control participants had learned to associate with neutral nouns. In contrast to directed forgetting studies, the authors provided multiple opportunities for the participants to practice the active suppression of the items to see if forgetting would increase with suppression training. The results demonstrated that depressed participants could be trained to forget negative words suggesting possible implications of this research for interventions.

4. Neurobiological correlates of inhibitory deficits in depression

Further support for a link between inhibitory dysfunction and depression comes from neurobiological research. Given that an extensive discussion of the neurobiology of inhibition was provided in the introductory article, we will restrict this section to studies that investigate neural correlates of inhibition in depression. As outlined by Dillon and Pizzagalli (this issue), inhibition is neurobiologically heterogenous. Still, brain regions that have been found to be critical in inhibition and working memory tasks have also been found to be differentially active in depressed and nondepressed individuals (see Davidson, Pizzagalli, Nitschke, & Putnam, 2002). Considering the importance of anterior cingulate cortex (ACC) activation in normal inhibitory functioning, it is possible that the failure of depressed individuals to inhibit negative stimuli is related to a lack of activation in the ACC. This perspective converges with findings from investigations that document abnormal ACC functioning in depression (for a review, see Davidson et al., 2002). In an early study, George et al. (1997) used positron emission tomography to compare brain activations in depressed and healthy participants while they were performing an emotional version of the Stroop task, in which they were required to name the colors in which depression-related words were presented (see Gotlib & McCann, 1984). The patient group showed an attenuated activation in left ACC and increased activation in dorsolateral prefrontal cortex (DLPFC) and parietal cortex despite no significant performance difference on the Stroop task. In contrast, Videbech et al. (2004) found no significant difference in brain activity using PET between partly medicated depressed patients and controls despite a significant group difference on the Stroop task.

More recently, Elliott and her colleagues (e.g., Elliott, Rubinsztein, Sahakian, & Dolan, 2000) used functional magnetic resonance imaging (fMRI) to measure changes in blood oxygenation level dependent (BOLD) signal in depressed and healthy participants while they performed an emotional go/no-go task (Murphy et al., 1999), in which they were required to respond to target words (*go*) but not to distractor words (*no-go*). These investigators found that, compared to nondepressed controls, depressed participants showed an attenuated response in the ventral/subgenual ACC when they responded to emotional words (vs. neutral words). Elliott et al. reported further that whereas healthy controls showed greater activation in rostral ACC than did depressed participants when they responded to happy target words, this pattern was reversed for sad target words. Depressed and nondepressed participants, however, did not differ in their performance on the go/no-go task. Group differences in patterns of neural activation, therefore, cannot be linked directly to behavior. Moreover, because Elliott, Rubinsztein, Sahakian, and Dolan (2002) used a block design, it is impossible to isolate activation patterns that were associated with the inhibitory component of the emotional go/no-go task and, therefore, to determine whether ACC abnormalities reflect specific deficits in inhibitory processing.

Activation of DLPFC has been observed in healthy participants during performance on go/no-go tasks (e.g., Garavan, Ross, Murphy, Roche, & Stein, 2002) whereas depressed participants have been found to demonstrate decreased activation in the DLPFC (e.g., Elliott, Sahakian, Herrod, Robbins, & Paykel, 1997; Mayberg et al., 1999). In a recent study using fMRI, unmedicated depressed participants exhibited significantly less activation in left DLPFC than controls while performing the digit sorting task that involves working memory and executive control despite the absence of group difference in their performance on the task (Siegle, Thompson, Carter, Steinhauser, & Thase, 2007). Other studies, however, found greater activation in DLPFC among depressed participants. For example, a recent study reported that depressed participants showed hyperactivity in the left DLPFC and in the rostral ACC compared to the control subject while performing the Stroop task (Wagner et al., 2006). Given that there were no group differences in behavioral performance, which might be attributable to an extensive pre-training of the task, hyperactivation of DLPFC in depressed participants in this study might reflect compensatory mechanism.

As outlined by Dillon and Pizzagalli (this issue), hypoactivity of the PFC may result in a decreased inhibitory influence over the amygdala, which may be closely related to reduced inhibition of emotional material, sustained negative affect, and rumination. Indeed, investigators have found the combination of DLPFC hypoactivity and sustained amygdala activity to be related to self-reported rumination in a study that examined prolonged elaborative processing of emotional information in depression using fMRI (Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). In this study, while non-depressed individuals displayed amygdala responses to all stimuli that quickly decayed after offset,

depressed individuals displayed sustained amygdala responses to negative words. This sustained response lasted throughout the following nonemotional processing trials for depressed but not for control participants. Moreover, the difference in sustained amygdala activity to negative and positive words was moderately related to self-reported rumination. In a more recent study, depressed participants showed increased and sustained amygdala activity in response to negative words while showing decreased DLPFC activity (Siegle et al., 2007). These results suggest that sustained processing of emotional information coupled with disrupted executive functioning might contribute to depression, and underscore a potentially important link between basic cognitive dysfunction and difficulties in emotion regulation, resulting in sustained negative affect in depression. In line with this hypothesis, investigators have underscored the importance of inhibitory control of limbic structures by the PFC in explaining individual differences in emotion regulation (e.g., Ochsner & Gross, 2005).

While these findings are exciting and provide more evidence for a link between inhibition, rumination and emotion regulation in depression, more studies are clearly needed. As Dillon and Pizzagalli (this issue) have suggested, interactions between ventromedial prefrontal cortex (VMPFC) and amygdala seem to play an important role in emotional inhibition. Recent studies have implicated this neural circuit in difficulties in emotion regulation and rumination in depression (Mayberg, Keightley, Mahurin, & Brannan, 2004; Ochsner & Gross, 2005; Pezawas et al., 2005). Inconsistencies in defining subregions of the PFC, however, and findings of hyper- as well as hypoactivation in these regions, sometimes on identical tasks, make it difficult to draw firm conclusions (see e.g., Elliott et al., 1997; Wagner et al., 2006). As outlined by Dillon and Pizzagalli (this issue), inhibition is neurobiologically heterogenous and different tasks likely assess different aspects of inhibition that are associated with distinctive neural pathways. Future research will hopefully increase our understanding of brain regions and neural circuits involved in cognitive inhibition and executive functioning, which will in turn lead to a better understanding of neurobiological correlates of inhibitory deficits in depression.

5. Conclusions and future directions

As outlined in the previous sections, deficits in inhibitory processes may play a central role in the occurrence of ruminative responses. According to Nolen-Hoeksema and her collaborators, what characterizes rumination and differentiates it from negative automatic thoughts is that it is a style of thought rather than just negative content (Nolen-Hoeksema, 1991). Thus, 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. While depressive rumination is characterized by negative self-focused thoughts, rumination in an angry or happy mood state features other contents although the process may be similar (Feldman, Joormann, & Johnson, in press; Rusting & Nolen-Hoeksema, 1998). More research that focuses on the underlying cognitive processes and the relation of rumination to biases in memory and attention processes is clearly needed. The majority of studies on rumination to date have been concerned with consequences of ruminative responses. While this line of research informs us about the devastating effects of rumination, it is not particularly helpful in determining why it is so difficult for some people to redirect their thoughts and control their attention before it becomes dysfunctional. Finding an answer to this question might increase our understanding of cognitive processes in depression and inform our interventions.

Although a comprehensive discussion of the following issue is beyond the scope of this article, we should nevertheless point out that the concept of inhibition has been criticized in research on attention and memory (e.g., Friedman & Miyake, 2004; MacLeod, Dodd, Sheard, Wilson, & Bibi, 2003). Thus, the construct validity of several measures that have been proposed to assess inhibition has been questioned, and some investigators have pointed out that researchers often do not justify their assumptions that specific measures actually involve inhibitory processes (Friedman & Miyake, 2004). Specifically, research on the negative priming task has led investigators to propose a number of alternative mechanisms that could underlie the observed effects. Indeed, MacLeod et al. (2003) argued that many results that are interpreted in terms of inhibitory processes can be explained without reference to this concept. This is an ongoing debate, however, and Tipper (2001) recently concluded that although different explanations have been discussed, there is no firm evidence to discount inhibition models; indeed, Anderson (2003) has been more forceful in discussing repeatedly why forgetting requires the recruitment of inhibitory control mechanisms to override prepotent responses (see also Anderson & Spellman, 1995). One possible alternative explanation for the presented results is that there is differential initial activation of positive and negative material in the depressed group in the absence of group differences in the strength of inhibition. Clearly, future studies are needed to investigate whether inhibition, or any of these proposed alternative mechanisms, provides the best explanation of the observed effects. We believe, however, that the findings of difficulties in redirecting attention, disengaging from negative material and increased elaboration of negative content that we have summarized in this article represent important result that can inform our models of cognition in depression even if the precise underlying mechanisms remain open to debate.

In summary, the studies we reviewed are important in beginning to elucidate the nature of the relations among rumination, inhibition, 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 the contents of working memory could be crucial in understanding and differentiating people who recover easily from negative affect from those who initiate a vicious cycle of increasingly negative ruminative thinking 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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