

Cognition and Depression: Current Status and Future Directions

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Abstract

Cognitive theories of depression posit that people's thoughts, inferences, attitudes, and interpretations, and the way in which they attend to and recall information, can increase their risk for depression. Three mechanisms have been implicated in the relation between biased cognitive processing and the dysregulation of emotion in depression: inhibitory processes and deficits in working memory, ruminative responses to negative mood states and negative life events, and the inability to use positive and rewarding stimuli to regulate negative mood. In this review, we present a contemporary characterization of depressive cognition and discuss how different cognitive processes are related not only to each other, but also to emotion dysregulation, the hallmark feature of depression. We conclude that depression is characterized by increased elaboration of negative information, by difficulties disengaging from negative material, and by deficits in cognitive control when processing negative information. We discuss treatment implications of these conclusions and argue that the study of cognitive aspects of depression must be broadened by investigating neural and genetic factors that are related to cognitive dysfunction in this disorder. Such integrative investigations should help us gain a more comprehensive understanding of how cognitive and biological factors interact to affect the onset, maintenance, and course of depression.

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INTRODUCTION

Depression not only changes the way we feel, it also changes how we perceive ourselves and the world around us. There is a long history of research investigating the interaction of cognition and emotion in Major Depressive Disorder (MDD). Clinicians and researchers alike have focused on cognitive processes and on the content of depressive cognition in trying to gain a more comprehensive understanding of MDD. Negative views of the self, the world, and the future, as well as recurrent and uncontrollable negative thoughts that often revolve around the self, are debilitating symptoms of depression. Moreover, biases in cognitive processes such as attention and memory may not only be correlates of depressive episodes; they may also play a critical role in increasing individuals' vulnerability for the first onset and recurrence of depression.

Cognitive theories of depression posit that people's thoughts, inferences, attitudes, and interpretations, and the way in which they attend to and recall events, can increase their risk for the development and recurrence of depressive episodes. Indeed, most cognitive theories propose vulnerability-stress hypotheses that posit that the onset of this disorder is due to the interaction of a psychological vulnerability (e.g., certain cognitions or particular ways of processing information) and a precipitating stressor (e.g., a negative life event or some other environmental factor). Importantly, one of the most effective interventions for depression, cognitive-behavioral therapy, focuses on modifying biased interpretations and dysfunctional automatic thoughts (Beck 1976) and proposes that changes in cognition will lead to improvement of other symptoms of the disorder, including sustained negative affect and anhedonia. Consequently, the primary goal of a large body of research has been to gain a more comprehensive understanding of difficulties in cognitive functioning that can result in the emotion dysregulation and sustained negative affect that are central to depression. A better understanding of the nature and role of depressive cognition promises to help advance theories of depression and improve treatments for this disorder.

More than three decades of research on cognitive factors in depression have provided impressive support for many aspects of cognitive formulations of depression. Early studies focused primarily on demonstrating that depressed and nondepressed people differ in the content of their thoughts. Recent investigations, however, have started to explore the nature of the cognitive deficits and biases in the processing of information that characterize depression. Whereas earlier studies used a variety of self-report measures, studies on cognitive biases have utilized a wide variety of experimental tasks. These investigations have generally provided support for the formulation that depression is characterized by negative automatic thoughts and biases in attention, interpretation, and memory (Mathews & MacLeod 2005).

Despite the early promise of cognitive theories of depression, important questions remain. The proposition that both depression and anxiety are characterized by biases in all aspects of information processing, for example, has received little support. A closer comparison of studies that have provided evidence for depression-related biases with studies that have not can lead to a more comprehensive characterization of cognitive processing in depression that could have important implications for models of, and interventions for, depression. Moreover, whereas numerous studies have provided evidence that cognitive biases are present during current episodes of depression, empirical support for the presence of these biases outside of current episodes is more elusive. And even fewer investigators have tested explicitly the diathesis-stress model of depression by, for example, assessing biased processing prior to the first onset of depression to examine whether it predicts depression following the experience of a negative life event. Finally, there has been little connection between cognitive theories of depression and other aspects of depressive functioning. Few studies have examined how deficits in recall, attentional biases for negative material, and mood-congruent memory are related to each other and, more importantly, how they are related to the hallmark feature of depression—sustained negative affect. Given this critical shortcoming, it is imperative that researchers attempt to understand the nature of the relation between difficulties or deficits in the processing of information and the dysregulation of emotion that is central to depressive disorders. As we describe in detail below, three important mechanisms have been implicated as potential links between biased attentional/memory processes and emotion dysregulation in depression: biased inhibitory processes and deficits in working memory (Joormann 2005); ruminative responses to negative mood states and negative life events (Nolen-Hoeksema 2000); and the inability to use positive and rewarding stimuli to regulate negative mood (Joormann & Siemer 2004, Joormann et al. 2007a).

In this review, we examine the literature on cognitive biases in depression to provide a contemporary characterization of depressive cognition. We discuss how the different biases identified in studies in this area may be related not only to each other, but also to emotion dysregulation, the hallmark feature of depression. We then briefly present treatment implications of the conclusions gleaned from these examinations. Finally, in closing, we underscore the position that the study of cognitive aspects of depression must be broadened by adopting a more integrative perspective on the processing of emotional stimuli in this disorder. In particular, we highlight the importance of investigating neural aspects of emotion regulation and of responsiveness to emotional stimuli in understanding MDD. We begin with a brief overview of the symptoms and prevalence of depression and then turn to a discussion of cognitive theories of this disorder.

DEPRESSION: CLINICAL AND EPIDEMIOLOGICAL INFORMATION

MDD is characterized by a constellation of behavioral, emotional, and cognitive symptoms, including psychomotor agitation or retardation, marked weight loss, insomnia or hypersomnia, decreased appetite, fatigue, extreme feelings of guilt or worthlessness, concentration difficulties, and suicidal ideation. To meet *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; Am. Psychiatr. Assoc. 2000) criteria for a diagnosis of MDD, a set of these symptoms has to be present for the same two-week period. Although all of these symptoms are, therefore, important in diagnosing MDD, depression is primarily a disorder of emotion dysregulation and sustained negative affect. Indeed, a diagnosis of depression requires the presence of either sustained negative affect or loss of pleasure. We should also note, however, that depression is a term that is often used imprecisely, ranging from the characterization of depressive disorders diagnosed according to DSM criteria, to the characterization of a

temporary mood state in otherwise healthy individuals. Moreover, researchers frequently use the terms “depression” and “dysphoria” interchangeably: dysphoria is commonly referred to as a form of subclinical depression defined by specific cut-off scores on measures assessing self-reported depressive symptomatology. It is important to be cognizant of these distinctions when reviewing the depression literature; in this review, we focus primarily on studies that examined participants diagnosed as depressed according to DSM criteria. Because few researchers have compared dysphoria and depression directly, it is still an open question whether results obtained with dysphoric samples generalize to clinically depressed participants.

Depression is among the most prevalent of all psychiatric disorders. Recent estimates indicate that almost 20% of the American population, or more than 30 million adults, will experience a clinically significant episode of depression at some point in their lives (Kessler & Wang 2009). In fact, the rates of depression are so high that the World Health Organization Global Burden of Disease Study ranked depression as the single most burdensome disease in the world in terms of total disability-adjusted years among people in the middle years of life (Murray & Lopez 1996). Depression is frequently comorbid with other mental and physical difficulties, most often with anxiety disorders, but also with cardiac problems and smoking (e.g., Carney & Freedland 2009). Finally, there are significant economic and social costs of depression. Kessler et al. (2006), for example, estimated that the annual salary-equivalent costs of depression-related lost productivity in the United States exceed \$36 billion. There is also mounting evidence that depression adversely affects the quality of interpersonal relationships and, in particular, relationships with spouses and children. Not only is the rate of divorce higher among depressed than among nondepressed individuals (e.g., Wade & Cairney 2000), but children of depressed parents have also been found to be at elevated risk for psychopathology (Joormann et al. 2008).

Importantly, depression is a highly recurrent disorder. More than 75% of depressed patients have more than one depressive episode, often relapsing within two years of recovery from a depressive episode (Boland & Keller 2009). Indeed, between one-half and two-thirds of people who have ever been clinically depressed will be in an episode in any given year over the remainder of their lives (Kessler & Wang 2009). This high recurrence rate in depression suggests that there are specific factors that increase people’s risk for developing repeated episodes of this disorder. In this review, we focus on one such factor: cognitive functioning and cognitive biases in the processing of emotional information.

COGNITIVE THEORIES OF DEPRESSION

Cognitive theories of depression were first formulated about 40 years ago. Even though these theories have provided the impetus for an extraordinarily productive program of research attempting to provide empirical evidence for their main propositions, this research has not led to substantive changes in cognitive models of this disorder. Beck (1976) postulated that existing memory representations, or schemas, lead individuals to filter stimuli from the environment such that their attention is directed toward information that is congruent with their schemas. Beck theorized that the schemas of depressed persons include themes of loss, separation, failure, worthlessness, and rejection; consequently, depressed individuals will exhibit a systematic bias in their processing of environmental stimuli or information that is relevant to these themes. Because of this bias, depressed people attend selectively to negative stimuli in their environment and interpret neutral and ambiguous stimuli in a schema-congruent way. Moreover, dysfunctional schemas and processing biases are presumed to endure beyond the depressive episode, representing stable vulnerability factors for depression onset and recurrence. When the dysfunctional schemas are activated by stressors, specific negative

cognitions are generated that take the form of automatic thoughts and revolve around pessimistic views about the self, the world, and the future—the cognitive triad. In more recent versions of this model, Beck differentiated between schemas that involve social relationships (sociotropy) and schemas that concern achievement (autonomy; Epstein et al. 1983). Finally, Beck's cognitive specificity hypothesis posits that these schemas are likely to be activated by congruent life events, thereby initiating a vicious cycle of negative automatic thoughts, processing biases, and depressed mood.

Research examining processing biases in depression was inspired primarily by Bower's (1981) work on mood and memory. These studies postulate that "associative networks" lead to cognitive biases in depressed individuals. These associative networks consist of numerous nodes, each containing specific semantic representations that can be activated by environmental stimuli. The activation of any one node causes the partial activation, or priming, of all the other nodes within its associative network through a process of spreading activation. Consequently, the representations of the primed nodes require less activation for access to occur than do the representations of nonprimed nodes, resulting in a processing advantage for stimuli that are related to these primed representations. Like Beck, Bower also postulates that associative networks are stable constructs; the attentional biases of depressed individuals are expected to endure beyond the depressive episode. Indeed, Ingram (1984) and Teasdale (1988) both have drawn on Bower's theory in explaining the onset, maintenance, and recurrence of depressive disorders. Importantly, virtually all cognitive theories predict that depression is characterized by biased processing of emotional material. We review the empirical evidence for this proposition below. First, however, we want to briefly discuss the important question of whether depression is associated specifically with biases in the processing of emotional material, or instead is characterized by a more general cognitive deficit that affects

the processing of both emotional and nonemotional material.

COGNITIVE DEFICITS IN DEPRESSION

Two distinct patterns of cognitive correlates of depression and dysphoria are frequently cited. First, depressed people report experiencing broad difficulties involving concentration and memory (Burt et al. 1995). Second, despite these difficulties, they report easily concentrating on negative self-focused thoughts, and they exhibit enhanced recall of mood-congruent (i.e., negatively valenced) material (Mathews & MacLeod 2005). General deficits in cognitive functioning that are associated with depression, such as concentration difficulties, distractibility, and impairments in memory, have typically been studied separately from cognitive biases, i.e., preferential processing of negative material. Few attempts have been made to integrate findings obtained in these separate lines of research.

One noteworthy exception is the resource-allocation hypothesis that postulates that because their cognitive capacity is reduced, depressed individuals have deficits in remembering and in engaging in other effortful cognitive processes (e.g., Ellis & Ashbrook 1988). The general assumptions are that the amount of resources available for cognitive operations is limited and that depression either occupies or functionally reduces these resources, for example, because resources are used by task-irrelevant emotional processing. Thus, deficits should become evident in effortful tasks; they should be detectable in effortful, resource-demanding components of memory tasks, for example, but not in automatic aspects of these tasks. Similarly, the affective interference hypothesis posits that because depressed persons are preoccupied with the processing of emotional material, their performance on tasks that require them to process emotional aspects of stimuli will be fine, but will suffer on tasks that require them to ignore emotional aspects of the stimuli and respond to other aspects of the

material (Siegle et al. 2002). For example, if depressed individuals are required to make valence judgments about words, their tendency to prioritize the processing of emotional material will aid them. If, however, they are instructed to ignore the emotional content of words while making a lexical decision judgment, their processing priorities will interfere with this demand and their performance will be impaired.

Depressed people often complain of concentration difficulties (Watts & Sharrock 1985); indeed, "difficulty concentrating" is a symptom of a major depressive episode in DSM-IV. In addition, there is a large literature that strongly suggests that depressed individuals are characterized by memory impairments (see Burt et al. 1995, Mathews & MacLeod 2005). In the frequently cited meta-analysis by Burt et al. (1995), memory impairments were reported more consistently for inpatients than for outpatients. Moreover, and not surprisingly, memory impairments were also reported in other psychological disorders. Burt et al. proposed, therefore, that memory deficits are associated with psychopathology in general rather than with depression specifically. Furthermore, in a series of studies, Hertel and collaborators (e.g., Hertel 1998, Hertel & Rude 1991) presented evidence indicating that depression-related impairments are not observed in all components of memory, but are found primarily in free recall tasks and in controlled aspects of recognition.

Overall, studies conducted thus far provide evidence that depression is associated with greater memory impairment in contexts in which (a) attention is not constrained by the task (e.g., Hertel & Rude 1991); (b) increased cognitive effort is required (see review by Hartlage et al. 1993); and (c) attention is easily allocated to personal concerns and other thoughts that are irrelevant to the task (Ellis & Ashbrook 1988). Importantly, Hertel & 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. Hertel (1998) also found comparable deficits in recall between 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. In contrast, no deficit was found for dysphoric students who were instructed 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 nondepressed people in structured situations but have problems doing this on their own initiative in unconstrained situations (Hertel 2004). Moreover, these results suggest that eliminating 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 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 be due instead to depression-related inhibitory difficulties in the processing of irrelevant information.

Other studies have investigated depression-related deficits in tasks that assess various components of working memory (WM). Channon et al. (1993), for example, found few differences between depressed and nondepressed participants on a variety of WM tasks (i.e., only on the backward digit span; see also Barch et al. 2003). Most of the tasks that are used to assess WM (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. These tasks have been criticized, however, because the relatively slow presentations that are used to ensure perception might allow for chunking and active rehearsal of material and, therefore, might reflect memory deficits rather than deficits in attention (Rokke et al.

2002). Using an attentional blink paradigm that involves rapid serial presentations, Rokke et al. found significant group differences in performance between moderately to severely dysphoric (Beck Depression Inventory scores over 21) and nondysphoric participants, but only under demanding dual-tasking conditions. It is important to note, however, that only nine moderately to severely dysphoric participants were included in this study, so these findings should be interpreted with caution.

Rose & Ebmeier (2006) reported that depressed patients were slower and less accurate than were controls 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 found further that the performance deficit on the n-back task was correlated with number of hospitalizations and with the longitudinal course of the disorder. Importantly, Harvey et al. did not find depression-associated differences on a number of other tasks assessing WM functioning, including a digit span. Consistent with these findings, Egeland et al. (2003) concluded from the results of their study that reduced performance on WM tasks in depression is due not to a specific deficit in executive functioning, but to a nonspecific reduction in speed and to a loss of vigilance that is consistent with a lack of effort. Grant et al. (2001) 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 fewer 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 the operation of depression-related deficits in the generation and maintenance of problem-solving strategies and difficulties in set switching (see also Harvey et al. 2004). Importantly, though, there was no evidence of deficits in executive functioning on any of the other tasks administered in this study. Grant et al. concluded that pervasive cognitive deficits most likely characterize

elderly depressed people and severely depressed inpatients who present with psychotic features (see Harvey et al. 2004 and Rose & Ebmeier 2006 for similar conclusions). In a recent review paper, Castaneda et al. (2008) concluded that deficits in certain aspects of executive control and attentional deficits most likely characterize depressed people whereas evidence for learning and memory deficits is more mixed. They also pointed out, however, that there is significant variability in the extent to which studies report deficits and that this variability is due to the subtype of depression (with deficits being most prominent in psychotic depression) and to the age of the participants (with deficits being most prominent in older depressed adults). It is also important to keep in mind that it can be difficult to differentiate between cognitive deficits and a lack of motivation that often characterizes depressed patients (Scheurich et al. 2008).

In sum, surprisingly little empirical support has been found to date for pervasive depressive deficits in general cognitive functioning. Indeed, the bulk of evidence points to depression-associated deficits in the control of attention rather than to limited processing capacity. When depressed participants' attention is well controlled by the demands of the task and they have no opportunity to ruminate, no depressive deficits are found. Focusing attention requires individuals to inhibit task-irrelevant thoughts. Thus, the findings reviewed here support the affective interference hypothesis and the proposition that depressed individuals are characterized by reduced cognitive control. These results also suggest that examining how depressed individuals process emotional information may help us gain a better understanding of their difficulties in the processing of neutral material.

BIASED PROCESSING OF EMOTIONAL INFORMATION IN DEPRESSION

Cognitive models of depression posit that depressed individuals exhibit cognitive biases in all aspects of information processing, including

memory, interpretation, and perception and attention (Mathews & MacLeod 2005). While these theoretical predictions are straightforward, the empirical results are not. For example, a current controversial question concerning cognitive theories of emotional disorders is whether there are, in fact, negative biases in memory, interpretation, and attention in depression.

Memory

Overall, there is strong evidence for biased memory processes in depression (Mathews & MacLeod 2005). Indeed, preferential recall of negative compared to positive material is one of the most robust findings in the depression literature (Mathews & MacLeod 2005, Matt et al. 1992, Williams et al. 1997). In a meta-analysis of studies assessing recall performance, Matt and colleagues found that people with major depression remembered 10% more negative words than they did positive words. In contrast, nondepressed controls exhibited a memory bias for positive information in 20 of 25 studies. It should be noted, however, that memory biases are found most consistently in free recall tasks, and may be restricted to explicit memory. Results of studies using recognition or implicit memory measures have been much less consistent. In his review of the implicit memory literature, Watkins (2002) reported that, across studies, no bias is found in depressed participants when the encoding and/or the recall of the emotional material depend purely on perceptual processing. For example, if depressed participants are asked to count the letters in emotional words at encoding and to complete word stems or word fragments at recall, no evidence of an implicit memory bias is obtained (Watkins et al. 2000). If, however, participants are asked to rate the recency of their experience with the word or to imagine themselves in a scene involving the word at encoding, and are asked to freely associate to a cue word or to provide a word that fits a given definition, implicit memory biases are obtained more consistently. Encoding and recall in these latter studies require

semantic instead of pure perceptual processing of the material. This suggests that depressive deficits are due in large part to differences in the elaboration of the emotional material.

Depression is associated not only with enhanced recall of negative events, but also with the recall of rather generic memories, despite instructions to recall specific events (i.e., overgeneral memory; see Williams et al. 2007 for a review). On the autobiographical memory test (AMT), depressed participants respond to positive and negative cues with memories that summarize a category of similar events. Importantly, this research has demonstrated that overgeneral memories are associated with difficulties in problem solving, with deficits in imagining specific future events, and with longer durations of depressive episodes (Raes et al. 2005). Moreover, overgeneral memories remain stable outside of episodes of the disorder and have been shown to predict later onset of depressive episodes in postpartum depression (Mackinger et al. 2000), and to predict depression following life events in students and following unsuccessful *in vitro* fertilization (van Minnen et al. 2005). Brittlebank et al. (1993) found that overgeneral recall of autobiographical memories, particularly for positive memories, predicted less complete recovery from major depression at a seven-month follow-up assessment. In contrast, Brewin et al. (1999) found that overgeneral recall of autobiographical memories did not predict recovery from depression, although intrusive memories of life events did predict recovery. Furthermore, the extent to which individuals retrieve overgeneral memories predicted delayed recovery from affective disorders (Daglish et al. 2001). Williams (1996) proposed that overgeneral memory is a form of emotion regulation. That is, individuals attempt to minimize negative affect attached to distressing memories by blocking access to details of such memories or by retrieving these memories in a less specific way. Williams et al. (2007) proposed further that individual differences in cognitive control, and specifically in inhibitory dysfunction, may

underlie overgeneral recall in depression, a proposition that we examine more closely later in this review. Thus, understanding overgeneral memory in the context of emotion regulation in depression is an important goal for future research. In sum, negative biases in memory appear to characterize depressed individuals; in fact, depression is associated not only with increased accessibility of negative material, but also with the recall of overgeneral memories.

Interpretation

There is consensus that anxious individuals favor negative interpretations of ambiguous stimuli and often do so in tasks that indicate that these biases operate on an automatic level (see Mathews & MacLeod 2005 and Zinbarg & Yoon 2008 for reviews). For example, individuals with panic disorder interpret descriptions of physical sensations as symptoms of catastrophic disease (Clark 1988), and individuals diagnosed with social phobia overestimate both the likelihood of negative social events and the negative consequences of these events (Foa et al. 1996). Because earlier studies relied heavily on participants' self-reports of their interpretations, their results may be confounded with response bias or demand issues (MacLeod & Cohen 1993). To overcome these limitations, researchers developed alternative techniques, mainly based on priming paradigms, to assess interpretive bias without asking participants to emit or endorse alternative response options. These more recent studies confirmed the presence of negative interpretive biases (e.g., MacLeod & Cohen 1993, Yoon & Zinbarg 2008) or the absence of positive interpretive biases (e.g., Hirsch & Mathews 2000) in anxious participants.

Results have been much more equivocal regarding whether depression is characterized by similar automatic biases in interpretation. Butler & Mathews (1983), for example, presented clinically depressed participants with ambiguous scenarios and found that, compared to nondepressed participants, depressed individuals ranked negative interpretations higher than they did other possible interpretations. In a

study assessing biases using participants' latencies to respond to target words that were presented after ambiguous sentences, Lawson & MacLeod (1999) found no interpretation bias in depressed individuals. Using a similar task, Bisson & Sears (2007) also failed to find evidence of an interpretive bias in depression even after a negative mood induction. Lawson et al. (2002) examined the magnitude of the startle response during imagery elicited by emotionally ambiguous text in depressed and nondepressed participants. Using this procedure, Lawson et al. found evidence of more negative interpretations in their depressed than in their nondepressed samples and concluded that the failure to find a bias in previous studies was due to the use of response latencies as the dependent variable. Rude et al. (2002) found that a measure of interpretation bias, the Scrambled Sentences Test, predicted increases in depressive symptoms after 4–6 weeks in a large sample of undergraduate students, especially when administered under cognitive load. Finally, Dearing & Gotlib (2009) recently reported a negative interpretation bias in never-disordered daughters of depressed mothers, providing evidence for a role of these biases in increasing risk for the onset of depression. Clearly, further studies are needed that carefully and systematically investigate interpretive biases in depression.

Perception and Attention

The question of whether depression is associated with enhanced and, perhaps, automatic perception of emotion-relevant cues has been investigated in studies using subliminal material, material with low degrees of emotional intensity, and fast presentation times. Studies have used disorder-specific material or material that is relevant to emotion across disorders, such as facial expressions of emotion. Numerous investigations using a number of different tasks have provided evidence in anxiety disorders for biased processing of subliminally presented anxiety-provoking stimuli (e.g., Bradley et al. 1995, Mathews et al. 1996). Strikingly, few studies to date have found similar biases

in clinically depressed participants, even when depression-relevant stimuli have been masked in order to investigate unconscious processing (see Mathews & MacLeod 2005 for a recent review of this literature).

Some older studies, for example, investigated perceptual thresholds for tachistoscopically presented emotional and neutral words in depressed and nondepressed participants. Powell & Hemsley (1984) used neutral words to individually calibrate presentation times for recognition thresholds and subsequently presented neutral and negative words at this threshold. The authors reported that depressed participants took longer than did controls to recognize neutral words but did not differ in response times to negative words. Moreover, the depressed participants recognized a higher ratio of negative to neutral words. Mogg et al. (1993) similarly used a modified Stroop task and presented anxiety-related, depression-related, positive, and neutral words either subliminally (followed by a mask) or supraliminally. In the emotion Stroop task, individuals with anxiety disorders typically take longer to respond to threat words compared to neutral words, suggesting that their attention is “grabbed” by the content of the threat words. Whereas anxious participants exhibited slower color naming of all negative words at both subliminal and supraliminal exposure durations, depressed participants did not differ from control participants in the subliminal condition (see also Lim & Kim 2005). Yovel & Mineka (2005) also failed to find a relation between Stroop biases for subliminally presented depression-related words and self-reported depressive symptoms in a sample of undergraduate students. Importantly, using an emotion Stroop task, Bradley et al. (1995) found that only patients with GAD who were *not* comorbid with depression exhibited biased processing for negative words; GAD patients who were also diagnosed with depression did not differ from the control participants.

Several investigators have used the visual-probe, or dot-probe, task with briefly presented and masked emotional words to examine

automatic processing biases in depression. In this 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 either the neutral or the emotional stimulus. Allocation of attention to the spatial position of the stimulus is determined from response latencies to the probes. Here, too, the results have not been encouraging. Mathews et al. (1996) found evidence for biased processing in depressed patients only when the stimuli were presented for relatively long exposure durations (permitting awareness of the stimuli). It is important to note, however, that Mathews et al. did not use depression-relevant stimuli but instead used anxiety-relevant words. Mogg et al. (1995) similarly reported biased processing in the subliminal condition of a dot-probe task for participants diagnosed with an anxiety disorder but no bias for their depressed sample. Mogg et al. used depression-relevant and anxiety-relevant words but did not find an effect of word category on the performance in the depression group. Finally, Bradley et al. (1997) reported that whereas induced or naturally occurring sad mood was associated with increased attention to negative words at long exposure durations, there was no effect when the words were presented briefly and masked (see also Donaldson et al. 2007). One important difference between the depression and anxiety studies is that whereas studies of anxiety have used both words and pictures, most of the subliminal Stroop and dot-probe studies in depression have used only words as stimuli. Few published dot-probe studies have examined biased processing of subliminally presented pictures or emotional faces in depressive disorders, and it remains to be seen whether these kinds of stimuli lead to different results.

The only exception to this general pattern of a lack of processing biases in depression when stimuli are presented subliminally is a handful of studies using lexical decision tasks, in which participants must decide whether a stimulus is a word. The lexical decision is preceded

by the presentation of a subliminally presented prime. The few studies using this design in depression research have produced mixed results. Matthews & Southall (1991), for example, found a priming effect for neutral words, but not for positive or negative words, in depressed patients using subliminal primes. In contrast, Bradley et al. (1994) used subliminal priming followed by a lexical decision task and found that nonclinical participants with high levels of self-reported negative affect showed greater subliminal priming of depression-relevant than of neutral words than did participants with low levels of negative affect. Moreover, the subliminal priming effect was more closely associated with level of depression than with level of anxiety. Bradley et al. (1995, 1997) replicated this finding in separate samples of students who scored high on a measure of negative affect and of diagnosed depressed participants.

Finally, some investigators have used a dichotic listening task to assess automatic processing of negative material in depression. Ingram et al. (1994), for example, found that remitted depressed participants who were exposed to a negative mood induction made more shadowing errors (indicating interference in processing the to-be-attended information) when negative or positive words were presented in the unattended channel; this effect was not found in participants who had no mood induction. McCabe & Gotlib (1993) combined the dichotic listening task with a secondary task in which participants' response times to a light probe were assessed while they were engaged in the dichotic listening task. Depressed participants took longer to respond to the light probe when negative words, compared to positive or neutral words, were presented on the attended channel. Interestingly, this effect disappeared after remission from the depressive episode.

Researchers have also examined the processing of facial expressions of emotion to investigate whether depressed participants are prone to perceive sadness. Facial expressions of emotion are powerful stimuli that represent salient features of the social environment

(Hansen & Hansen 1994). Individuals use facial expressions to monitor emotional reactions of their interaction partners and adjust their behavior accordingly (Salovey & Mayer 1990). Misreading facial expressions, particularly when they are subtle, can have profound consequences for the interpretation of a situation and can affect the selection and the effectiveness of emotion-regulation strategies such as reappraisal. Whereas some investigators have found that depressed individuals are characterized by deficits in the processing of all (i.e., emotional and neutral) facial expressions (e.g., Carton et al. 1999), other researchers have failed to replicate this finding (e.g., Ridout et al. 2003). Numerous investigators have found depression to be associated with negative biases in the processing of specific types of emotional faces (Rubinow & Post 1992). All of these studies, however, have examined full-intensity facial expressions. The question of whether depression is characterized by early perception of emotional expressions has been examined in studies using subtle expressions of emotion. These studies suggest that depression is associated primarily with difficulties identifying subtle positive emotional expressions; depressed individuals do not more easily identify low-intensity sadness (Joormann & Gotlib 2006).

Taken together, empirical findings that depression is associated with faster identification of mood-congruent material or a faster orienting toward negative stimuli are mixed. These results are in line with Williams et al. (1997), who proposed that depressed persons are not characterized by biases in early processing. Instead, these authors suggested that anxiety-congruent biases are observed in tasks that assess the early, orienting stage of processing, prior to awareness. In contrast, depressive biases are observed in strategic elaboration and make it difficult for depressed people to disengage from negative material. Importantly, there is empirical evidence to support these predictions.

Individuals diagnosed with anxiety disorders have been found to attend to threat stimuli that

are presented supraliminally (for reviews, see Bar-Haim et al. 2007, Mathews & MacLeod 2005, Zinbarg & Yoon 2008). Most studies in this area of research have used either the modified Stroop task or an attention allocation paradigm, such as the dot-probe task, but with longer-exposure durations of the emotional stimuli. Interestingly, again, numerous studies have failed to find similar biases in depression (e.g., Mogg et al. 1993). Most investigations using the Stroop task do not find differences between depressed participants and controls, even if stimuli are presented supraliminally (e.g., Holmes & Pizzagalli 2008, Mogg et al. 1993). Bradley et al. (1995), for example, found that depression was not associated with increased Stroop interference, and the well-replicated interference effect in anxiety was not present in GAD patients who had comorbid depression. Several investigators have used the dot-probe task with supraliminal stimuli to investigate processing biases in depression. Here, too, results have not been encouraging (e.g., Mogg et al. 1995). Further, in a recent study using an attention task with happy, sad, angry, and neutral faces at both long and short exposure durations, Koster et al. (2006) found no correlations with symptoms of depression or anxiety.

It may be premature, however, to conclude that depressed persons are not characterized by attentional biases in later stages of processing. Recent studies using the dot-probe task, for example, have reported selective attention in depression, but only under conditions of long stimuli exposures. Bradley et al. (1997) reported a mood-congruent bias on the dot-probe task for both induced and naturally occurring dysphoria when stimuli were presented for 500 or 1000 ms, but not when they were presented for brief durations (14 ms). Using a dot-probe task with emotional faces as stimuli, Gotlib et al. (2004) found an attentional bias for negative faces that were presented for 1000 ms in clinically diagnosed depressed participants (see also Donaldson et al. 2007). In two recent studies, Joormann & Gotlib (2007) and Joormann et al. (2007b)

replicated these findings in samples of remitted depressed adults and nondisordered girls at high risk for depression due to their mothers' psychopathology, respectively. These findings suggest that attentional biases are not simply a symptom of depression or a scar of a previous depressive episode, but may play an important role in the vulnerability to depression.

Overall, these results indicate that depressed individuals do not direct their attention to negative information more frequently than do control participants, but once it captures their attention, they exhibit difficulties disengaging from it. Similar difficulties in disengaging attention from negative material have now been demonstrated using other attention tasks. Rinck & Becker (2005), for example, reported that depressed participants did not show enhanced detection of depression-related words in a visual search task but were more easily distracted by negative words. Eizenman et al. (2003) used eye-tracking technology to continuously monitor point of gaze. Depressed individuals spent significantly more time looking at pictures featuring sadness and loss and had significantly longer average glance durations for these pictures than did nondepressed controls. Similarly, Caseras et al. (2007) found in an eye-tracking study that depressed individuals were no more likely than controls to shift their attention toward negative stimuli, but once their attention was focused on negative stimuli, they spent significantly more time looking at these stimuli than did nondepressed controls.

In sum, these findings suggest that depression is characterized by a selective bias for negative information, but that the nature of this bias is different from that reported in anxiety disorders. Depressed individuals may not automatically orient their attention toward negative information in the environment, but once such information has come to be the focus of their attention, they may have greater difficulty disengaging from it. Thus, depression appears to be characterized by problems with disengagement from negative stimuli. Considered together, these findings suggest that depression is associated with a selective bias for negative

information, but that this bias does not operate throughout all aspects of attention.

INHIBITION AND COGNITIVE CONTROL

Difficulties disengaging attention from negative material may reflect deficits in inhibitory control that are associated with depression. Overriding prepotent responses and inhibiting the processing of irrelevant material that captures attention are core abilities that allow us to respond flexibly and to adjust our behavior and emotional responses to changing situations. Cognitive control is related to the functioning of executive control processes, such as inhibition in WM (Hasher et al. 1999). WM is commonly described as a system for the active maintenance and manipulation of information and for the control of attention (Baddeley 1986). It is a limited-capacity system that provides temporary access to a select set of representations in the service of current cognitive processes (Cowan 1999). Thus, WM reflects the focus of attention and the temporary activation of representations that are the content of awareness. Given the capacity limitation of this system, it is important that the contents of WM be updated efficiently, a task controlled by executive processes (e.g., Friedman & Miyake 2004, Hasher et al. 1999). Executive processes must selectively gate access to WM, shielding it from intrusion from irrelevant material, and must also discard information that is no longer relevant. In this context, individual differences in the experience and resolution of interference are likely to affect cognitive and emotional functioning. The occurrence of intrusive thoughts might be one consequence of poor interference resolution. Indeed, increased interference from irrelevant representations has been proposed as a source of low WM capacity (Engle et al. 1999) and has been found to characterize various populations, including older adults (Hasher et al. 1991), children with attention deficit disorder (Bjorklund & Harnishfeger 1990), patients with obsessive-compulsive disorder (Enright & Beech

1990), and patients with schizophrenia (Frith 1979).

Several researchers have suggested that depression is associated with deficits in executive functioning (Hertel 1997, Joormann 2005). Indeed, there is emerging evidence that depression is characterized by difficulties in the inhibition of mood-congruent material that could result in prolonged processing of negative, goal-irrelevant aspects of presented information and thereby hinder recovery from negative mood and lead to the sustained negative affect that characterizes depressive episodes. Theorists have suggested that deficits in cognitive inhibition lie at the heart of biases in memory and attention in depression and set the stage for ruminative responses to negative events and negative mood states. A number of experimental paradigms have been developed that have the potential to test inhibition models (e.g., Anderson & Bjork 1994). Below we discuss several of these designs, such as negative priming (Tipper 1985) and directed forgetting (Bjork 1972).

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 longer 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 inhibition 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). The area of research in which negative priming has been used most widely involves the identification of individual differences associated with the effect. Linville (1996) was the first to investigate negative priming in depression. She reported that depressed individuals were less likely to inhibit distracting information than were nondepressed controls. 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. Whereas control participants were slower to respond to letter strings that they had been asked to ignore on an earlier trial, depressed individuals failed to show this effect. Similarly, MacQueen et al. (2000) used a negative priming task in which they systematically varied color and location of prime and target, and reported reduced inhibition of distractors in depressed participants. Although these results support the proposition that depression is related to inhibitory dysfunction, inhibitory deficits might be even more prevalent in the processing of emotional information. In particular, the observation of negative automatic thoughts and ruminations about negative information in depression leads to the hypothesis that there is a valence-specific inhibitory deficit in depression in which inhibition is selectively reduced for negative stimuli.

The negative affective priming (NAP) task was designed to assess inhibition in the processing of emotional information (Joormann 2004). This task assesses response times to positive and negative material that participants were instructed to ignore. Joormann (2004) found that dysphoric participants and participants with a history of depressive episodes exhibited reduced inhibition of negative material. Thus, these participants responded more quickly when a negative target was presented after a to-be-ignored negative distractor on the previous trial. As predicted, no group

differences were found for the positive adjectives. In a related study, participants who obtained high scores 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). Importantly, 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.

Negative priming tasks assess only one aspect of inhibition: the ability to control the access of relevant and irrelevant material to WM. 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, Nigg 2000). In addition, cognitive inhibition operates at different stages of the processing of information, for example, by preventing off-goal information from having access to WM or by reducing the activation of information that was once relevant, but now is irrelevant because of a change in goals. Whereas results from NAP studies suggest that depression involves difficulties keeping irrelevant emotional information from entering WM, it is unclear whether depression is also associated with difficulties removing previously relevant negative material from WM. 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 & Gotlib (2008) used a modified Sternberg task 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 WM that were declared to be no longer relevant and, therefore, assesses a person's ability to remove irrelevant information from WM (Oberauer 2005a,b). We found that participants diagnosed with MDD exhibited difficulties removing irrelevant negative material from WM. Specifically, 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 WM that were declared to be no longer relevant. Importantly, this pattern was not found for positive material. Joormann & Gotlib (2008) also found that difficulty removing negative irrelevant words from WM was highly correlated with self-reported rumination, even after controlling for level of depressive symptoms.

In sum, therefore, these findings indicate that depression is associated with inhibitory impairments in the processing of emotional material, specifically, with difficulties removing irrelevant negative material from WM. We recently replicated these findings using a task developed by Nee & Jonides (2008) that allows us to assess two aspects of inhibition within the same task: access of information to WM and discarding irrelevant material from WM. Interestingly, in this task, depressed participants did not differ from nondepressed controls in their ability to keep irrelevant negative material from entering WM. We did, however, obtain a group

difference in the ability to discard negative material from WM; no group differences were obtained in the processing of positive or neutral material. Difficulties discarding negative material in the depressed group were also correlated with self-reported rumination. Finally, using a task that requires participants to resort material in WM, Joormann et al. (2009b) demonstrated that MDD participants had difficulties manipulating negative material in WM and that this deficit was associated with self-reported proneness to rumination.

It is likely that deficits in cognitive control not only affect people's ability to disengage attention from irrelevant material, thereby increasing unwanted thoughts, but also make it difficult for them to intentionally forget unwanted material. As we described above, investigators have consistently documented the operation of memory biases in depression. Theorists have suggested that analogous processes underlie both selective retrieval of target items from memory and selective attention to objects in the external environment (Anderson & Spellman 1995). Consequently, if inhibitory dysfunction is found in depressed individuals on selective attention tasks, it may also be detectable on memory tasks. The postulation that depression is associated with such deficits in executive control has been tested in directed-forgetting tasks in which participants are instructed to forget previously studied material at some point during the experiment. Later recall is tested, however, for material that was to be remembered and material that was to be forgotten (Bjork 1972). In a study using neutral material, Cottencin et al. (2008) reported increased recall of to-be-forgotten words and decreased recall of to-be-remembered words in depression. Using positive and negative words, Power et al. (2000) reported differential directed-forgetting effects for depressed and nondepressed participants. Specifically, the depressed participants exhibited a facilitation effect for negative words after the "forget" instruction. It is important to note that the effect was only found when the adjectives were processed in a self-relevant manner. Similar

findings were obtained by Joormann & Tran (2009), who demonstrated that participants who scored high on a trait measure of rumination exhibited reduced forgetting of negative material in this task. These participants also exhibited increased recall of negative words that were not presented during the learning phase. These results remained stable when depression scores were included as a covariate. Interestingly, a recent study reported that depressed participants were more likely to falsely recall negative material that had never been presented during the learning trials (Joormann et al. 2009c).

Hertel & Gerstle (2003) reported additional evidence for reduced inhibition of negative words in dysphoric students. These authors used a paradigm that was originally proposed by Anderson & 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 either recalling the target word or suppressing (i.e., making 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 & Gerstle (2003) found that recall from sets assigned for suppression practice was greater in the group of dysphoric participants, with a tendency toward 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 difficulties. Using a slightly modified version of the Anderson & Green (2001) task in which participants were instructed to remember or forget positive and negative nouns, Joormann et al. (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 examine whether

forgetting would increase with suppression training. Importantly, depressed participants could be trained to forget negative words, suggesting intriguing implications of this research for interventions (see also Joormann et al. 2009e).

In sum, the literature reviewed above does not indicate that depression is associated with biases in all aspects of information processing; rather, it suggests a very specific difference between depressed and nondepressed individuals in cognitive functioning. Depression is not necessarily characterized by a general cognitive deficit or by a high level of alertness in the processing of negative material. Instead, whereas anxiety disorders are associated with quick detection of and fast orienting toward threat-related stimuli, results of studies investigating the automatic processing of negative material in depression are equivocal. Once negative material has become the focus of attention, however, depressed individuals are prone to elaborate on it and have difficulty stopping or inhibiting the processing of this material. This specific difficulty is likely to have important consequences for depressed people's ability to recover from negative affect and, consequently, may represent an important link between cognition and emotion dysregulation in this disorder.

COGNITIVE BIASES AND EMOTION REGULATION IN DEPRESSION

Sustained negative affect is a core feature of depression. It is likely that there are a number of factors that affect people's ability to recover from negative affect in this disorder, including knowledge of effective strategies and motivation to implement them (Campbell-Sills & Barlow 2007). Biases in the processing of emotional material, however, may be particularly detrimental for people's ability to regulate negative emotional states. Indeed, cognition plays a critical role in human emotion. According to cognitive theories of emotion, cognitive appraisals determine whether an emotion is experienced and, if it is, which emotion is

experienced. Thus, cognition is the primary route through which emotions are regulated. Biases and deficits in cognitive functioning, therefore, are posited to affect people's ability to regulate emotion and mood states, potentially increasing their vulnerability to develop emotional disorders (Joormann et al. 2009d).

Individual differences in cognitive functioning may affect emotion regulation in various ways. For example, the ability to reinterpret emotion-eliciting situations and to quickly disengage attention can directly affect emotional experience. Other cognitive processes, however, do not change emotional responding directly, but instead affect the ease with which individuals can utilize emotion-regulation strategies. Individual differences in cognitive control, for example, can influence a person's ability to reappraise events. Biases in attention and memory, in particular, may lead to inflexible and automatic appraisals that make it difficult to use deliberate reappraisals of situations to regulate emotions (Siemer & Reisenzein 2007). Indeed, such biases can lead persons to use regulatory strategies that are maladaptive. For example, implicit memory for details of a traumatic event may lead an individual to select or avoid a specific situation, even though that situation may not be as aversive or elicit the degree of negative affect as was expected. Given that these processes do not affect emotion directly, cognitive flexibility and cognitive biases are not traditionally considered emotion-regulation strategies. Nevertheless, it is important to examine their role in emotion regulation because they can affect the selection and the effectiveness of various regulation strategies.

Surprisingly few studies have examined emotion-regulation strategies in depression. Moreover, most of these investigations have relied on self-report data and have studied individuals who score high on depression inventories rather than samples of diagnosed depressed persons. Still, the findings of these studies support the formulation that the more-frequent use of certain strategies (e.g., emotion suppression, rumination, catastrophizing) and less-frequent use of other strategies (e.g., reappraisal,

self-disclosure) are related to symptoms of depression and anxiety (Garnefski & Kraaij 2007). Moreover, recent studies suggest that impaired emotion regulation is not only a characteristic of individuals who are currently depressed, but also can be evident following recovery from this disorder (e.g., Ehring et al. 2008).

As we note above, depression is associated with difficulties disengaging attention from negative material, with deficits in inhibitory control, and with biases in memory. All of these anomalies have the potential to affect the use and effectiveness of emotion-regulation strategies, as well as the efficacy of more-automatic regulation processes. The ability to ignore situational cues that elicit an emotion or to quickly disengage from these cues may play an important role in preventing the onset of emotions and in regulating emotional responses. Thus, difficulties in disengaging attention are likely to hinder people's use of attentional redirection or deployment as an emotion-regulation strategy. This in turn increases the probability that certain appraisals will follow. Given depressed people's difficulties in disengaging from negative material, their ability to use attentional deployment as a regulatory strategy is likely to be impaired. Indeed, some studies have provided evidence for an association between attentional biases and mood changes in response to exposure to a stressor. Compton et al. (2000), for example, demonstrated that a reduced ability to disengage attention was associated with increased reactivity to a distressing film clip. Individual differences in orienting, however, were not found to be related to the mood response. Similarly, Ellenbogen et al. (2002) reported that people undergoing a stressful task were faster to shift attention away from negative material and that these attentional shifts were associated with more negative mood in response to the stressor. In a related study, these authors showed that the ability to disengage from supraliminally presented dysphoric pictures was associated with less change in negative mood ratings in response to a subsequent stress task but did not affect neuroendocrine responding. In contrast, rapid disengagement from masked threat

pictures affected neuroendocrine responding to the stressor but not mood (Ellenbogen et al. 2006). Beevers & Carver (2003) demonstrated that changes in attentional bias for negative but not positive words following a negative mood induction interacted with life stress to predict onset of depressive symptoms in college students, and MacLeod & Hagan (1992) found that women who displayed the most pronounced bias toward negative information later reported the greatest amount of distress upon learning that they had been diagnosed with cervical cancer. Thus, there is evidence both that depression is characterized by difficulties disengaging from negative material and that difficulties in disengagement are related to impaired emotion regulation.

Biases in memory may also affect emotion regulation in important ways. For example, memory biases may influence people's perception of a specific situation, change their appraisals, and guide their attention toward specific aspects of that situation. In particular, biased recall can have significant consequences if it depends on implicit memory. Implicit memory for stimuli that were present during a traumatic event may, for example, lead to unintentional selection (or avoidance) of specific situations and to the appraisal of relatively harmless situations as life threatening, as frequently observed in posttraumatic stress disorder (McNally 1997). The accessibility of information in memory may affect emotional responses and interfere with the selection and use of effective emotion-regulation strategies. Moreover, memories themselves can be potent in regulating emotions. For example, recent studies have demonstrated that memories of unpleasant events fade more quickly than do memories of pleasant events and that this differential fading is associated with happiness (Walker et al. 2003). Further, recalling positive autobiographical memories can repair an induced negative mood state (Joormann & Siemer 2004), and remembering positive events and forgetting negative events has been found to be associated with increased well-being over the lifespan (Charles et al. 2003). Thus,

selective recall not only can affect other emotion-regulation strategies (e.g., situation attention or selection), it is also an effective strategy for directly changing emotions and mood states. Indeed, investigators have documented that mood-incongruent recall is often used as a mood-repair strategy in response to a negative mood induction (e.g., Rusting & DeHart 2000).

Studies investigating the use and effectiveness of mood-regulation strategies in depression suggest that, in contrast to non-depressed persons, depressed individuals are unable to use positive autobiographical memories to regulate induced negative mood states. In two studies, we examined the formulation that dysphoria and rumination are critical factors in determining the occurrence of mood-congruent memory retrieval as opposed to mood-repair processes (Joormann & Siemer 2004). Although nondysphoric participants' mood ratings improved under distraction as well as under mood-incongruent recall instructions, dysphoric participants did not benefit from the recall of positive memories, whereas distraction seemed to alleviate their sad mood. In a recent investigation, we replicated these findings in a sample of currently depressed participants. Interestingly, previously depressed participants exhibited similar difficulties in repairing their negative mood with positive memories (Joormann et al. 2007a). Overall, the results are largely consistent with the literature on mood regulation (Rusting & DeHart 2000) and support the notion that mood-incongruent recall is used as a mood-repair strategy in response to a negative mood induction. These results suggest that depression is associated with problems in utilizing a common and effective emotion-regulation strategy and that these difficulties may be related to the biases in memory that characterize this disorder.

Reappraisal is also an important emotion-regulation strategy. Cognitive processing, including biases in attention and memory and individual differences in cognitive control, are likely to affect individuals' ability to use this strategy effectively. Because the experience of a

mood state or an emotion is generally associated with the activation of mood-congruent representations in WM, difficulties in controlling the contents of WM should affect emotion regulation (Siemer 2005). In addition, effective reappraisal may depend on a person's ability to override (automatic) attention and interpretation biases that lead to unwanted appraisals of the emotion-eliciting cues. Because replacing automatic appraisals with alternative evaluations of the situation requires cognitive control, the ability to control the contents of WM is certain to play an important role in emotion regulation. Thus, an inability to appropriately expel mood-congruent items from WM as they become irrelevant would lead to difficulties attending to and processing new information and might also result in rumination and the use of other maladaptive emotion-regulation strategies. Difficulties inhibiting salient but irrelevant thoughts and memories would also discourage the use of more effective emotion-regulation strategies, such as reappraisal.

As we outline above, deficits in inhibitory processes may also play a central role in the occurrence of ruminative responses. According to Nolen-Hoeksema and her colleagues, rumination is a particularly detrimental response to negative affect that hinders recovery from negative mood and prolongs depressive episodes (Nolen-Hoeksema et al. 2008). 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 et al. 2008). 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 the thoughts. In an extensive program of experimental and correlational studies, Nolen-Hoeksema and colleagues investigated rumination in depression and dysphoria and examined how this response style exacerbates sad moods and predicts future depressive episodes (e.g., Morrow & Nolen-Hoeksema 1990). Self-reported levels of rumination have been found to predict higher levels of dysphoria in prospective studies

with nonclinical samples, even after controlling for initial differential depression levels (e.g., Nolen-Hoeksema & Morrow 1991). Moreover, studies have shown that rumination predicts higher levels of depressive symptoms and onset of major depressive episodes and mediates the gender difference in depressive symptoms (Nolen-Hoeksema 2000, Nolen-Hoeksema et al. 2007). Research also indicates that rumination enhances the operation of cognitive biases. Thus, dysphoric participants who were induced to ruminate endorsed more negative interpretations of hypothetical situations, generated less-effective problem-solving strategies (Lyubomirsky & Nolen-Hoeksema 1995), and exhibited increased recall of negative autobiographical memories (Lyubomirsky et al. 1998). Similarly, Singer & 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.

Davis & Nolen-Hoeksema (2000) reported that ruminators made more errors on the WCST than did nonruminators. Because the WCST measures executive control and cognitive flexibility, these results provide empirical support for the hypothesis that rumination is related to the executive control component of WM. In addition, Joormann (2006) reported a correlation between rumination and deficits in cognitive inhibition as assessed by negative priming, and Joormann & Gotlib (2008) found a correlation between rumination and the ability to remove irrelevant negative material from WM. These findings suggest that deficits in executive control and inhibition are related to sustained processing of negative material and rumination, which in turn maintains the negative mood state and hinders recovery from negative affect.

TREATMENT IMPLICATIONS

In this final section of this review, we describe what we believe are exciting developments in research examining clinical interventions that

build on the findings discussed above. Recent studies examining the effectiveness of modifying cognitive biases have demonstrated that training biases and improving cognitive control reduces other symptoms of emotional disorders. For example, training highly anxious people to disengage their attention from threat material has been found to lead to changes in mood and reduced reactivity to stressful events. In a typical training paradigm, the dot probe is presented more frequently in the location of the nonthreat-relevant stimuli (rather than equally following threat and nonthreat stimuli) so that participants learn to attend to neutral stimuli. Using this approach, MacLeod et al. (2002) first established that attentional biases could be modified by working with participants who scored in the middle third of the distribution on the State-Trait Anxiety Inventory (STAI; Spielberger et al. 1971). They found that after attentional training away from the threat, participants reported reduced negative affect to a standardized stress manipulation. In a subsequent study, Mathews & MacLeod (2002) established that the attentional training was effective in reducing anxiety in high-STAI scorers one month before a school examination. More recently, Wadlinger & Isaacowitz (2008) extended MacLeod and colleagues' (2002) findings by demonstrating that positive attentional biases can also be trained. Moreover, participants who had been trained to attend to positive stimuli subsequently looked less at negative images during a stress induction. On the basis of these findings, researchers have begun to examine attentional training with clinical samples; thus far, however, no studies have examined whether modifying cognitive biases in depressed participants leads to improved emotion regulation.

Researchers have also begun to modify interpretive biases in anxiety with the goal of improving adaptive emotional responding. Mathews & Mackintosh (2000), for example, used ambiguous scenarios to train individuals to make either positive (nonanxious) or negative (anxious) interpretations of ambiguous text. These authors found that participants in the

negative training condition exhibited higher levels of anxiety following the training than did participants in the positive training condition, suggesting that interpretive biases play a causal role in affecting anxiety levels. Yiend et al. (2005) demonstrated that the effects of interpretive training on anxiety were still present after a 24-hour delay between the training and a subsequent test. In a related study, participants who received interpretive training using ambiguous homophones were subsequently presented four distressing television clips of real-life emergency rescue situations (Wilson et al. 2006). Participants who were trained to interpret ambiguity in a nonthreatening manner had an attenuated anxiety reaction to the subsequent video stressor. Holmes et al. (2009) found in a nonclinical sample that training positive biases using imagery helped to alleviate an induced negative mood state. Considered collectively, these results indicate that changes in interpretation biases can instigate changes in emotional responding. Again, however, no studies have yet investigated whether modifying interpretive biases in depression affects emotional responding. In a recent investigation, Tran et al. (2009) demonstrated that an interpretative bias training affected memory in a subsequent free-recall task in nondepressed participants. Participants who were trained to adopt a positive interpretation bias exhibited more positive than negative intrusions (i.e., increased "recall" of positive material that was not presented at encoding; in contrast, participants who were trained to adopt a negative interpretation bias exhibited more negative than positive intrusions. This finding is important in suggesting that interpretation and memory biases are closely related and that training interpretation in depression may be effective in modifying memory biases. Clearly, more studies are needed to examine this proposition explicitly.

Recently, investigators have begun to examine the effects of training cognitive control on emotion regulation in depression. One particularly promising study found that training dysphoric individuals to be more concrete and less overgeneral in their thinking led to a

significant reduction in depressive symptoms and rumination (Watkins et al. 2009). Likewise, Raes et al. (2009) presented preliminary findings that memory-specificity training led to changes in memory retrieval that were accompanied by changes in rumination and problem solving in depressed patients. Joormann et al. (2005, 2009e) showed that depressed participants could be trained to forget negative material by practicing active suppression and did particularly well when they were provided with a strategy of how to keep irrelevant material from entering WM by using thought substitutes. Unfortunately, these authors did not assess whether suppression training affected mood or emotional reactivity to a stressor. Siegle et al. (2007), however, 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 indicates that training in attentional control may be an effective treatment for depression (Siegle et al. 2007). In this training, patients learn to selectively attend to certain sounds while ignoring irrelevant sounds. After two weeks of training, patients exhibited greater decreases in depressive symptoms than did patients who received treatment as usual. Notably, the training consisted of short sessions (15 minutes) that used nonaffective stimuli, such as the sound of birds. This suggests that cognitive control can be improved with practice and further supports the formulation that difficulties in cognitive control are implicated in emotion dysregulation.

SUMMARY AND CONCLUSIONS

In sum, depression is characterized by a specific pattern of biased processing of emotional material that includes increased elaboration of negative information, difficulties disengaging from negative material, and deficits in cognitive control when processing negative material. In this review, we discussed how these biases may be linked to emotion dysregulation in depression and, thereby, to sustained negative affect, the

core feature of depressive episodes. Because the experience of negative mood states and negative life events is associated with the activation of mood-congruent cognitions in WM, the ability to control the contents of WM is likely to be critical in differentiating people who recover easily from negative affect from those who initiate a vicious cycle of increasingly negative ruminative thinking and deepening sad mood. Interventions focused on directly modifying cognitive biases and on increasing cognitive control hold considerable promise for improving the effectiveness of the treatment of depression.

Clearly, studies are needed that examine more explicitly and systematically the mechanisms that underlie depressive cognition and the role of cognition in the regulation of emotion. In this context, investigations integrating biological and psychological aspects of depression will be particularly important in increasing our understanding of the relation between cognitive dysfunction and depression. For example, a growing literature is beginning to elucidate the neural circuitry of emotions (e.g., Ochsner & Gross 2008) and, in particular, the brain structures implicated in depression (Cooney et al. 2007, Gotlib & Hamilton 2008). These studies have documented the involvement of the limbic system, including the amygdala, hippocampus, and parts of the anterior cingulate cortex in the experience of both emotional states and depression, as well as the dorsolateral prefrontal cortex in the regulation of emotion. Interestingly, Johnstone et al. (2007) reported that during reappraisal of emotional pictures, nondepressed individuals, but not their depressed counterparts, were characterized by increased dorsolateral prefrontal cortex activation and decreased amygdala activation that appeared to be mediated by the ventromedial prefrontal cortex, suggesting depression-associated difficulties in recruiting brain regions involved in the cognitive control of emotions. Similarly, given the importance of inhibitory functioning in depression, Eugene et al. (2009) presented study results that underscore the importance of assessing activation in the rostral anterior cingulate cortex as

depressed individuals attempt to inhibit the processing of negative stimuli, and Johnson et al. (2009) found reduced activation in medial frontal cortex in depressed participants during ambiguous self-referential processing. Although these findings are exciting and provide evidence that documents associations among inhibition, reappraisal, rumination, and emotion regulation in depression, more studies are clearly needed to increase our understanding of the brain regions and neural circuits involved in cognitive inhibition and executive functioning in depression.

The recent surge in studies of specific genes that may be implicated in depression is also becoming increasingly relevant to the study of cognitive functioning in this disorder. Several studies have now linked the short allele of a functional 5' promoter polymorphism of the serotonin transporter gene (5-HTTLPR)

to the onset of depression, particularly in the context of stressful life events (see Uher & McGuffin 2008 and Lau & Eley 2010 for recent reviews of this literature; see Risch et al. 2009 for a meta-analysis). Investigators have now begun to examine associations of 5-HTTLPR with cognitive biases in depression. Beevers et al. (2007), for example, reported a link between 5-HTTLPR and attentional bias on the dot-probe task in psychiatric inpatients; Hayden et al. (2008) similarly found 5-HTTLPR to be associated with biased recall on a self-referent encoding task in girls at risk for depression. Such investigations that integrate the examination of patterns of neural activation, genetics, and cognitive processing hold great promise to help us gain a more comprehensive understanding of how psychological and biological factors interact to affect the onset, maintenance, and course of depression.

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