Emotional Facilitation and Disruption of Memory
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Cognitive performance varies as a function of phenomena conventionally considered “emotional” in various ways. A large body of research demonstrates that, to the extent that cognition and emotion can be distinguished (see Miller, 1996, for reservations about that), emotion modulates memory, learning, attention, and executive function (e.g., Bar-Haim, Lamy, Pergamin, Bakersmans-Kranenburg, & van Ijzendoorn, 2007; Cahill & McGaugh, 1995; Davidson, 2002; Engels et al., 2007; Gray, 2004; Gray, Braver, & Raichle, 2002; Heller & Nitschke, 1997; Herrington, Koven, Heller, Miller, & Nitschke, in press; Herrington, Koven, Miller, & Heller, 2006; Herrington et al., 2005; Levin, Heller, Mohanty, Herrington, & Miller, 2007; Mohanty et al., 2005, 2007). Emotion can have an impact on various cognitive mechanisms that influence memory depending on the nature of the task, the type of emotion, and the circumstances under which the individual is engaged in the cognitive work. Furthermore, emotion can affect memory mechanisms via state conditions (e.g., anxious arousal) as well as via more chronic or trait conditions (e.g., dysthymia, posttraumatic stress disorder, avoidance temperament). These can combine to have distinct influences on memory, such as in the case where a person who is depressed and prone to anxious apprehension or worry becomes anxiously aroused (Nitschke, Heller, Palmieri, & Miller, 1999).

1. Dimensional vs. categories approaches to emotion

To understand the diverse relationships between emotion, memory, and relevant brain function, one strategy has been to explore emotion in terms of more fundamental components. Major approaches have either placed emotion in a multidimensional space or cast it as a series of categories,
either lacking a specific relational structure or employing a simple hierarchy. Dimensional models have more often been applied to brain data, including variations on the circumplex model of emotion, in which emotion is represented as two orthogonal components such as valence and arousal (Lang, Greenwald, Bradley, & Hamm, 1993; Russell, 1980). The valence dimension varies from pleasant to unpleasant, with neutral in the middle, and arousal refers to a continuum that varies from calm to excitement. Relying on converging evidence from neuropsychological, peripheral physiological, and brain hemodynamic studies as well as developing notions of brain lateralization and emotion (e.g., Davidson, 1984; Tucker, Stenslie, Roth, & Shearer, 1981), Heller (1990, 1993) and Heller, Nitschke, Etienne, & Miller (1997) proposed a neuropsychological model of emotion in which valence is associated with differential activity in anterior cortical regions (pleasantness with more left than right activity and unpleasantness with more right than left), and arousal is associated with activity in more posterior, right parietotemporal cortex. The right posterior system was hypothesized to operate distinctly from but in concert with the frontal lobe. How the interaction of these systems plays out was hypothesized to be a function of individual differences in affective style (including features of personality and psychopathology) that contribute to diverse emotional consequences for cognition (Heller, 1990). Variants of a valence/arousal approach have been proposed as well. For example, a rotation of the valence and arousal axes produces positive and negative affect dimensions (Clark & Watson, 1991), discussed below. Related conceptualizations emphasize appetitive and defensive (Lang, Bradley, & Cuthbert, 1997) or approach and avoidance motivation (Davidson & Irwin, 1999) as organizational principles for brain function in emotion (for comparative reviews, see Elliott & Thrash, 2002, and Shankman & Klein, 2003).

To date, such dimensional approaches have proven more fruitful than a categorical approach in which different emotions are presumed to be instantiated in different regions of the brain (e.g., happiness in one area, anger in another, fear in a third). Although it is indeed the case that particular brain areas play a more prominent role in particular emotions, such approaches have not had much to say about specific brain mechanisms likely to operate in such spatially discrete regions and able to differentiate emotions qualitatively. Another trend in the literature is away from the traditional relegation of neural factors in emotion to subcortical and phylogenetically old cortical structures. Thus, complementing an early emphasis on the limbic system and particularly the amygdala for emotional processing, other newer regions such as prefrontal cortex (PFC) are receiving more attention (Dolcos, LaBar, & Cabeza, 2004). Further, as tools for analyzing macrolevel interregional brain connectivity advance, the emphasis is moving to multiregional networks. This, in turn, fosters an
appreciation of emotion and cognition as thoroughly interacting or perhaps as not even distinct types of phenomena (Miller, 1996; Miller, Engels, & Herrington, 2007; Mohanty et al., 2007).

Arousing events, whether positive or negative, are remembered better than neutral events. This effect has been attributed to the “memory-modulation” hypothesis about the amygdala, in which this brain structure is thought to play a particular role in memory for emotionally arousing information (Cahill, 2000; McGaugh, Ferry, Vazdarjanova, & Roozendaal, 2000). Converging functional brain imaging studies support the hypothesis that the amygdala modulates memory storage processes involving other brain regions (Cahill et al., 1996; Canli, Zhao, Brewer, Gabrieli, & Cahill, 2000; Canli, Desmond, Zhao, & Gabrieli, 2002; Dolcos, Graham, LaBar, & Cabeza, 2003; Dolcos, Labar, & Cabeza, 2004; Hamann, Ely, Grafton, & Kilts, 1999; Mohanty et al., 2005, 2007). Since limbic regions such as the amygdala project to PFC (Barbas, 2000), it is reasonable to assume that presentation of emotional stimuli can modulate PFC activity during memory processes. Using an fMRI paradigm, Dolcos et al. (2004) found that during emotional evaluation, PFC activity showed a hemispheric asymmetry consistent with the valence hypothesis (left PFC activity greater for positive than negative pictures, right PFC greater for negative than positive pictures). In addition, dorsomedial PFC activity was sensitive to arousal, whereas ventromedial PFC activity was sensitive to positive valence. Successful encoding was enhanced by arousal in left ventrolateral and dorsolateral PFC regions, hypothesized to reflect an enhancement of strategic, semantic, and working memory processes. These results suggest that PFC regions play an important role in the evaluation of emotional stimuli and are sensitive to both valence and arousal. Using an emotion-word Stroop fMRI paradigm, Herrington et al. (2005) further supported the prediction of an association between the valence dimension and frontal laterality in an unselected sample, and Engels et al. (2007) did so in subjects selected for high or low anxiety.

2. Depression and memory

Many of the findings in the neuropsychology and cognitive neuroscience literatures bearing on whether valence affects memory to the extent emotional arousal does come from studies of psychopathology. Emotional stimulus qualities appear to enhance or impair memory depending on the nature of the task and the roles of the brain regions involved. For example, executive functions are enhanced by positive affect associated with activity in regions of the left PFC (Ashby, Isen, & Turken, 1999; Engels et al., 2007; Herrington et al., 2005). In contrast, depression has been associated
with impaired memory performance, via prefrontal deactivation with consequent impairment in the use of mnemonic strategies (Heller & Nitschke, 1997; Levin et al., 2007; Mohanty & Heller, 2002; Nitschke, Heller, Etienne, & Miller, 2004). For example, depression has been associated with impairments in autobiographical memory (Williams & Broadbent, 1986; Williams & Dritschel, 1988; Williams & Scott, 1988), episodic memory recall (Cabeza, Locantore, & Anderson, 2003), and working memory (Elliot et al., 1996). In addition, depressed subjects perform poorly on explicit memory tasks such as free recall, cued recall, and recognition. A meta-analysis of recall and recognition studies by Burt, Zembar, and Niederehe (1995) revealed a stable association between memory and depression, demonstrating that depression is linked with particular aspects of memory impairment, specifically deficits in explicit (and not implicit) memory tasks. These deficits are not explainable as secondary to reductions in motivation. For example, using a motivation-enhancing manipulation, Richards and Ruff (1989) demonstrated that motivated depressed patients performed the same on neuropsychological measures as did a nonmotivated depressed group.

In other studies, biases have been reported for valenced information, whereby depression is associated with a tendency to recall negative better than positive material (for reviews, see Blaney, 1986; Watkins, 2002). Mood induction studies in clinically depressed and nonpatient individuals have shown a bias toward recall of negative autobiographical memories (Clark & Teasdale, 1982; Williams & Scott, 1988). Other research has implicated withdrawal-related negative emotions and threat perception as factors that may play a role in negative cognitive biases (Bar-Haim et al., 2007; Heller & Nitschke, 1997; Nitschke & Heller, 2002). Heller and Nitschke (1997) proposed that executive function impairments account for a large part of the observed memory deficits in depression. Individuals with depression demonstrate difficulties in initiating cognitive strategies that enhance their ability to process and remember information (for review, see Levin et al., 2007). Prefrontal cortex, especially dorsolateral sectors (DLPFC), is frequently associated with cognitive control and executive function. A primary function of DLPFC is the representation of goals and the maintenance of context information that promotes the means to achieve these goals (Braver & Barch, 2002; Davidson, 2002; Nitschke et al., 2004). Context information might include task demands, information regarding the results of previous behavior, emotional state, or any aspect of the internal or external environment that would influence the accomplishment of the represented goals (Nitschke et al., 2004). Recruitment of DLPFC in cognitive control tasks has been found to be central to performance on various memory tasks, including working memory and episodic long-term memory (for review, see Nitschke et al., 2004). In studies examining strategy utilization in depression, Hertel and colleagues (for reviews, see Hertel, 1994, 1997, 2000) showed that memory
deficits are eliminated when strategies are provided prior to the start of the task. In line with this research, patients with DLPFC lesions showed impairments when using organizational strategies during episodic memory tasks but showed improvement when instructed in the use of such strategies (Gershberg & Shimamura, 1995; Incisa della Rocchetta & Milner, 1993).

Given that depressed individuals can perform cognitively demanding tasks in the presence of explicit instructions or task constraints, memory impairments may not be the result of reduced attentional resources but rather an impairment in the deployment of these resources. Hertel (1994) proposed that attentional resources are sufficient in patients with depression but that the initiative to control these resources is missing. This diminished initiative to attend can be manifested in underrecruitment of PFC. In an EEG study examining brain mechanisms accompanying the initiative deficit, Nitschke et al. (2004) found that bilateral activity recorded over PFC during a preparatory period immediately preceding a sad narrative was associated with better recall performance in controls but not in a depressed group. Depressed participants also showed a negative memory bias. Hyperactivity in right PFC was observed during exposure to the sad narrative and was associated with improved recognition of words in that narrative.

In depressed individuals, poor performance on memory tasks may therefore be explained in part by a failure to recruit PFC in preparation for information processing (Nitschke et al., 2004). This impairment may be associated with a lack of initiative in allocating attentional resources for performance on cognitive tasks (Hertel & Harden, 1990, Hertel & Rude, 1991) or with problems with sustained attention (Burt et al., 1995). Nitschke et al.’s (2004) findings support Hertel’s (1994, 2000) model of memory performance in depression and highlight the importance of distinguishing different processes influencing memory performance and cognitive bias. Specifically, the failure to recruit PFC in preparation for information processing may result in poorer performance on memory tasks. Recruiting right PFC under conditions of negative emotion or threat may serve to enhance memory performance (Heller & Nitschke, 1997).

3. Depression/anxiety comorbidity

An important issue to consider in understanding the impact of emotion on memory and other cognitive processes is the considerable but variable comorbidity of depression with anxiety. The majority of studies of either depression or anxiety have not taken this comorbidity into consideration, nor have they distinguished between types of anxiety such as anxious apprehension and anxious arousal. Heller, Miller, and colleagues
Engels et al. (2007); Keller et al. (2000); Levin et al. (2007); Nitschke et al. (1999) have demonstrated that different types of anxiety and depression are associated with distinct patterns of regional brain activity using neuropsychological, EEG, and fMRI methods, as well as having distinct psychometric relationships.

Crucially, unaccounted-for comorbidity may undermine the interpretation of results in much of the literature on emotion–cognition relationships. For example, the presence of comorbid anxiety has been shown to cancel out the effects of depression on neuropsychological performance (Heller et al., 1995) or to have both additive and nonadditive effects (Keller et al., 2000). In addition, the literature on brain activity in anxiety and depression during cognition is inconsistent. Heller and Nitschke (1998) proposed that these discrepancies can be explained in terms of the differing subtypes of anxiety and depression represented in different studies. Thus, a failure to separate anxiety and depression, either experimentally or statistically, may explain a large portion of the variability in reported executive function impairments in depression (Levin et al., 2007).

Given the substantial conceptual and epidemiological overlap of anxiety and depression, researchers and clinicians have long desired to understand the relationship between them. An influential line of research has focused on how they differ in their affective structure. The tripartite model proposed by Clark and Watson (1991), Mineka, Watson, & Clark (1998), Watson, Clark, et al. (1995), Watson, Weber, et al. (1995), and Watson, Weise, Vaidya, & Tellegen (1999) includes a shared general distress factor characterized by high levels of negative affect that is common to both anxiety and depression. A separate, positive affect/anhedonia factor is characterized by low levels of pleasurable engagement with the environment and is specific to depression. Lastly, arousal characterizes anxiety and not depression. Heller, Miller, and colleagues have argued that these components of depression and anxiety are implemented in different brain regions (e.g., Heller & Nitschke, 1997, 1998; Levin et al., 2007). Furthermore, they have emphasized that the type of anxiety described by Watson and colleagues (anxious arousal) should be differentiated from anxious apprehension, or worry, in psychological and neuropsychological terms. To the degree that one of these types of anxiety is associated with activity in a particular brain region, it is possible that its presence could disrupt (either enhance or impair) ongoing cognitive processing typically implemented or influenced by that brain region. For example, reduced brain activity in PFC associated with depression could account for impaired performance on various executive function tasks, reviewed above. In contrast, anxious arousal and anxious apprehension could have other influences on cognitive processing that would manifest in distinct patterns of brain activity.
4. Anxiety and memory

Supporting the proposals of Heller, Miller, and colleagues, Shackman et al. (2006) demonstrated that threat-induced anxiety (essentially anxious arousal) selectively disrupts spatial but not verbal working memory performance. The tasks used were verbal (letter identity) and spatial (location) variants of a three-item N-back task. Threat of shock served as the affect induction procedure. The authors concluded that threat-induced anxiety (verified via EMG) disrupted spatial performance (indexed by accuracy) and not verbal performance because right PFC resources were engaged in anxiety-related processing and hence were less available to support working memory performance. Similar results were obtained in a second experiment testing individuals with high scores on a self-report measure presumed to index the predisposition to react more strongly to perceived threat. These findings are consistent with lateralization for visuospatial working memory in prefrontal and parietal cortices, supporting the arousal portion of the model of Heller, Miller, and colleagues.

Supporting the model’s proposal of valence lateralization in PFC, with concomitant effects on cognitive processing, Gray (2001) found a double dissociation between the effects of inducing positively and negatively valenced mood on spatial and verbal working memory. Specifically, performance on tasks relying on verbal working memory, for which there is considerable evidence of left prefrontal specificity, was enhanced by the induction of positive mood and impaired by the induction of negative mood. In contrast, performance on tasks relying on spatial working memory, for which there is considerable evidence of right prefrontal specificity, was enhanced by the induction of negative mood and impaired by the induction of positive mood, supporting the valence portion of the model of Heller, Miller, and colleagues. Some aspects of Gray’s results are not consistent with those of Shackman et al. (see Shackman et al., 2006, for extensive discussion of these discrepancies) but nevertheless serve to demonstrate differential effects of affect on lateralized cognitive processes.

In contrast to anxious arousal, anxious apprehension would be expected to influence tasks associated with the left-hemisphere regions hypothesized to be involved in this type of anxiety (Engels et al., 2007; Nitschke, Heller, & Miller, 2000). Heller, Miller, and colleagues have suggested that many of the general decrements in cognitive processing associated with anxiety (Eysenck & Calvo, 1992; Eysenck, Derakshan, Santos, & Calvo, 2007) can be attributed to the effects of anxious apprehension on the availability of left-hemisphere processes that support attention, verbal rehearsal, and verbal working memory (for reviews, see Nitschke & Heller, 1998; Nitschke et al., 2000).
A particular type of impact on cognition believed to result from anxiety is cognitive bias. Although memory biases are more common in depressed individuals, attentional biases are observed in anxious individuals (Bar-Haim et al., 2007). Anxiety impairs performance on many tasks, particularly when they are difficult or must be performed under stressful conditions. Most theorists agree that such deficits in performance on tasks that require high attentional or short-term memory demands can be attributed to the interference of worrisome thoughts with attention to task-relevant information (e.g., McNally, 1998; Sarason, 1988). Reduced recruitment of cognitive control mechanisms may play a role in anxiety-driven performance impairments. For example, participants with high anxiety levels showed reduced recruitment of DLPFC during threat-related distractors in an emotional Stroop task (Mathews & Mackintosh, 1998). High anxiety is also associated with reduced rostral anterior cingulate activity, a region of the brain associated with the assessment of emotionally salient information (Bush, Luu, & Posner, 2000; Engels et al., 2007; Mohanty et al., 2007) and with reduced recruitment of lateral PFC when the expectation of threatening distractors is established (Bishop, Duncan, Brett, & Lawrence, 2004). Thus, cognitive control mechanisms that are required to maintain ongoing task processing are diminished in anxiety during the presence of threat-related distractors.

Cognitive biases have also been observed to affect the interpretation of information as well as the ability to remember this information in anxious individuals (McNally, 1998; Mineka et al., 1998). Anxious individuals have demonstrated an increased likelihood of interpreting ambiguous information in a negative manner across multiple paradigms (Mineka et al., 1998). For example, anxious individuals are likely to interpret ambiguous homophones (e.g., die/dye, pain/pane; Mathews, Richards, & Eysenck, 1989) and sentences such as “The doctor examined Little Emma’s growth” (Eysenck, Mogg, May, Richards, & Mathews, 1991) in a more threatening manner than do controls.

Evidence also suggests that anxiety disorders are accompanied by enhanced memory for negative and threatening information (McNally, 1998). Intrusive memories are a common symptom in anxiety disorders. Some individuals with posttraumatic stress disorder are plagued by horrific memories reflected in nightmares, intrusive thoughts, and flashbacks. Individuals with panic disorder frequently experience fear of impending heart attack, insanity, and death, possibly fueled by memories of their first or worst episode. Those with obsessive–compulsive disorder may experience recurrent obsessions about harm, whereas those who suffer from generalized anxiety disorder experience uncontrollable worry about looming threats (Barlow, 2001; Coles & Heimberg, 2002; McNally, 1998). These
phenomenological observations suggest that memory in anxiety disorders is characterized by enhanced access to threat-related information (Coles & Heimberg, 2002; Lang, 1979; McNally, 1998).

Such biases in attention and memory may be linked to a right-hemisphere system differentially involved in responding to threat (Compston, Heller, Banich, Palmieri, & Miller, 2000; Nitschke et al., 2000). The right hemisphere is particularly suited to evaluate emotional stimuli (e.g., Borod et al., 1998) and has also been identified as important for cognitive processes such as scanning both sides of space, processing spatial relationships, and maintaining vigilance. Under normal circumstances, an integration of functions across prefrontal, parietal, and temporal regions of the right hemisphere would confer a highly adaptive capacity to monitor the environment for emotional stimuli and modulate responses to these stimuli (Nitschke & Heller, 2002; Nitschke et al., 2000). In cases of psychopathology, abnormal emotional responding could be associated with abnormal patterns of brain activity in these and other regions as well as with altered (either enhanced or impaired) attention, learning, and memory.

5. Conclusion

This brief review emphasizes a few aspects of a substantial and growing literature on relationships between the emotional qualities of stimuli, the emotion-related processing they prompt, and their effects on memory. These effects are prominent in, but not confined to, some types of psychopathology. Indeed, it should be understood that emotion is an ongoing modulator of memory even in healthy or “normal” individuals, just as, conversely, memory feeds and alters emotion. Thus, both emotion phenomena and memory phenomena are better understood if each is better understood. A particularly challenging frontier in this area of research is determining causal mechanisms, not only between emotion and memory as psychological processes, or between the brain processes that implement emotion and memory, but between the psychological processes and biological processes. Choice of language in this literature often implies causal relationships, either psychology driving biology or biology driving psychology, but to date there is no articulated mechanism by which such causal relationships could arise (Miller, 1996; Miller et al., 2007). Neither dismissing nor embracing dualism as a type of relationship between psychological and biological events provides the causal mechanisms, if any, that link them. The present review provides a sampling of observed associations between these phenomena, awaiting a mechanistic account.
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References


