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Emotional memory function, personality structure and psychopathology: A neural system approach to the identification of vulnerability markers[☆]

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Abstract

It is well established that emotional events are ingrained stronger into memory relative to neutral events. Facilitated emotional memory is highly variable between individuals within the normal population and is particularly exacerbated in those diagnosed with mood and anxiety disorders. In order to elucidate how variation of enhanced emotional memory within the normal population may manifest into psychopathological states, we explored the convergence between studies investigating the neural systems engaged in emotional memory facilitation and studies investigating how these systems differ from person to person. Converging evidence highlights the roles of three neural systems (1. Amygdala function and attention, 2. Neuroendocrine function, 3. Interactive effects with mood) that all govern emotional memory facilitation and are highly variable between individuals as a function of personality. We applied this neural system approach to models of vulnerability of three forms of psychopathology that are particularly characterized by atypical emotional memory function (depression, generalized anxiety disorder and post-traumatic stress disorder). This application suggests that the incorporation of known vulnerability markers across psychological, neuroimaging and neuroendocrinological domains is cardinal to how susceptibility is conceptualized and assessed in these disorders.

Keywords

Emotional memory; Amygdala; Neuroendocrine; Mood-congruent; Personality; fMRI; Depression; Anxiety; PTSD

1. Introduction

“What you see and hear depends a great deal on where you are standing; it also depends on what sort of person you are” — C.S. Lewis

We commonly remember the emotional highs and lows of our lives more vividly than the mundane events in-between. However, the emotional salience of these experiences also depends on unique characteristics that define each of us as individuals, such as subjective

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appraisal and sensitivity to particular mood states. Indeed, a substantial body of research has documented that emotionally arousing memories are better remembered than less emotionally arousing memories (Bradley et al., 1992; Kleinsmith and Kaplan, 1963; Libkuman et al., 2004), and that this process is highly variable between person to person (Rusting, 1999). Neuroimaging studies have begun to identify the neural systems that are engaged during the enhancement of emotional memories in humans (Cahill et al., 1996; Canli et al., 2000; Dolan, 2002; Hamann, 2001; Phelps, 2004) and have also identified how individual differences such as in personality structure correspond to variation of this process (Hamann and Canli, 2004). Additionally, emotional memory facilitation is a critical feature of several forms of psychopathology and variation of personality structure within the normal population is associated with the vulnerability to developing these types of disorders. By investigating how the neural systems engaged in emotional memory facilitation vary between individuals, we may develop a clearer understanding of what constitutes vulnerability to developing disorders that are characterized by emotional memory dysfunctions. In the current review, we aim to explore the convergence between studies investigating the neural systems engaged in emotional memory facilitation and studies investigating how these neural systems differ from person to person as a function of a known systematic variability factor that is associated with affective processing (eg., personality). We will then utilize this converging evidence in order to develop a model of how variation of these neural systems may serve as vulnerability markers for developing psychopathological states in three disorders (depression, generalized anxiety disorder and PTSD).

2. Neural systems of emotional memory and personality structure

Advancements in neuroscientific techniques have yielded insights into the systems that govern how emotional memories are processed as well as to how changes in these systems are associated with individual differences such as with personality or psychopathological states. Research on emotional memory has used a wide range of stimuli, tasks, and measurement tools in order to elucidate the role of a particular set of neural systems integral to how emotional memories are processed. These systems include the amygdala as a modulator of attention (Phelps and Anderson, 1997; Phelps, 2004), neuroendocrine function (Buchanan and Lovallo, 2001; Cahill et al., 1994) and limbic system function as a moderator of mood states (Lewis et al., 2005; Ramel et al., 2007). Interestingly, the function of these systems is also highly variable between people of differing personality structures. This convergence suggests that the variation of these systems underlies individual differences in emotional memory facilitation. In the following section, we will explore how three psychological/neuronal systems (a. Attention and amygdala function, b. Neuroendocrine function, c. Interactive effects with mood) influence the facilitation of emotional memories and vary as function of personality. We will now turn to a more detailed review of how these systems are fundamental to emotional memory function and individual differences.

2.1. Attention and amygdala function

Similarities exist between studies of emotional memory encoding and studies of attentional systems associated with personality (see Box 1). These studies suggest that attention modulates how emotional memories are encoded (Anderson and Phelps, 2001; Arntz et al., 2005; Keil and Ihssen, 2004; Phelps, 2004) and that attention to emotional stimuli differs as a function of personality (Amin et al., 2004; Derryberry and Reed, 1994). The neural networks engaged in these two processes likely influence how a particular memory bias for emotional information is developed and maintained in an individual. One structure that has been identified to play a prominent role in emotional memory, attention and to be associated with individual differences in personality is the amygdala.

A potential mechanism that may underlie why emotional events are better remembered relative to neutral ones is that they receive more attention during encoding (Phelps, 2004). This is supported by attentional blink studies demonstrating that when stimuli are presented for very short periods of time, emotional items are identified more frequently compared to neutral items (Keil and Ihssen, 2004; Öhman et al., 2001). This difference in attention likely corresponds to a deeper level of encoding which is associated with better subsequent memory (Martin, 1999). Further evidence for the role of attention in the enhancement of emotional memories is gleaned from neuroimaging studies during the stage of encoding.

Across a range of experimental paradigms and analysis techniques, a substantial amount of evidence supports the role of the amygdala during the encoding of emotional memories (Cahill et al., 1995; Phelps and Anderson, 1997). For example, by taking an individual differences perspective, Cahill et al. (1996) related between-subject brain activity in response to emotional stimuli with subsequent memory. This study identified that each subject's amygdala activity when encoding film clips was predictive of recall for emotionally arousing film clips but not for neutral film clips. Subsequent studies have addressed the valence and temporal specificity of this finding (Canli et al., 1999; Hamann et al., 1999). Hamann et al. (1999) identified that amygdala activity during encoding is predictive of recognition of aversive as well as positive stimuli and Canli et al. (1999) identified that amygdala activity during encoding is associated with each subject's recognition score of emotional pictures an average of 211 days following encoding. Interestingly, recent studies have also documented how the amygdala is involved in heightened attention to emotional stimuli.

Box 1

Converging evidence suggests that increased amygdala activity during attending/encoding to emotional stimuli corresponds to facilitated retention of memory (left) and that amygdala activity varies as function of personality when attending to emotional stimuli (right). The use of neuroimaging techniques demonstrates that each subject's amygdala reactivity during the viewing of emotional film clips (A) or pictures (B) is predictive of subsequent memory. Recent neuroimaging studies have also demonstrated that each subjects' Extraversion (C) or Neuroticism (D) score is predictive of amygdala activity when attending to emotional stimuli. This between-subject variation of amygdala reactivity likely governs variability in the extent to which emotional memories are facilitated in some but not others.

A patient study supports a critical role for the amygdala in the attentional blink paradigm (Anderson and Phelps, 2001). This study indicated that normal subjects displayed increased perception of emotional compared to neutral target words, while patients with amygdala damage did not display this effect. The role of the amygdala for increased emotional attention is further supported by our understanding of the functional anatomy of this structure (Aggleton, 2000). The amygdala has reciprocal connections to several attentional modulating regions (Swanson and Petrovich, 1998) and in particular the visual cortex (Amaral et al., 2003). Neuroimaging research using functional connectivity analyses has identified that the amygdala is more correlated with the occipital cortex during the rating of unpleasant words relative to neutral words (Tabert et al., 2001). Other research has found that the amygdala differentially modulates the activity in the extrastriate cortex as a function of the emotional valence of faces (Morris et al., 1998). Combined, these studies indicate that a primary role of the amygdala is to modulate sensory and attentional resources when processing emotional stimuli (Whalen, 1998; Whalen et al., 1998). Individual differences in attention may be a behavioral manifestation of variability of the extent that the amygdala modulates these recourses between people.

Personality is associated with individual differences in attention to emotionally valenced stimuli (Amin et al., 2004; Derryberry and Reed, 1994; Haas et al., 2006; Reed and Derryberry, 1995). For example, those scoring higher on extraversion exhibit greater attention towards positively relative to negatively valenced emotional stimuli (Derryberry and Reed, 1994) and longer reaction times when responding to positive relative to neutral words (Haas et al., 2006) compared to those scoring lower on extraversion. Additionally, other studies have identified attentional biases to be associated with other traits such as neuroticism (Reed and Derryberry, 1995), harm-avoidance (Most et al., 2006) as well as the behavioral activating system (BAS) (Putman et al., 2004).

Recently, the advancement of neuroimaging techniques has provided the ability to investigate the relationship between personality and changes in brain activity when attending to emotional stimuli. Changes in amygdala activity during emotional processing are associated with several personality traits. For example, using a between-subjects correlational design, Canli et al. (2001) identified that higher scores of extraversion are associated with greater activity in the right amygdala when viewing positive relative to negative pictures. Subsequent research has identified a relationship to exist between extraversion and amygdala activity when processing emotional facial expressions (Canli et al., 2002b), neuroticism and amygdala activity when processing emotionally conflicting stimuli (Haas et al., 2007b) and harm-avoidance and

amygdala activity when processing emotionally distracting stimuli (Most et al., 2006). The relationship between personality and amygdala activity during attention to emotional stimuli is likely an underlying component of how personality is associated with emotional memory biases.

Studies have documented how personality is associated with particular emotional memory biases. For example, higher scores of neuroticism are associated with a bias towards remembering emotionally negative information (Bradley and Mogg, 1994; Lishman, 1972; Lishman, 1974; Mayo, 1983; Martin et al., 1983; Young and Martin, 1981) and higher scores of extraversion are associated with a bias towards remembering emotionally positive information (Lishman, 1972; Mayo, 1983; Rusting, 1999). Additionally, biases of emotional memory have also been identified to be associated with trait anxiety (Nugent and Mineka, 1994; Reidy and Richards, 1997a,b), sensitivity (Hock and Krohne, 2004) and self-esteem (Smith and Petty, 1995).

Individual differences in emotional memory biases linked to personality are likely rooted in an attentional network that is primarily driven by amygdala reactivity during the encoding of emotional stimuli (Fig. 1A). Research has shown that attention modulates how emotional memories are encoded (Arntz et al., 2005) and that the amygdala is engaged when attending to (Morris et al., 1998) and encoding (Cahill et al., 1996; Canli et al., 1999; Hamann et al., 1999) emotional stimuli. Additionally, individual differences in personality are associated with variations in attention to emotional stimuli (Reed and Derryberry, 1995), emotional memory biases (Rusting, 1999) and amygdala activity when processing emotional stimuli (Canli, 2004). During the initial stages of attention and encoding, heightened levels of amygdala activity may correspond to the recruitment of greater amounts of attentional resources and therefore facilitate the formation of stronger and more long lasting memories. Given that particular personality profiles such as extraversion and neuroticism are linked to heightened levels of amygdala activity when attending to positive and negative stimuli respectively, this greater initial activity may correspond to the specific emotional memory biases associated with these personality traits. Specifically in the case of extraversion, those who score higher on this trait exhibit greater attention towards positive emotional stimuli and greater amygdala activity during this process. Highly extraverted individuals may remember more positive emotional information because this information received more attention and was paired with greater amygdala reactivity during encoding. Similar patterns are likely the case with neuroticism and amygdala reactivity to negative stimuli and trait anxiety and amygdala reactivity to fearful stimuli.

2.2. Neuroendocrine function

Studies investigating the mechanisms underlying how memories are consolidated over time have demonstrated that several post-learning treatments such as electrical stimulation, protein synthesis inhibitors and peripheral and central drug administration can effect later memory (McGaugh, 2000; McIntyre et al., 2003; Nadel and Land, 2000; Pare, 2003). In particular, recent studies have investigated the neuroendocrine system and documented that manipulation and/or between-subject variation of stress hormones such as epinephrine (Cahill et al., 1994), norepinephrine (Harmer et al., 2003) and cortisol (Buchanan and Lovallo, 2001) affect the retention of emotional memories. Interestingly, variation of these stress hormones has also been identified to be associated with individual differences in personality. The following section will explore how research on neuroendocrine function, emotional memory and personality converge.

Stress hormones such as epinephrine (Cahill et al., 1994), norepinephrine (Harmer et al., 2003) and cortisol (Buchanan and Lovallo, 2001; Het et al., 2005) influence the consolidation of emotional memories. In particular, blockage of the action of epinephrine on Beta-adrenergic

receptors diminishes the consolidation of emotional memories. This is supported by a study by Cahill et al. (1994) that identified that individuals receiving a Beta-adrenergic receptor blocker (propranolol) prior to encoding remembered less emotional items (relative to neutral) compared to the placebo group. Further investigations have demonstrated that this effect is dose and time dependent (van Stegeren et al., 2002) and acts through the central but not the peripheral nervous system (van Stegeren et al., 1998).

Blockage of Beta-adrenergic receptors is associated with changes in amygdala activity during the encoding of emotional memory. Strange and Dolan (2004) identified that individuals receiving propranolol did not display any emotional memory bias and did not display any increase in amygdala activity during encoding (relative to a placebo group). Specifically, epinephrine is thought to act primarily upon Beta-adrenergic receptors in the basolateral division of amygdala when consolidating emotional memories (McGaugh, 2004; McIntyre et al., 2003). Combined, these findings indicate that the action epinephrine has on Beta-adrenergic receptors is critical to how emotional memories are formed and consolidated.

Cortisol variation, as measured at baseline or in response to stress, influences the consolidation of emotional memories (Buchanan and Lovallo, 2001; Kuhlmann and Wolf, 2006). Buchanan and Lovallo (2001) identified a relationship to exist between cortisol and emotional memory function by comparing emotional memory retention between groups that either received an oral administration of cortisol or placebo prior to encoding emotional and neutral stimuli. Subjects that received cortisol remembered a greater amount of emotional relative to neutral stimuli compared to the placebo group. This enhancement of emotional memory has also been identified to be influenced by manipulating cortisol levels by means of post-learning stress (Cahill et al., 2003) and to vary as a function of individual differences in cortisol reactivity to stress (Buchanan and Tranel, 2008; Buchanan et al., 2006). These studies support a critical role of cortisol in enhancing emotional memories and indicate that variations in the stress response between individuals may underlie how emotional memory biases are formed and maintained.

Interestingly, variability of the neuroendocrine system either measured at baseline or in response to stress is associated with individual differences in personality (Pruessner et al., 1997, 2004). For example, higher scores of extraversion and agreeableness correspond to decreased epinephrine levels (Miller et al., 1999). Higher scores of harm-avoidance (Gerra et al., 2000) and novelty-seeking (Gerra et al., 1999) are associated with increased norepinephrine levels and higher scores of extraversion and lower scores of neuroticism are associated with increased plasma levels of cortisol (Leblanc and Ducharme, 2005). These between-subject variations are thought to be a biological marker of sensitivity to stress. Recent studies have supported this claim by assessing the relationship between personality and cortisol response to stress. These studies have identified that traits such as openness, extraversion and neuroticism correspond to the amount of change between cortisol levels measured at baseline and following a stressor (Mangold and Wand, 2006; Oswald et al., 2006).

The convergence between studies identifying a relationship between neuroendocrine function and emotional memory and studies identifying a relationship between neuroendocrine function and personality traits suggests that individual differences in these hormones may govern how emotional memory biases are manifested (Fig. 1B). In the case of epinephrine, blockage of its action of Beta-adrenergic receptors results in a relative blunting of emotional memory formation. Individuals that exhibit lower baseline levels of epinephrine may remember less emotional information because of less action of existing epinephrine on Beta-adrenergic receptors. The existence of this mechanism must be considered as rather speculative at this time due to the inconsistency between studies that have manipulated and/or assessed epinephrine levels. Specifically, studies that have manipulated epinephrine action in order to investigate emotional memory function have done so through the central nervous system and

studies that have investigated between-subject variation of epinephrine have done so by measuring levels in the periphery.

The development and maintenance of emotional memory biases are likely linked to variation of baseline and stress-induced levels of cortisol. Higher levels of cortisol correspond to increased retention of emotional memories and personality traits that are linked to emotional memory biases (ex. extraversion and neuroticism) correspond to variation in cortisol levels. A particular bias towards emotional memory may develop as a result of the magnitude by which the adrenal gland produces cortisol and/or the sensitivity of existing receptors responsive to cortisol. Additionally, the induction of stress modulates how cortisol influences emotional memory as well as affects the relationship between cortisol and personality. Greater amounts of stress corresponds to increased emotional memory retention. Certain personality traits that are particularly characterized as being sensitive to stress may be paired with specific biases towards emotional information in part due to how stress is subjectively appraised and is manifested by cortisol response.

2.3. Interactive effects with mood

Changes in mood states are associated with the processing of emotional memories and are associated with individual differences in personality. In particular, variations in mood states facilitate the retrieval of emotional memories when the valence of the current mood state is consistent with the valence of the emotional memory being retrieved (mood-congruent memory facilitation). This facilitative effect of mood on emotional memory retrieval has been identified to correspond to changes in activity within the limbic system (Buchanan, 2007; Lewis et al., 2005; Ramel et al., 2007). Interestingly, these relatively transient variations of mood states interact with more stable variation of personality traits. The interaction between states and traits has been identified to influence the retrieval of emotional memories and to also correspond to changes in activity within the limbic system. The convergence between these findings indicates that a primary function of the limbic system may be to utilize emotional state information in order to guide the retrieval of memories of corresponding valence. The between-person variation in the affinity towards experiencing certain mood states likely relates to the development and maintenance of biases towards particular emotional memories. The following section will explore how research investigating the effect of mood on emotional memory retrieval and the interaction of mood and personality converge.

Variation of positive and negative moods corresponds to the facilitated retrieval of positively and negatively valenced emotional memories, respectively (Matt et al., 1992; Rusting, 1998). The facilitation of emotional memory retrieval has been identified to occur following the experimental induction of mood states (Brown and Taylor, 1986) as well as during the comparison of between-subject variation in natural mood states (Mayer et al., 1995). This research suggests that variation in mood is strongly linked to the likelihood that a particular emotional memory will be retrieved either through active or automatic recall (Blaney, 1986; Rusting, 1998). Specifically, mood states are thought to facilitate retrieval by activating similar networks that were engaged during the stage of encoding (Rusting and DeHart, 2000). This is supported by neuroimaging research investigating patterns of brain activity associated with mood fluctuation and during the retrieval of emotional memories.

Activity within the amygdala and the anterior cingulate is altered by mood induction and also corresponds to between-subject variation in natural mood states (Mitterschiffthaler et al., 2007; Wang et al., 2006). For example, Wang et al. (2006) identified that amygdala and anterior cingulate activity in response to sad stimuli is enhanced following the induction of sad mood states. Enhanced activity in these regions also occurs in response to happy mood induction (Habel et al., 2005) and by the induction of mood in response to music (Mitterschiffthaler et al., 2007). Additionally, a study from our laboratory identified that between-subject variation

of self-reported negative mood is associated with changes in anterior cingulate activity when responding to negatively valenced emotional words (Canli et al., 2004). Combined, these studies indicate that fluctuation of particular mood states correspond to changes in activity within limbic structures such as the amygdala and anterior cingulate.

The enhancement of brain activity in response to mood induction is associated with the enhancement of emotional memories. As evinced by a study by Lewis et al. (2005), enhanced anterior cingulate activity in response to positive mood induction facilitates the retrieval of positive emotional memories. Additionally, in a sample of subjects at high risk for developing depression, Ramel et al. (2007) demonstrated that enhanced amygdala activity in response to sad mood induction facilitates the retrieval of negative self-referent information. These studies indicate that individual differences in the sensitivity to experiencing particular mood states is associated with the extent that regions subserving emotional memory processes facilitate retrieval.

Personality is associated with individual differences in the sensitivity to experiencing particular mood states. Specifically, higher scores of personality traits such as extraversion (Costa and McCrae, 1980; Lucas and Fujita, 2000) and agreeableness (DeNeve and Cooper, 1998) render individuals more likely to experiencing positive mood states, while personality traits such as neuroticism (Suls and Martin, 2005) and harm-avoidance (Stewart et al., 2005) render individuals more likely to experiencing negative mood states. The variation of mood linked to personality is thought to influence several cognitive, emotional and behavioral tendencies including the bias towards particular emotional memories (Rusting, 1998).

Studies have identified that mood states and personality traits interact with one another in order to facilitate the retrieval of emotional memories. Rusting (1999) identified an interactive effect between extraversion and positive mood states facilitating the retrieval of positively valenced emotional words and an interactive effect between neuroticism and negative mood states facilitating the retrieval of negatively valenced emotional words. Additional research has identified interactive effects between self-esteem and negative mood facilitating the retrieval of negative autobiographical memories (Smith and Petty, 1995). These findings suggest that the interaction between mood and personality corresponds to the engagement of a neural network that facilitates the retrieval of emotional memories.

Recently, neuroimaging research has identified that the interaction between mood and personality corresponds to changes in limbic system activation. Specifically, the interaction between extraversion and positive moods as well as the interaction between neuroticism and negative mood is associated with anterior cingulate activation (Canli et al., 2004; Keightley et al., 2003). Additionally, based on findings relating changes in amygdala activity with both mood (Wang et al., 2006) and with personality (Canli et al., 2002b; Haas et al., 2007a,b) this structure is also likely engaged in this interaction.

The convergence between studies investigating interactive effects between mood, personality and emotional memory suggests that the anterior cingulate and amygdala govern how people vary in the likelihood of experiencing particular mood states which therefore facilitates the tendency towards mood-congruent emotional memory retrieval (Fig. 1C). For example, highly neurotic individuals experience increased levels of negative mood. During these states of heightened negative mood, the likelihood of retrieving a negatively valenced emotional memory is enhanced. Clearly, this process has the characteristics that may snowball to result in increases in both the experience of negative mood states and the retrieval of negative emotional memories. Specifically, greater amounts of negative mood that are associated with neuroticism correspond to increased negative emotional memory retrieval. This increased retrieval of negative emotional memories may boost existing levels of negative mood and

therefore further facilitate the likelihood of retrieving additional negative emotional memories (exacerbating the entire process). In the following section of this review, we will explore how interactions such as this may contribute to the vulnerability and development of psychopathology.

3. Emotional memory systems, personality and psychopathological vulnerability

A cardinal feature of several forms of psychopathology is the bias towards perseverating over (usually negative) emotional experiences. For example, depressed individuals are characterized as ruminating over their failures and short-comings, anxious individuals dwell on fearful or worrisome experiences, and those with post-traumatic stress disorder (PTSD) cannot effectively block out negative trauma-related memories. Each of these disorders has been associated with individual differences in personality and has recently been investigated utilizing neuroscientific techniques such as those described in the previous section. In the following section, we will explore how the three neural systems described above may relate to the vulnerability and development of three forms of psychopathology: depression, generalized anxiety disorder and PTSD.

3.1. Depression

A bias towards remembering particularly negative information and forgetting positive information is associated with the diagnosis of major depressive disorder (MDD). The diagnosis of MDD is particularly characterized by the tendency towards remembering negative information that is self-relevant and is associated with the current depressive state (Matt et al., 1992). Additionally, research has identified emotional memory biases linked with depression using both explicit (Bradley et al., 1995) and implicit (Watkins et al., 1996) tasks and in samples such as in adolescents (Neshat-Doost et al., 1998) and in recovered clinical depressives (Bradley and Mathews, 1988). Clearly, cognitive impairments such as the tendency towards memories of primarily negative valence are a cardinal symptom that exacerbates a host of clinical features in MDD (Castaneda et al., 2007).

Individual differences in personality are associated with both depressive traits and the diagnosis of MDD. Higher scores of neuroticism correspond to an increased likelihood of experiencing depressive symptoms such as rumination (Bagby and Parker, 2001; Nolan et al., 1998; Roberts et al., 1998) and sad mood states (Stewart et al., 2005). Neuroticism is composed of several sub-facets, one of which is termed depression (N1) and specifically maps onto behavioral and cognitive characteristics associated with this disorder (Bagby et al., 1997; Costa and McCrae, 1992). Studies have investigated personality structure in individuals with a current diagnosis of MDD and have identified that those diagnosed with MDD exhibit higher scores of neuroticism and lower scores of both extraversion and agreeableness relative to those with no diagnosis (Bagby et al., 1997; Cuijpers et al., 2005; del Barrio et al., 1997; van Straten et al., 2007). The systems that govern emotional memory biases associated with personality may influence the relative susceptibility of developing depression and may foster the experience of emotional memory related symptoms in this disorder.

A clear case for how personality and clinical research converge is evinced from the example of neuroticism and depression. Highly neurotic individuals are at increased risk for developing depression (Durrett and Trull, 2005), though not all highly neurotic individuals eventually experience any depressive symptoms. A critical question is what differentiates the highly neurotic individuals who do develop depression from those who do not. One way to address this question is to identify converging patterns between at risk individuals and those that ultimately develop the disorder.

One mechanism that is common to both highly neurotic individuals and currently depressed individuals is the tendency towards attending to negative emotional stimuli (paired with increased amygdala activity). Depressed subjects exhibit greater amounts of attention towards negative stimuli and exhibit greater amounts of amygdala activity when attending to negatively valenced stimuli such as words, faces or complex pictures (Leppanen, 2006). Based on research investigating this process in non-clinical samples as well as how this process is associated with non-clinical variation in personality, this tendency may serve as a behavioral and biological marker for vulnerability. This phenotypical profile may elucidate the differentiation between highly neurotic individuals that are at increased risk for developing depression and those that are not. A highly neurotic individual that also exhibits particularly heightened attention towards negative stimuli and greater amygdala reactivity during this process is likely at greater risk than a highly neurotic individual who exhibits a less pronounced pattern of this profile.

Another potential mechanism associated with the vulnerability to developing depression is gleaned from research investigating the relationship between neuroendocrine function, personality, emotional memory and MDD diagnosis. The convergence between these studies indicates that both non-clinical variation in personality and the diagnosis of depression are associated with changes in baseline and stress-induced levels of cortisol. Additionally, between-subject variations, as well as experimentally induced levels of cortisol, influence the retention of negative emotional memories, a primary symptom of MDD. Regarding depression, a large body of research has associated relative hypercortisolemia with MDD (Burke et al., 2005; Parker et al., 2003; Plotsky et al., 1998) and identified that cortisol variation is associated with personality traits such as extraversion and neuroticism which are risk factors for developing this disorder (Leblanc and Ducharme, 2005; Mangold and Wand, 2006; Oswald et al., 2006). This convergence implicates HPA (hypothalamic–pituitary–adrenal) axis function as a moderator of non-clinical variation of emotional memory function and as a potential biological vulnerability marker for MDD and emotional memory related symptoms in this disorder.

A primary symptom associated with MDD is the experience of negative, particularly sad, mood states and the tendency to ruminate over negative experiences (Conway et al., 2000; Roberts et al., 1998). Non-clinical variation of personality is also associated with changes in mood and ruminative tendencies (Nolan et al., 1998). Converging evidence from these lines of research, along with recent studies investigating brain function associated with mood-congruent memory facilitation in MDD indicates that the amygdala acts to promote the retrieval of mood (or diagnosis) relevant information (Elliott et al., 2002; Ramel et al., 2007) in depression. These findings suggest that mood-congruent emotional memory facilitation is a behavioral and biological component that influences the susceptibility to developing depression. The delineation between high and low-risk individuals may be elucidated through the assessment of personality structure, behavioral performance and brain function in response to mood-congruent memory tasks.

3.2. Generalized anxiety disorder

One of the central features of generalized anxiety disorder (GAD) is a bias towards the processing of threat-related stimuli (Bishop, 2007; Tyrer and Baldwin, 2006). Studies investigating this bias as applied to memory (Coles and Heimberg, 2002) have identified implicit (MacLeod and McLaughlin, 1995; Mathews et al., 1989) but not explicit (Becker et al., 1999; Mogg et al., 1987) memory biases towards threat-related stimuli in patients with GAD. These studies suggest that the memory and attentional biases associated with clinical anxiety are manifested automatically (implicitly) and may influence several of the cognitive symptoms that characterize this disorder (Eysenck et al., 2007).

Variation of personality structure is linked to the experience of anxiety-related symptoms as well as the diagnosis of GAD. Spielberger's state and trait anxiety inventory (STAI) (Spielberger et al., 1970) specifically maps onto features and symptoms experienced in GAD and can also be used in order to assess variation of anxiety-related traits within non-clinical samples. One of the underlying sub-facets of neuroticism is anxiety (N3) and also maps onto several of the features associated with GAD (Bagby et al., 1997; Costa and McCrae, 1992). Additionally, studies have identified a relationship to exist between neuroticism and GAD diagnosis (Bienvenu et al., 2004; del Barrio et al., 1997; Sharma, 2003). Clearly, the investigation of how personality, cognitive/affective performance and brain function interact with one another and converge with clinical research of anxiety will further our understanding of how vulnerability mechanisms are linked with this disorder.

Both non-clinical variation of anxiety-related traits and clinical diagnosis of GAD are associated with greater attention towards fear or threat-related stimuli and with greater amygdala reactivity during this process. Specifically, higher scores of STAI are linked with increased attention to threat-related stimuli (Bar-Haim et al., 2005; Fox et al., 2005) as well as to increased amygdala reactivity during this process (Bishop et al., 2004b; Etkin et al., 2004). Additionally, heightened amygdala reactivity to fear or threat-related stimuli is a primary neural feature of those diagnosed with GAD (Rauch et al., 2003). This convergence suggests that biased attention towards threat or fear-related stimuli paired with increased amygdala reactivity may serve as a biobehavioral marker for susceptibility to developing GAD and may foster the emotional memory related symptoms in this disorder.

Modulation of stress hormones via the HPA axis likely contributes to the development of memory biases in anxiety. The diagnosis of GAD is associated with aberrant levels of both cortisol and epinephrine (Cameron et al., 2004; Gorman et al., 2002; Kallen et al., 2007; Sullivan et al., 1998). Individual differences of traits linked to GAD (STAI and neuroticism) are related to variation of both cortisol (Schlotz et al., 2006) and epinephrine (Jezova et al., 2004). As previously described, these hormones influence the retention of emotional memories. Combined, these studies implicate that atypical HPA axis function may relate to the susceptibility of developing GAD and likely contributes to the emotional memory biases identified to occur in anxiety.

Consistent with the diagnostical features of GAD, those scoring higher on STAI and neuroticism exhibit greater amounts of anxious mood states. Recent studies have investigated mood-congruent memory facilitation in GAD (Coles et al., 2007) and trait anxiety (Russo et al., 2001; Russo et al., 2006) and have reported converging results. Specifically, these studies report that during states of high anxiety, a greater amount of threat-related information is recalled. Brain imaging studies have identified that anxious mood states are associated with anterior cingulate reactivity (Bishop et al., 2004a). Combined, this research implicates that the affinity that individuals have toward the experience of anxiety-related moods, paired with cingulate reactivity, contributes to the development and maintenance of emotional memory biases in GAD.

3.3. Post-traumatic stress disorder

Those suffering from post-traumatic stress disorder are particularly concerned, directed, and focused on remembering their traumatic life experiences (Mollica et al., 2007; O'Donnell et al., 2004a). Individuals with PTSD exhibit enhanced priming (Michael et al., 2005) and memory (McNally, 1997) for emotional trauma-related words as well as memory deficits for emotionally neutral information (Brewin et al., 2007). These abnormalities in memory function are linked to a host of clinically pervasive cognitive and behavioral symptoms ranging from attentional deficits (Jenkins et al., 2000) to aberrant sleep (McNally, 2006). Risk factors such

as age, gender, and personality structure have been identified to be associated with the vulnerability of developing PTSD as well as with specific patterns of symptomology.

Variation in personality structure is associated with the relative susceptibility to developing PTSD (Bramsen et al., 2000; Engelhard et al., 2006; Fauerbach et al., 2000; Gil and Caspi, 2006; Knezevic et al., 2005) as well as the severity of symptoms within patients that are currently diagnosed (Chung et al., 2007; Hyer et al., 1994). These studies have identified that higher scores of neuroticism and harm-avoidance measured either before or after a traumatic event can serve to prospectively predict the onset of a PTSD diagnosis. Additionally, within populations that are currently diagnosed, higher scores of neuroticism are associated with increased severity of PTSD related symptoms (Chung et al., 2007; Hyer et al., 1994). Combined, these studies suggest a possible modulatory relationship to exist between personality traits and PTSD onset. This is consistent with the established relationship between personality and amygdala and neuroendocrine function, two systems that are strongly linked with this disorder.

Amygdala function is implicated in the development as well as the maintenance of PTSD. The amygdala is thought to facilitate the consolidation of the emotional memory trace linked to the traumatic event that contributes to the onset of PTSD (McIntyre et al., 2003). This is supported by research demonstrating that amygdala activity increases when processing fearful or trauma-related stimuli (Nitschke et al., 2006; Whalen, 1998) and that the subjective appraisal of these stimuli corresponds to how well those stimuli are encoded into memory (Canli et al., 2000). Additionally, patients with a current diagnosis of PTSD exhibit greater amygdala reactivity in response to fearful or trauma-related stimuli either presented consciously (Francati et al., 2007) or unconsciously (Bryant et al., 2007; Hendler et al., 2003). This pattern of reactivity is consistent with the pattern identified to be associated with traits identified that correspond to relative vulnerability, harm-avoidance (Most et al., 2006) and neuroticism (Haas et al., 2007b). Clearly, these studies indicate that an understanding of the relationship between personality variables and amygdala reactivity to aversive stimuli may be cardinal to the identification of vulnerability markers of PTSD.

PTSD is associated with atypical neuroendocrine variation (Olf et al., 2006; Yehuda, 1998). Patients with PTSD exhibit increased epinephrine (Delahanty et al., 2005) and norepinephrine (O'Donnell et al., 2004b) levels. Patients also exhibit blunted cortisol levels, though this finding is thought to be inconsistent (Yehuda, 2006) and exaggerated cortisol response to psychological and pharmacological stressors (de Kloet et al., 2006). These associations suggest that manipulating neuroendocrine levels *in vivo* may be a promising strategy in order to ameliorate PTSD symptoms. Recent studies have supported this claim (Debiec and LeDoux, 2006; Pitman et al., 2002; Taylor and Cahill, 2002), though further validation is necessary prior to clinical use.

Interestingly, variation of neuroendocrine function is associated with personality traits that have been implicated as vulnerability markers for PTSD. For example, subjects scoring high on neuroticism exhibit exaggerated dose-dependant cortisol responses to pharmacological stressors (Mangold and Wand, 2006), similar to those described in those with PTSD (de Kloet et al., 2006). Additionally, the blockage of the action of epinephrine on Beta-adrenergic receptors is associated with diminished emotional memory retention (Cahill et al., 1994) and variation of epinephrine is associated with both individual differences in personality (Miller et al., 1999) and with PTSD (Delahanty et al., 2005). This convergence suggests that baseline epinephrine levels along with Beta-adrenergic receptor sensitivity and personality structure (relating to stress sensitivity) may serve as psychoneuroendocrinological susceptibility markers for this disorder.

4. General discussion

In this review, we have explored how three neural systems engaged in emotional memory differ according to personality structure and may therefore relate to vulnerability to developing psychopathological states characterized by emotional memory dysfunctions. In doing so, we have provided evidence that variation in these systems is likely related to variability of emotional memory biases within non-clinical populations and to the severity of emotional memory related symptoms in those with psychopathology. The systems that we have explored are not unique to facilitating emotional memory and likely contribute to a host of cognitive, motivational and behavioral functions that are not dealt with depth in the current review and are therefore in need of further consideration.

The amygdala is implicated in governing a wide range of emotional and motivational functions. These attributed functions stem from early animal research associating amygdala function with fear conditioning. In our current system approach, we have highlighted the amygdala's role in the attention to emotional stimuli as well as to how this attention may vary between individuals. Clearly, as evinced from the growing body of studies associating the amygdala with functions ranging from olfactory discrimination (Schoenbaum, Setlow, Nugent, Saddoris, and Gallagher, 2003) to behavioral inhibition (Morgan, 2006) this structure's function will likely continue to be linked to models of several types of behaviors. The convergence identified in this review documents the growing evidence implicating that the amygdala governs how we process our emotional experiences and how this processing can manifest into psychopathological states.

Neuroendocrine function linked to the HPA axis is a complex and interdependent system with several feed-forward and feedback mechanisms. In reviewing the current literature, we provide evidence that variation of these hormones is critical to how emotional memories are stored and likely influences variability in emotional memory facilitation between people. We have considered the functions of cortisol and the action of epinephrine of Beta-adrenergic receptors as relatively independent entities. This is clearly a comparatively simplistic view given the complex interrelated functions of these stress hormones. We therefore suggest that the attributed functions described in this review be considered as only one aspect of this highly complex system. Future studies may elucidate this interdependency by combining measurements of several types of hormones and from different locations (central vs. periphery).

This review focused on three neural systems and non-clinical individual variation as assessed by personality structure. However, there is also evidence supporting the roles of other neural systems engaged in emotional memory as well as evidence identifying how other variables such as genes (Fahim et al., 2004) and gender (Canli et al., 2002a) are linked to emotional memory variation. For example, the prefrontal cortex is thought to be critical in distinguishing between valence and arousal during the encoding of emotional memory (Kensinger and Corkin, 2004) and exhibits varying amounts of activity associated with personality (Gray et al., 2005). The hippocampus is engaged in emotional memory function (Richardson et al., 2004) and also exhibits varying amounts of activity associated with personality (Canli et al., 2001). Additionally, serotonergic function is associated with emotional memory (Merens et al., 2007) and with personality structure (Carver and Miller, 2006). In the current review, we aimed to primarily focus on the three systems (amygdala and attention, neuroendocrine function and interactive effects with mood) based on the substantial amount of evidence supporting the roles of these systems utilizing a wide range of experimental paradigms and neuroscientific measurement techniques. As studies continue to investigate the neural basis of emotional memory facilitation and individual differences in affective processing, the roles of these other systems will likely be elucidated and will motivate further considerations.

In conclusion, the extent to which we remember our experiences has a strong bearing on what characterizes each of us as individuals. Two people may experience the same event, but come away from it with completely different emotional memories. These differences are rooted in neural systems that are likely associated with the relative resilience and vulnerability that each of us have to developing psychological dysfunctions. By investigating the variability of these systems within normal and clinical populations we may identify vulnerability markers and contribute to the development of more effective diagnostic and preventative techniques.

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Fig. 1.

Schematic representation of neural systems engaged in emotional memory that vary as a function of personality. Personality is associated with differences in the attention to emotional stimuli (A). Attention to emotion stimuli is linked with amygdala (AMG) activity and with the interaction between the amygdala and the extrastriate cortex (EXS). Differences in attention (paired with amygdala activity) are associated with the facilitation of emotional memory. Personality is associated with differences in the stress response (B). The stress response is linked with HPA axis function and the action of epinephrine on Beta-adrenergic receptors within the basolateral amygdala (bAMG). Differences in HPA axis function and Beta-adrenergic receptor sensitivity are associated with the facilitation of emotional memories. Personality is associated with differences in mood states (C). Changes in mood states are linked with changes in anterior cingulate (AC) and amygdala (AMG) activity. Variation in mood paired with cingulate and amygdala activity is associated with emotional memory facilitation. AMG = amygdala; EXS = extrastriate cortex; bAMG = basolateral amygdala; HPA axis = hypothalamic–pituitary–adrenal axis; AC = anterior cingulate.