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Differential effects of stress-induced cortisol responses on recollection and familiarity-based recognition memory

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ABSTRACT

Stress-induced changes in cortisol can impact memory in various ways. However, the precise relationship between cortisol and recognition memory is still poorly understood. For instance, there is reason to believe that stress could differentially affect recollection-based memory, which depends on the hippocampus, and familiarity-based recognition, which can be supported by neocortical areas alone. Accordingly, in the current study we examined the effects of stress-related changes in cortisol on the processes underlying recognition memory. Stress was induced with a cold-pressor test after incidental encoding of emotional and neutral pictures, and recollection and familiarity-based recognition memory were measured one day later. The relationship between stress-induced cortisol responses and recollection was non-monotonic, such that subjects with moderate stress-related increases in cortisol had the highest levels of recollection. In contrast, stress-related cortisol responses were linearly related to increases in familiarity. In addition, measures of cortisol taken at the onset of the experiment showed that individuals with higher levels of pre-learning cortisol had lower levels of both recollection and familiarity. The results are consistent with the proposition that hippocampal-dependent memory processes such as recollection function optimally under moderate levels of stress, whereas more cortically-based processes such as familiarity are enhanced even with higher levels of stress. These results indicate that whether post-encoding stress improves or disrupts recognition memory depends on the specific memory process examined as well as the magnitude of the stress-induced cortisol response.

1. Introduction

Stress can have detrimental effects on memory. For example, acute stress, such as that induced in the laboratory by briefly submerging one's arm in ice water (i.e., the cold-pressor test) can reduce the ability to retrieve information from memory (e.g., Smeets, Otgaar, Candel, & Wolf, 2008). This impairment can also be induced by administration of the stress-related hormone cortisol just prior to retrieval (e.g., Wolf, Kulhmann, Buss, Hellhammer, & Kirschbaum, 2004). Moreover, chronic stress can lead to long-term memory impairments, as seen in individuals suffering from post-traumatic stress disorder (PTSD, e.g., Lindauer, Olff, van Meijel, Carlier, & Gersons, 2006). But stress does not always have detrimental effects on memory, and a number of studies have now shown that acute stressful experiences that occur shortly after learning can facilitate memory (e.g., Andreano & Cahill, 2006; Cahill, Gorski, & Le, 2003; McCullough & Yonelinas, 2013). Because in each of those studies, the stress was administered after learning and well before retrieval, the enhancement of memory cannot be attributed to encoding or retrieval processes, but must reflect enhanced consolidation or slowed forgetting.

The effects of stress on memory are thought to be mediated by glucocorticoid and adrenergic hormones that act on the medial temporal lobe (MTL) regions supporting memory, such as the hippocampus and the amygdala (for reviews, see McEwen & Sapolsky, 1995; McGaugh & Roozendaal, 2002). For example, it is thought that acute stress leads to an increase in the glucocorticoid hormone cortisol, which can enhance the retention of recently encoded memories by facilitating long-term potentiation in the MTL (McEwen & Sapolsky, 1995). Prolonged stress, on the other hand, can lead to abnormal basal cortisol levels and diurnal rhythms (Schulz, Kirschbaum, Prüßner, & Hellhammer, 1998), which have been associated with hippocampal volume reductions and cell death (Lupien et al., 1998), and thus can have long-term detrimental effects on memory.

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The effects of post-encoding stress and cortisol release on memory, however, are not yet fully understood. For example, while a number of studies have found that post-encoding stress improved recall memory for emotional, but not neutral materials (e.g., Cahill et al., 2003; Smeets et al., 2008), other studies have found that post-encoding stress improved recall of neutral and emotional materials (e.g., Nielson & Lorber, 2009), and yet others have found recall enhancements for neutral, but not emotional materials (e.g., Preuß & Wolf, 2009). Studies of recognition memory have found that post-encoding stress induced by skydiving (Yonelinas, Parks, Koen, Jorgenson, & Mendoza, 2011) or by the cold pressor test (McCullough & Yonelinas, 2013) enhanced recognition for neutral pictures more than emotional pictures, while not influencing subsequent recall of either. In both of those studies, further analysis of the recognition data revealed that the stress-related enhancements were localized primarily to familiarity-based recognition, and recollection was unaffected by stress. Thus, stress after learning has generally been shown to enhance subsequent memory, but the reported effects of post-encoding stress have not been consistent with respect to the emotional content of the to-be-remembered information, and it is not yet clear when stress will influence different memory processes (e.g., recollection, familiarity, recall).

One potential explanation for these mixed results is that there could be a non-monotonic relationship between stress and memory. In fact, animal studies have shown that memory is related to glucocorticoid levels with an inverted-U shaped function (e.g., Roozendaal, 2000, 2002), suggesting that moderate levels of post-encoding stress may be associated with better subsequent memory performance than what is observed after lower or higher levels of stress. To our knowledge, there has only been one human study that has reported a non-monotonic relationship between an endogenous cortisol release and subsequent memory (Andreano & Cahill, 2006). In that experiment, participants first read a moderately arousing story and then completed either a cold-pressor test in which they submerged their arm in ice water for 3 min or a control task using warm water. On a subsequent free recall test they observed better overall performance in the stress group than the control group. Among male participants in the stress condition, however, recall performance was higher for those who showed a moderate stress-related cortisol increase, relative to participants who showed small or large cortisol increases.

However, it is unknown whether this type of non-monotonic response function relates stress-induced cortisol responses to other memory measures, such as recollection or familiarity processes supporting recognition memory. Recollection is thought to rely on the hippocampus whereas familiarity relies on the surrounding MTL cortex (for a review, see Eichenbaum, Yonelinas, & Ranganath, 2007). Given that the density of glucocorticoid and mineralocorticoid receptors is particularly high within the hippocampus (Seckl, Dickson, Yates, & Fink, 1991; Watzka et al., 2000), one may expect these two recognition processes to be differentially sensitive to stress-related changes in cortisol. In fact, given that different memory tests are supported by recollection and familiarity to different degrees, such a dissociation might help explain the mixed results in the literature.

In addition to stress-induced changes in cortisol, basal levels of cortisol vary widely across individuals, and little is known about how this variability relates to memory in healthy young adults. It is well established that basal cortisol levels can be abnormal in groups showing memory deficits such as in aging populations (Li et al., 2006; Lupien et al., 1994), patients with PTSD (Lindauer et al., 2006), and clinically depressed individuals (Belanoff, Kalehzan, Sund, Ficek, & Schatzberg, 2001; but see Barnhofer, Kuehn, & de Jong-Meyer, 2005), and in some cases basal cortisol levels have been associated with hippocampal volume in these populations (e.g., Lindauer et al., 2006; Lupien et al., 1998). A few studies have examined the relationship between pre-learning baseline cortisol levels and free recall in healthy young adults, and have either reported no significant relationship (e.g., Ackermann, Hartmann, Papassotiropoulos, de Quervain, & Rasch, 2013), or a significant association only under certain conditions, such as after intentional but not incidental encoding (Preuß, Schoofs, & Wolf, 2009), after a night of sleep but not after a no-sleep delay of equal length (Bennion, Steinmetz, Kensinger, & Payne, 2013), and when the materials are emotional rather than neutral (Preuß et al., 2009). Although in the two latter studies a positive association was observed between pre-learning cortisol levels and memory performance, negative relationships between baseline cortisol and memory performance have also been reported in healthy young adults (e.g., van Honk et al., 2003). However, no previous study that we are aware of has examined the relationship between pre-learning cortisol levels and recognition memory or the processes of recollection and familiarity.

In the current study, we examined the relationship between stress-induced cortisol responses and recognition memory processes, as well as between pre-learning baseline cortisol levels taken at the beginning of the study and recognition processes in a sample of healthy young adult men. We note that our measure of pre-learning baseline cortisol can be impacted by many factors such as the subject's expectation that they will be tested in a stressful experiment. Nonetheless, baseline levels of cortisol are important to examine as they may influence the learning phase itself and be related to memory in different ways than cortisol changes induced by the post-encoding experimental manipulation of stress. We restricted our sample to males because previous human and animal work has indicated that the effects of stress on memory are pronounced in males (e.g., Andreano & Cahill, 2006; Conrad et al., 2004). In addition, we used a common laboratory stressor (i.e., the cold-pressor test), which, in combination with brain imaging procedures, elicited a large change in cortisol levels.

Participants encoded both negative emotional images and neutral images. Immediately after encoding, participants in the stress group submerged one arm in ice water, whereas participants in the control group submerged one arm in warm water. After a 24-h delay, participants in both groups were given a recognition memory test, in which they were asked to indicate whether they recollected each image, and if not, to rate their recognition confidence (to assess familiarity). We used the confidence judgments to compute estimates of recollection and familiarity using a dual-process ROC approach and a Remember/Know approach (Yonelinas et al., 2011), as well as two measures of performance based on single process models of recognition: d' for medium-confidence responses and d' for high-confidence responses (MacMillan & Creelman, 2005).

Salivary cortisol was measured at the beginning of the experiment, then 20 min after the stressor, when stress-induced cortisol responses were expected to be maximal (Schwabe, Böhringer, Chatterjee, & Schachinger, 2008; Schwabe, Böhringer, & Wolf; 2009; Schwabe & Wolf, 2009), and again just prior to the recognition memory test. We examined the relationship between the magnitude of post-encoding stress-induced cortisol release, as measured by the difference in cortisol between the initial cortisol measure and the sample taken shortly after the stress manipulation, and recollection and familiarity-based recognition responses. In addition, we examined whether pre-learning levels of cortisol were differentially related to the processes supporting recognition memory, which we determined by examining the relation between the initial cortisol measure and estimates of recollection and familiarity.