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# Modulatory effects of stress on reactivated emotional memories

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## KEYWORDS

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**Summary** Previous studies have shown that stress, through secretion of stress hormones, increases the consolidation of memory while it exerts negative effects on memory retrieval. Other studies show that the process of memory retrieval serves as a reactivation mechanism whereby the memory trace that is reactivated during the retrieval process is once again sensitive to modifications by pharmacological or environmental manipulations. In this study, we assessed whether exposure to stress after retrieval of neutral and emotional information modulates the immediate and long-term recall of these reactivated memory traces. Three groups of participants (total *N* of 47) encoded on Day 1 a movie containing neutral and emotional information. Two days later (Day 2), one group was asked to retrieve (reactivate) the story before being exposed to a stressful condition (reactivation/stress group), while the second group was asked to retrieve the story and was not exposed to a stressful condition (reactivation/no stress group). A third group did not recall the story but was exposed to a stressful condition (no reactivation/stress group). All participants were asked to recall the story immediately after exposure to the stress/no stress condition (immediate recall) as well as 5 days later (delayed recall). Results show that immediate recall of emotional information was significantly increased in the reactivation/stress group when compared to the reactivation/no stress group while no effect of stress on reactivated neutral memories was found. Moreover, evidence suggests that the enhanced memory trace is maintained across time, suggesting a potential long-lasting effect of stress on reactivated memory traces. We also found that the enhanced emotional memory trace observed in the reactivation/stress group was not present in the no reactivation/stress group, showing that stress has the capacity to enhance memory only when the memory trace is acutely reactivated before exposure to stress. Altogether, these results suggest that stress differentially modulates reactivated emotional and neutral memory traces and that this effect is long-lasting. These results have important implications for the potential influence of acute stress on reactivated memories in individuals exposed to traumatic events.

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## 1. Introduction

Upon perception of stress, the hypothalamic–pituitary–adrenal (HPA) axis is activated, which results in the secretion of glucocorticoids (GCs; cortisol in humans). Given its liposolubility, this stress hormone has the capacity to rapidly cross the blood–brain barrier and bind to GC receptors localized in the hippocampus, the amygdala and the prefrontal cortex (McEwen et al., 1968; Reul and de Kloet, 1985; Gray and Bingaman, 1996; Sanchez et al., 2000; Joels, 2001). Considering the importance of these structures in memory function, various studies have assessed the acute effects of increased levels of GCs on memory performance in humans (for reviews see Lupien and McEwen, 1997; Roozendaal, 2000, 2002; Lupien and Lepage, 2001; Lupien et al., 2007; Roozendaal et al., 2008). In the majority of these studies, elevations of circulating levels of GCs have been induced either by the administration of exogenous GCs, or by exposure to psychosocial stressors, which leads to an endogenous increase in GC levels.

In general, these studies have shown that acute increases of GCs either immediately before or after the encoding process increase memory consolidation (Buchanan and Lovallo, 2001; Abercrombie et al., 2003; Cahill et al., 2003; Andreano and Cahill, 2006; Beckner et al., 2006; Kuhlmann and Wolf, 2006a; Payne et al., 2007; Schwabe et al., 2008). With regards to the retrieval process, most studies reveal impairing effects of acute increases of circulating GCs on retrieval (de Quervain et al., 1998, 2000; Kuhlmann et al., 2005a,b; Kuhlmann and Wolf, 2005; Buchanan et al., 2006; Buchanan and Tranel, 2008; Tollenaar et al., 2009). Some studies suggest that these effects are particularly pronounced for emotional material (Buchanan and Lovallo, 2001; Cahill et al., 2003; Kuhlmann et al., 2005a,b; Kuhlmann and Wolf, 2006b; Payne et al., 2007). Altogether, these results show that GCs have strong modulatory influences on both the consolidation and the retrieval processes, and that emotional material seems to be affected differently than neutral material.

Other studies in the field of cognitive neurosciences have demonstrated that the process of memory retrieval serves as a reactivation mechanism whereby the memory trace that is reactivated during the retrieval process is once again sensitive to modifications by pharmacological or environmental manipulations (Misanin et al., 1968; Nader et al., 2000). This is an important finding, as it implies that immediately after being reactivated, the nature and/or the amplitude of the memory trace could theoretically be modified or modulated by environmental influences, and this effect could potentially persist over time.

To this day, only a handful of studies have assessed the association between variations in stress hormones and memory reactivation. Animal studies demonstrated that the administration of a noradrenergic blocker in the amygdala following reactivation of a fear memory can decrease this memory trace in a long-lasting manner (Debiec and LeDoux, 2006). Other animal studies using GC showed that injection of corticosterone after memory reactivation impairs both a conditioned-fear memory trace (Cai et al., 2006) and performance on an object-recognition task (Maroun and Akirav, 2008) in a long-lasting manner. In humans, Tollenaar et al. (2009) have reported an impairing effect of cortisol admin-

istration on the retrieval (reactivation) of neutral and emotional words, and the effect persisted after a week. However, it is important to note that this study administered cortisol before memory retrieval. Another recent experiment performed in abstinent heroin addicts administered the psychosocial stressor after the reactivation of words (neutral words, heroin-related positive and heroin-related negative words). The results showed that memory for the drug-related words (positive and negative) was decreased when assessed one day after stress exposure (Zhao et al., 2009). However, the immediate effects of stress on memory performance were not assessed in that study.

So far, no studies have assessed the immediate and long-term effects of a stressor after memory reactivation of neutral and emotional material in healthy humans. Given the differential effects of stress hormones on the modulation of neutral and emotional memory, it thus becomes important to assess whether stress can differentially modulate the nature and/or the amplitude of retrieved (reactivated) neutral and emotional memory traces. Consequently, the current study, performed in healthy human participants, tested whether exposure to a psychosocial stressor following the reactivation of a neutral and an emotional memory trace can modulate the short- and long-term recall of this information. Given that an elevation in stress hormone levels has been shown to increase memory consolidation for emotional information, and that the few human studies that have investigated the question of cortisol elevation after memory reactivation have reported impairing effects on both neutral and emotional memories, we were expecting an effect of stress on memory performance, but we did not have a clear hypothesis about the direction of this effect as a function of the valence of the material to be remembered.

## 2. Method

### 2.1. Participants

Forty-seven healthy participants (22 men and 25 women) aged between 18 and 35 years (mean  $\pm$  S.E.M. = 22.09  $\pm$  0.50) took part in this study. This study was approved by the Ethic Research Committee of the Douglas Mental Health University Institute. All participants were screened over the phone to make sure that they did not suffer from any physiological or psychological condition that could impact on the results of the study. They were all non-smokers and free of medication except for some women taking contraceptive pills ( $n = 16$ ). None of the women were tested when they were having their periods. Before the start of the procedure, participants gave their informed consent to take part in the study. In order to assess the effects of stress on reactivated memories, three groups were tested. Sixteen participants were randomly assigned to the reactivation/stress condition (8 males and 8 females), sixteen others (7 males and 9 females) were assigned to the reactivation/no stress condition and fifteen others (7 males and 8 females) were assigned to the no reactivation/stress condition. Participants were asked to refrain from eating and drinking (except water) 1 h before the start of every testing session. Upon their arrival, participants were asked the time at which they woke up on that day, and time of awakening was compared across groups and sessions to determine potential differences that may impact on the results.

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