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True or false? Memory is differentially affected by stress-induced cortisol elevations and sympathetic activity at consolidation and retrieval

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Summary Adrenal stress hormones released in response to acute stress may yield memory-enhancing effects when released post-learning and impairing effects at memory retrieval, especially for emotional memory material. However, so far these differential effects of stress hormones on the various memory phases for neutral and emotional memory material have not been demonstrated within one experiment. This study investigated whether, in line with their effects on true memory, stress and stress-induced adrenal stress hormones affect the encoding, consolidation, and retrieval of emotional and neutral false memories. Participants ($N = 90$) were exposed to a stressor before encoding, during consolidation, before retrieval, or were not stressed and then were subjected to neutral and emotional versions of the Deese—Roediger—McDermott word list learning paradigm. Twenty-four hours later, recall of presented words (true recall) and non-presented critical lure words (false recall) was assessed. Results show that stress exposure resulted in superior true memory performance in the consolidation stress group and reduced true memory performance in the retrieval stress group compared to the other groups, predominantly for emotional words. These memory-enhancing and memory-impairing effects were strongly related to stress-induced cortisol and sympathetic activity measured via salivary alpha-amylase levels. Neutral and emotional false recall, on the other hand, was neither affected by stress exposure, nor related to cortisol and sympathetic activity following stress. These results demonstrate the importance of stress-induced hormone-related activity in enhancing memory consolidation and in impairing memory retrieval, in particular for emotional memory material.

1. Introduction

A plethora of research has shown that emotional events are better remembered than neutral ones (LaBar and Cabeza, 2006), an effect driven by adrenal stress hormones that act on brain structures central to memory (e.g., McGaugh and

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Roozendaal, 2002). That is, noradrenaline release and β-adrenoceptor activation within the basolateral amygdala (BLA) modulates memory consolidation. One important issue is that adrenal stress hormones such as noradrenaline and glucocorticoids (GCs) may encompass differential effects on the various memory phases. Specifically, while stress hormones impair retrieval (e.g., de Quervain et al., 2000; Kuhlmann et al., 2005a,b; Buchanan and Tranel, 2008), they can enhance memory when released post-learning (i.e., during consolidation; e.g., Cahill et al., 2003; Andreano and Cahill, 2006). So far, the differential effects of stress hormones on the various memory phases for neutral and emotional memory material have not been demonstrated within a single study.

In contrast to the massive amount of studies on the effects of stress hormones on true memory (for reviews, see Het et al., 2005; Lupien et al., 2005; Wolf, 2008), only few studies have looked at the effects of stress on false memories. One paradigm aimed at eliciting false memories is the Deese–Roediger–McDermott paradigm (DRM; Deese, 1959; Roediger and McDermott, 1995). Here, participants are presented with lists of semantic associates (e.g., ''bed'', ''tired'', ''dream'') after which recall performance is assessed. Typically, people often falsely recall the semantically related, non-presented theme words (termed ''critical lures''; in this case ''sleep''). In a study by Payne et al. (2002), participants were exposed to the Trier Social Stress Test (TSST; Kirschbaum et al., 1993) or a non-stressful filler task after which they had to listen to 20 DRM word lists, each followed by a computerized recognition task. Compared to controls, participants exposed to the TSST showed elevated rates of false recognition for the critical lures. Thus, the Payne et al. (2002) findings imply that people under stressful circumstances are more vulnerable to false recollections. In contrast, Smeets et al. (2006a) showed that neither stress-exposure (Study 1) nor stress-induced GC (i.e., cortisol) responses (Study 2) are sufficient to potentiate false recollections in a DRM paradigm. One explanation for these divergent findings would be that Payne et al.'s findings showing increased levels of false memories are not GC driven, but rather relate to the Sympatho-Adrenal Medullary (SAM) axis driven memory effects. Many studies indeed revealed that GCs interact with adrenergic hormones and noradrenergic activation in the BLA in modulating memory performance (i.e., enhanced memory consolidation and exacerbated memory retrieval; e.g., McGaugh, 2000; Roozendaal, 2000; Roozendaal et al., 2004, 2006; Kuhlmann and Wolf, 2006; de Quervain et al., 2007).

The primary aim of this study was to assess the effects of stress-induced activity of the SAM and HPA axes on false recall following exposure to an acute stressor, in comparison to their effects on true recall. A secondary aim of this study was to specifically look at how stress-induced hormonal changes affect the encoding, consolidation, and retrieval phase of the DRM paradigm. To the best of our knowledge there are no studies that have looked at whether, in line with their effects on true memory, adrenal stress hormones have differential effects on false memories for emotional versus neutral stimuli. Thus, another aim of this study was to investigate this issue by concurrently looking at adrenergic activity and GC involvement in stress-induced neutral and emotional false recall.

2. Materials and methods

2.1. Participants

Ninety undergraduate students (84 women[1]) with a mean age of 20.6 years (S.D. = 1.4; range: 18–25) participated in this study. All were right-handed, non-smoking individuals with a normal Body Mass Index (BMI; Mean ± S.D.: 21.8 ± 2.5; range: 17.4–28.5). Suffering from cardiovascular diseases, severe physical illnesses (e.g., fybromyalgia), hypertension, endocrine disorders, or being on any kind of medication served as additional exclusion criteria. Test protocols were approved by the standing ethics committee of the Psychology Faculty of Maastricht University. All participants signed a written informed consent and were financially compensated (12.5€; approximately 18$) in return for their participation.

2.2. Cold pressor stress

Stress was induced by exposing participants to cold pressor stress (CPS). The CPS is a widely used, low-risk technique in medical research to expose participants to painful stressors and is known to induce robust and reliable stress responses (e.g., Lovallo, 1975; Bohus et al., 2000; Cahill et al., 2003; Mitchell et al., 2004). As is typical in research employing CPS, participants were instructed to immerse their dominant arm up to the elbow in ice-cold (0–1 °C) water for as long as possible with a maximum of 3 min. They were explicitly told that, as the procedure could be very uncomfortable, they could remove their arm from the ice-cold water at their own discretion without consequences. Participants who fully endured CPS were told to remove their arm after 3 min. In the control condition, participants were instructed to place their arm in warm (37–40 °C) water until they were instructed to remove their arm. This instruction was given pseudo-randomly across participants after 1, 2, or 3 min following arm immersion. Arm immersion always occurred single-blind. That is, participants were not informed beforehand to which group they were assigned until immediately before arm immersion, even though they did know at the outset that they could be asked to put their arm in ice-cold water. During the CPS or control test, the experimenter always remained in the test room to monitor participants' compliance with the test instructions. Following CPS, all participants had to rest their arm covered by a blanket for 3 min. In line with Cahill et al. (2003), participants were asked to rate the level of discomfort they experienced during water immersion. To this end, they first were asked to think back at the most intense physical pain they had ever experienced and rate this experience by appropriately marking a 0–100 scale (anchors: 0 = *no pain or discomfort*; 100 = *the worst pain or discomfort imaginable*). After this ''calibration'' scale, participants rated the peak level of discomfort they had experienced during the CPS on an analogous scale.

[1] Of the six men that participated in this study, three were in the encoding stress group, two were in the retrieval stress group, and one was in the control group.