



The role of stress during memory reactivation on intrusive memories



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ABSTRACT

Intrusive memories are unwanted recollections that maintain distress in psychological disorders. Increasing evidence suggests that memories that are reactivated through retrieval become temporarily vulnerable to environmental or pharmacological manipulation, including changes in levels of circulating stress hormones. This study investigated the influence of stress during memory reactivation of an emotionally arousing trauma film on subsequent intrusive memories. Three groups of participants ($N = 63$) viewed a trauma film depicting a serious car accident at baseline. Two days later (Time 2), one group received a reactivation induction following a socially evaluated cold pressor test (SECPT; Stress/Reactivation condition), whilst the second group reactivated the memory after a control procedure (Reactivation condition). A third group underwent the SECPT but was not asked to reactivate memory of the trauma film (Stress condition). Two days later (Time 3), all participants received a surprise cued memory recall test and intrusions questionnaire which they completed online. Results showed that those in the Stress/Reactivation group had higher intrusions scores than the other two groups, suggesting that acute stress promotes intrusive memories only when the memory trace is reactivated shortly afterwards. Increased cortisol predicted enhanced intrusive experiences in the Stress/Reactivation condition but not in the other conditions. This pattern of results suggests that acute stress during the reactivation of emotional material impacts on involuntary emotional memories. These findings suggest a possible explanation for the mechanism underlying the maintenance of intrusive memories in clinical disorders.

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1. Introduction

Intrusive memories are repetitive, unwanted sensory–perceptual recollections that are experienced involuntarily and are often associated with negative affect (Clark & Rhyno, 2005). These memories are primarily visual, however, can contain certain sounds, smells and tastes (Brewin, Gregory, Lipton, & Burgess, 2010). They are a common feature of numerous psychological disorders (Krans, Näring, Becker, & Holmes, 2009). Within clinical populations, intrusive memories are commonly associated with high levels of distress, which may maintain cognitive avoidance (Dunmore, Clark, & Ehlers, 1999; Wenzlaff & Wegner, 2000) or rumination (Ehlers, Mayou, & Bryant, 1998; Murray, Ehlers, & Mayou, 2002).

Cognitive models have provided several explanations for the development of intrusive memories. These theories suggest that intrusive memories result from (a) a lack of integration within the autobiographical memory base (Conway & Pleydell-Pearce, 2000), (b) enhanced sensory–perceptual processing at the expense of verbal, contextual processing (Brewin et al., 2010), (c) a lack of a

coherent trauma narrative that has conceptual meaning (Ehlers & Clark, 2000), and (d) fear networks in memory containing associations between trauma cues and fear responding (Foa, Steketee, & Rothbaum, 1989). A high level of stress during the encoding of traumatic events is the main factor thought to lead to these memory processes, however, a lack of experimental studies have examined the mechanism by which this occurs.

Stress hormones have repeatedly been shown to modulate memory for emotional material such that they are better remembered than neutral information. Endogenous and exogenous sources of cortisol and noradrenaline during the encoding and consolidation of emotionally arousing stimuli leads to memory enhancement (Abercrombie, Speck, & Monticelli, 2006; Buchanan & Lovallo, 2001; Cahill & Alkire, 2003; Cahill, Gorski, & Le, 2003). This memory modulation relies on a critical interaction between cortisol and noradrenaline within the basolateral nucleus of the amygdala (Kuhlmann & Wolf, 2006; van Stegeren et al., 2007). In contrast to encoding and consolidation, high levels of circulating stress hormones during the retrieval of emotional memories impairs performance (de Quervain, Aerni, & Roozendaal, 2007; Smeets, Otgaar, Candel, & Wolf, 2008). Although there is some evidence of a positive relationship between levels of emotional

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memory recall and intrusive recollections (Ferree & Cahill, 2009), there is little study of the role of stress hormones on development of intrusive memories. Consistent with the memory modulation hypothesis, one recent study found that the interactive influence of noradrenergic and glucocorticoid activation predicted intrusive memories (Bryant, McGrath, & Felmingham, 2013).

Many studies have now demonstrated that the reactivation of long-term memory reverts it to a labile state that is open to disruption, and requires subsequent consolidation to stabilize it again (Sara, 2000; Schiller & Phelps, 2011). Some evidence from animal studies suggests that increased glucocorticoids after reactivation can result in poorer memory at test (Maroun & Akirav, 2008; Wang, Zhao, Ghitza, Li, & Lu, 2008), although the results are conflicting (Akirav & Maroun, 2013). There are also mixed findings in human studies. In one study memory was impaired for negative (but not neutral) words encoded 5 weeks prior (Tollenaar, Elzinga, Spinhoven, & Everaerd, 2008), and this impairment can endure for one week (Tollenaar, Elzinga, Spinhoven, & Everaerd, 2009); in both these studies, reactivation occurred after the stress manipulation was introduced. In contrast, another study found that decreasing glucocorticoid levels prior to reactivation reduced emotional memory recall in a long-lasting manner (Marin, Hubbach, Maheu, Nader, & Lupien, 2011). Consistent with this finding, another study showed that acute stress enhanced memory for an emotional story only when the memory had first been reactivated (Marin, Pilgrim, & Lupien, 2010). Contrary to the findings of other studies, emotional autobiographical memories have not been influenced by administration of a post-reactivation stressor, however this has been attributed to possible inadequate strength of the stress manipulation (Schwabe & Wolf, 2010). The variations in these findings may be explained, in part, by differences in timing and form of stress manipulation, scheduling of memory testing, and the type of stimuli presented. Despite the attention given to memory reactivation on emotional memory, it has not been studied in relation to intrusive memories.

The present study explored the mechanisms maintaining intrusive memories using a memory reactivation paradigm. We used a trauma film paradigm which has been shown to elicit intrusions in nonclinical populations (Holmes & Bourne, 2008). In addition, we administered a stressor in the form of the socially evaluated cold pressor test (SCEPT), which results in elevated higher hypothalamus–pituitary–adrenal (HPA) responsivity (Schwabe, Haddad, & Schachinger, 2008). Participants were randomized to either a Stress/Reactivation, Stress, or Reactivation group. Two days after encoding, the SCEPT was administered prior to memory reactivation. The role of the noradrenergic and glucocorticoid stress response in predicting memory was indexed using saliva sampling at various time points across the experiment. Finally, two days later participants received a surprise cued recall test and measure of intrusive memories. In terms of hypotheses, the current evidence is very mixed concerning the role of acute stress in the context of reactivation on subsequent emotional memories, and no studies have been conducted in relation to intrusive memories. However, on the basis of the evidence that acute stress manipulation does enhance reactivated emotional memories (Marin et al., 2010), we predicted more intrusive emotional memories in participants who received both the stressor and reactivation induction.

2. Method

2.1. Participants

Sixty-three undergraduate psychology students (29 males and 34 females) of mean age 19.89 years ($SD = 2.30$) participated in the study in return for course credit. Participants were randomly

allocated to one of three groups: Stress ($n = 21$; 12 women, 9 men); Stress/Reactivation ($n = 21$; 11 women, 10 men); and Reactivation ($n = 21$; 11 women, 10 men).

2.2. Measures

2.2.1. Depression, anxiety and stress scales 21 (DASS 21; (Lovibond & Lovibond, 1995)

The DASS 21 was used to index the severity of depression, anxiety and stress related symptoms in the past week. This scale has demonstrated a stable factor structure and good internal consistency (Tully, Zajac, & Venning, 2009).

2.2.2. Traumatic screening questionnaire (TSQ)

The TSQ is a 10-item questionnaire developed as a brief screening tool to predict current and past posttraumatic stress disorder (PTSD). Endorsement of six or more symptoms occurring within a week-long period indicates possible PTSD. This measure was included to ensure that participants did not suffer PTSD at the time of the experiment because this condition is the exemplar condition for intrusive memories (Krans et al., 2009).

2.2.3. Intrusions questionnaire

Intrusive memories of the trauma film were measured using selected items from the Intrusions Subscale of the Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979). Participants were asked to rate the degree to which they agreed with each item on a 5-point Likert scale from 0 (“not at all”) to 4 (“extremely”). The items were “Pictures about it popped into mind”, “Other things kept making me think about it”, and “I thought about it when I didn’t mean to”. These items were framed in terms of memory occurrences of the movie initially presented to participants and that occurred in the intervening two days since the experiment.

2.2.4. Cued recall test

A previously adapted cued recall test for this trauma film was employed to index recall (Deville, Varker, Hansen, & Gist, 2007). We included 24 items with some questions pertaining directly to the victims of the car accident (e.g. “How many of the injured victims had dark skin and how many had light skin?”), whilst others tested memory for details related to the scene’s surroundings (e.g. “What material did the stretcher appear to be made of?”).

2.2.5. Salivary hormone concentration

Levels of circulating endogenous stress hormones were indexed via saliva sampling before and after each task. Sampling was via the passive drool method (without induction), which required participants to fill a self-collection container with 10 mm of saliva. Salivary alpha amylase (sAA) was measured as it has been shown to be a sensitive biomarker of sympathetic nervous system activity (38). In addition, cortisol concentration was measured to assess HPA axis activation. To minimize the effects of natural stress hormone variation across the day, testing was restricted to the hours between 13:00 and 18:00. Participants were also instructed to refrain from exercising 24 h before, eating 1 h before, and ingesting caffeine, alcohol or nicotine 3 h before each experimental session. To coincide with peak levels of sAA and salivary cortisol, respectively, sAA was measured immediately after the stressor was applied, and cortisol was measured approximately 15 min after the onset of the stressor.

2.3. Materials

2.3.1. Trauma film

The trauma film consisted of approximately 10 min of real-life footage of the aftermath of a highway accident. The film depicted

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