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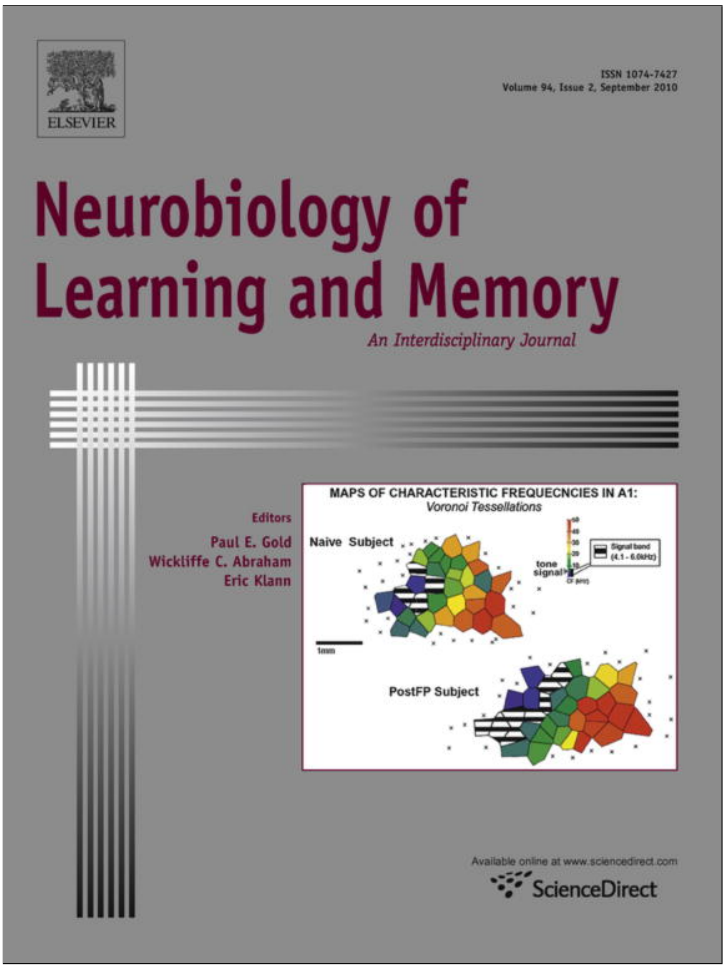

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Neurobiology of Learning and Memory 94 (2010) 153–157

Contents lists available at ScienceDirect

Neurobiology of Learning and Memory

journal homepage: www.elsevier.com/locate/ynlme

Stress impairs the reconsolidation of autobiographical memories

Lars Schwabe *, Oliver T. Wolf

Department of Cognitive Psychology, Ruhr-University Bochum, Bochum 44780, Germany

ARTICLE INFO

Article history:
Received 19 March 2010
Revised 22 April 2010
Accepted 2 May 2010
Available online 22 May 2010

Keywords:
Memory reconsolidation
Stress

ABSTRACT

Stress enhances memory consolidation, in particular for emotional material. When reactivated, consolidated memories return to a fragile state again and thus require another period of stabilization, called reconsolidation. Rodent studies suggest that memory reconsolidation is impaired by stress. Here we examined in healthy humans the effect of stress on the reconsolidation of autobiographical memories. Participants recalled positive, negative and neutral episodes from their recent past and were afterwards exposed to a stressor (socially evaluated cold pressor test) or a non-arousing control condition. Additional groups of participants were exposed to the stressor without prior memory reactivation or were neither stressed nor asked to recall episodes from their past. Stress after memory reactivation impaired the memory for the neutral episodes 1 week later whereas the subsequent memory for the emotional episodes was not affected by stress after reactivation. Reactivation per se or stress without prior memory reactivation had no effect on memory performance. These findings suggest that the effect of stress on memory reconsolidation is opposite to the stress effect on memory consolidation supporting the view that consolidation and reconsolidation are distinct processes.

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1. Introduction

Emotionally arousing experiences are well remembered. This has been attributed to facilitating effects of stress hormones such as glucocorticoids (GCs; cortisol in humans) and catecholamines (adrenaline and noradrenaline) on memory consolidation. Indeed, it is well documented that stress or stress hormones shortly after learning enhance subsequent memory performance (Buchanan & Lovallo, 2001; Cahill, Gorski, & Le, 2003; Kuhlmann & Wolf, 2006; for reviews see McGaugh, 2000; Roozendaal, McEwen, & Chattarji, 2009; Wolf, 2009). These effects are particularly strong for emotionally arousing information (Buchanan & Lovallo, 2001; Cahill et al., 2003) suggesting that an activation of the amygdala is required for stress (hormone) effects on memory consolidation (Roozendaal et al., 2009).

In the past decade, the idea that memory consolidation is not a one-time process, which was first expressed more than 40 years ago (Misanin, Miller, & Lewis, 1968; Schneider & Sherman, 1968), has been revitalized (Dudai & Eisenberg, 2004; Nadel, 2000; Nader & Hardt, 2009; Nader, Schafe, & LeDoux, 2000b; Sara, 2000). There is by now ample evidence from animal and human studies indicating that consolidated memories become labile again when they are reactivated and hence require another period of stabilization which is referred to as reconsolidation. During the reconsolidation window reactivated memories can be changed by amnestic agents or behavioral manipulations (Eisenberg & Dudai, 2004; Hupbach, Gomez, Hardt, & Nadel, 2007; Kindt, Soeter, & Vervliet, 2009; Nader, Schafe, & LeDoux, 2000a; Przybyslawski, Roullet, & Sara, 1999; Schwabe & Wolf, 2009a).

Is memory reconsolidation, same as the original consolidation process, affected by stress and GCs? There is some first evidence from rodent studies that stress or GC administration after memory reactivation reduces subsequent memory (Cai, Blundell, Han, Greene, & Powell, 2006; Maroun & Akirav, 2008; Wang, Zhao, Ghitza, Li, & Lu, 2008). For instance, stress blocked the reconsolidation of object-recognition memory in rats and this effect was reversed by the infusion of a glucocorticoid receptor antagonist into the amygdala (Maroun & Akirav, 2008). Comparable evidence from healthy humans is largely missing.

Therefore, we examined in the present experiment in humans whether the reconsolidation of autobiographical memories is influenced by stress. Autobiographical memories were reactivated by means of the autobiographical memory cueing test (Williams & Broadbent, 1986). Shortly after recalling episodes from their past, participants were exposed to stress (socially evaluated cold pressor test, SECPT) or a non-stressful control condition. The effect of stress on memory reconsolidation was assessed in a memory test 7 days after reactivation. To ensure that stress did not affect memory independent of memory reactivation, we included a group of subjects that were stressed without prior reactivation. In order to control for the effect of time on memory, we had another control

* Corresponding author. Address: Ruhr-University Bochum, Faculty of Psychology, Department of Cognitive Psychology, Universitaetsstrasse 150, 44780 Bochum, Germany. Fax: +49 234 3214308.
E-mail address: Lars.Schwabe@rub.de (L. Schwabe).

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doi:10.1016/j.nlm.2010.05.001

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group that was not stressed and did not reactivate experiences from their past. Based on the existing rodent data (Maroun & Akirav, 2008; Wang et al., 2008) we predicted that stress would impair memory reconsolidation.

2. Methods

2.1. Participants and design

Sixty-four healthy, non-smoking students of the Ruhr-University Bochum participated in this experiment (32 men, 32 women; age: M = 23.3 years, SEM = 0.4 years). Participants were asked to refrain from meals, drinking alcohol or caffeine, and severe physical exercise within the 2 h before the experiment. All subjects provided written informed consent for their participation in the study protocol which was approved by the ethics committee of the German Psychological Society (DGPs).

There were four experimental groups (eight men and eight women per group). Participants were either exposed to a stressor or a non-stressful control condition after they had recalled neutral and emotional experiences from their past (*react + stress* and *react + control* group, respectively). Another group of participants was exposed to the stressor without prior memory reactivation to control for the possibility that memory was influenced by the stress exposure irrespective of memory reactivation (*stress only* group). Finally, a fourth group of subjects was neither stressed nor did they reactivate autobiographical memories, i.e. they omitted experimental day 1 (*control* group). The critical memory test was given 7 days after day 1 and was basically the same in the four groups. All experiments were carried out in the afternoon between 1 pm and 6 pm.

2.2. Reactivation of autobiographical memories

We used a modified version of the autobiographical memory cueing test (Williams & Broadbent, 1986) to reactivate participants' autobiographical memories. Participants of the *react + stress* and *react + control* groups were presented two positive (happy, interesting), two neutral (concentrated, busy) and two negative adjectives (sad, angry) in randomized order. They were instructed to remember, in as much detail as possible, for each adjective one specific episode, including, e.g. a specific time and specific place, from their own past. In retrospect, participants recalled indeed specific events they had experienced. For example, one participant mentioned for the adjective "sad": "I was sad, when I heard of the death of the German national keeper Robert Enke last Tuesday. It was about 10 pm and I was working on my desk when my sister came to my room and told me that he was dead (...)".

To control for the age of the reactivated memories, participants were asked to remember events that were at least 24 h and at maximum 3 weeks old. There was a time limit of 4 min for each of the six adjectives. After participants had written the events down, they were asked to indicate when each event occurred and to give each memory a title (which should help to refer to the events on experimental day 2).

2.3. Stress protocol

About 10 min after the reactivation of the autobiographical memories, participants in the *react + stress* group were exposed to the socially evaluated cold pressor test (SECPT), a stress protocol that has been described in detail elsewhere (Schwabe, Haddad, & Schachinger, 2008). Briefly, participants were asked to immerse their right hand up to and including the wrist for 3 min into ice water (0–2 °C). During hand immersion they were recorded by a video camera and monitored by a rather cold experimenter. This procedure is known to elicit significant increases in cortisol and autonomic activity (Schwabe, Bohringer, & Wolf, 2009; Schwabe & Wolf, 2009b; Schwabe et al., 2008). Participants in the *react + control* group submerged their right hand up to and including the wrist into warm water (35–37 °C); they were neither monitored nor videotaped.

To assess the success of the stress induction by the SECPT, we measured blood pressure, salivary cortisol and subjective feeling at several time points across the experiment.

2.3.1. Blood pressure

Blood pressure was measured immediately before, during and immediately after the SECPT or control condition with the Dinamap system (Critikon, Tampa, Florida); the cuff was placed at the left upper arm.

2.3.2. Saliva sampling and cortisol analyses

Saliva samples were collected with the help of Salivette (Saarstedt, Germany) collection devices immediately before and immediately after the SECPT or control condition as well as 30 min after the treatment when the cortisol peak was expected (Schwabe et al., 2008). Furthermore, we took one saliva sample before the retention test on experimental day 2 to ensure that groups did not differ in their cortisol levels at test. Free cortisol concentrations were measured from saliva using an immunoassay (IBL, Hamburg).

2.3.3. Subjective assessment

After the SECPT or control condition, participants rated on a scale from 0 ("not at all") to 100 ("very much") how stressful, painful and unpleasant they had experienced the previous treatment.

2.4. Memory test

One week after experimental day 1, subjects in the *react + stress* and *react + control* groups were presented the titles of the autobiographical events they had recalled the week before. They were asked to remember again as many details as possible of the referring event. Participants in the *stress only* and *control* groups completed the autobiographical memory cueing test as did the other two groups on day 1, except that they were instructed to recall events that were at least 1 week and at most 3 weeks old. Again, there was a time limit of 4 min for each adjective and memory title, respectively.

The autobiographical memories were assessed by two independent raters. One point was given for each remembered detail (i.e. for each person, location, time, feeling, etc. that was mentioned; e.g. the above mentioned example memory received eight points). The agreement between the two raters was very high (interrater reliability r_{icc} = .93). Discrepancies were discussed until an agreement was reached. Points were first summed up for each event and then averaged for the positive, neutral and negative events. Because the *control* group omitted experimental day 1, our analyses focused on memory performance on day 2.

2.5. Mood assessment

To control for possible effects of mood-dependent memory (Lewis & Critchley, 2003), participants completed a multidimensional German mood scale (MDBF; Steyer, Schwenkmezger, Notz, & Eid, 1994) at the beginning of experimental days 1 and 2. This questionnaire measures three dimensions of subjective feeling ("elevated vs. depressed mood", "wakefulness vs. sleepiness", "calmness vs. restlessness") on a 5-point rating scale ranging from "not at all" (=1) to "very much" (=5).

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3. Results

3.1. Autobiographical memories on day 1

Participants in the *react + stress* and *react + control* groups remembered on average 13.5 (SEM: 0.6) details per event on experimental day 1. Memory performance was comparable in the two groups and not affected by the emotionality of the memories (all $F < 1$, all $p > .35$, all $\eta^2 \leqslant .03$).

3.2. Stress responses to the socially evaluated cold pressor test

Salivary cortisol and blood pressure responses as well as participants' subjective ratings verified the successful stress induction by the SECPT. All but three participants of the *react + stress* and two participants of the *stress only* groups (mean duration: 81.4 s) kept their hand for the full 3 min in the ice water. These five subjects did not differ in their stress responses from the rest of the stressed participants.

3.2.1. Salivary cortisol

As shown in Table 1, salivary cortisol increased in the *react + stress* and *stress only* groups but not in the *react + control* group (group × time interaction and main effects of group and time: all $F > 7.90$, all $p < .01$, all $\eta^2 > .20$). The three groups did not differ in their cortisol levels before the stress and control manipulation, respectively, whereas the two stress groups had significantly higher cortisol levels 30 min after the treatment (both $p < .05$). The *react + stress* and *stress only* groups did not differ in their cortisol responses to the SECPT (group and group × time effects: both $F < 1.48$, both $p > .23$, both $\eta^2 \leqslant .05$). There were no group differences in salivary cortisol before the critical memory test on day 2 ($F(3, 60) = 0.88$, $p = .45$, $\eta^2 = .04$).

3.2.2. Blood pressure

The exposure to the SECPT caused a significant elevation in systolic and diastolic blood pressure while there was no such elevation in response to the control condition (group × time effects: both $F(4, 88) > 13.03$, both $p < .001$, both $\eta^2 > .37$). Table 1 shows that participants in the *react + stress* and *stress only* groups had significantly higher systolic and diastolic blood pressure than those in the *react + control* group during (LSD post hoc tests, all $p < .05$) but neither before nor after the hand immersion. The two stress groups did not differ in their blood pressure responses to the SECPT (both $F(2, 58) < 1$, both $p > .46$, both $\eta^2 < .03$).

Table 1
Salivary cortisol and blood pressure responses to and subjective ratings of the treatment (stress vs. control condition).

	React + stress	*React + control*	Stress only	Control
Salivary cortisol (in nmol/l)				
Before treatment	8.3 ± 0.9	8.0 ± 0.8	8.3 ± 1.0	–
1 min after treatment	10.5 ± 1.6	7.8 ± 0.9	10.0 ± 1.1	–
30 min after treatment	12.0 ± 1.7*	6.3 ± 0.6	13.0 ± 1.2*	–
Day 2	7.4 ± 1.1	8.3 ± 1.0	8.2 ± 1.1	7.1 ± 0.7
Systolic blood pressure (in mm Hg)				
Before treatment	117.6 ± 4.4	123.8 ± 2.9	121.7 ± 4.2	–
During treatment	131.4 ± 5.4*	120.4 ± 2.5	139.7 ± 5.0*	–
After treatment	116.7 ± 4.3	119.9 ± 2.7	121.2 ± 4.9	–
Diastolic blood pressure (in mm Hg)				
Before treatment	69.2 ± 1.7	70.3 ± 1.9	70.1 ± 2.5	–
During treatment	79.3 ± 1.9*	71.3 ± 1.8	83.6 ± 2.7*	–
After treatment	67.4 ± 1.9	70.5 ± 2.1	68.3 ± 2.5	–
Subjective assessments				
Stressfulness	50.0 ± 7.2*	3.8 ± 1.5	51.9 ± 6.3*	–
Painfulness	65.6 ± 6.2*	1.9 ± 1.4	69.4 ± 5.9*	–
Unpleasanteness	60.6 ± 7.3*	8.8 ± 3.3	64.4 ± 6.1*	–

Note that the participants in the control group omitted experimental day 1 and were neither exposed to the stress nor to the control manipulation. Subjective assessments were given on a scale from 0 ("not at all") to 100 ("very much"). Data represent means ± s.e.m.

* Significantly different from the *react + control* group (post hoc LSD test, $p < .05$).

3.2.3. Subjective ratings

As expected, participants that were exposed to the SECPT experienced the treatment as significantly more stressful, unpleasant and painful than those exposed to the control condition (all $F(2, 45) > 23.65$, all $p < .001$, all $\eta^2 > .51$; Table 1).

3.3. Effects of reactivation and stress on memory performance

Memory performance on experimental day 2 was analyzed with a group × emotionality mixed-design ANOVA. We obtained no main effect of group ($F(3, 60) = 1.29$, $p = .29$, $\eta^2 = .06$) but a significant group × emotionality interaction ($F(6, 120) = 2.25$, $p < .05$, $\eta^2 = .10$). Follow-up tests revealed a significant group effect for neutral ($F(3, 60) = 3.57$, $p = .02$, $\eta^2 = .15$) but not for positive or negative memories (both $F(3, 60) < 1$, both $p > .40$, both $\eta^2 < .05$). As can be seen in Fig. 1, participants that were exposed to stress after the reactivation of events from their past remembered significantly less details of the neutral events than participants of the other three groups (LSD post hoc tests, all $p < .05$), which did not differ (all $p > .64$).

Moreover, there was a significant main effect of memory emotionality ($F(2, 120) = 4.00$, $p = .02$, $\eta^2 = .06$). Neutral memories were less detailed than positive and neutral memories (LSD post hoc test, both $p < .02$). This effect, however, is mainly owing to the relatively low memory performance of the *react + stress* group for the neutral events.

To specifically assess the effect of stress after memory reactivation we compared the memory change from day 1 to day 2 in the *react + stress* and *react + control* groups with a day (day 1 vs. day 2) × group (*react + stress* vs. *react + control*) × emotionality (positive vs. negative vs. neutral) ANOVA. This analysis showed a significant three-way interaction ($F(2, 60) = 9.03$, $p < .001$, $\eta^2 = .23$). Follow-up day × group ANOVAs for the neutral, positive and negative events revealed that the memory for the neutral events was significantly affected by stress after reactivation (day × group interaction effect:

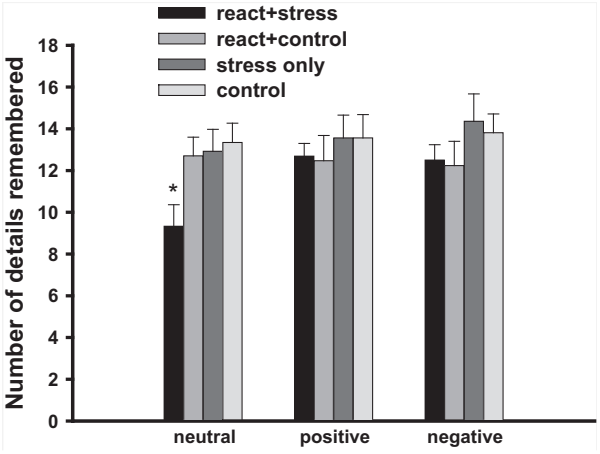

Fig. 1. Number of details remembered for the neutral, positive and negative events on experimental day 2. Participants that were stressed after the reactivation of autobiographical memories remembered significantly less details for the neutral events 1 week later. Memory for emotional events was not affected by stress after reactivation. Data represent means ± s.e.m. *Significantly different from the other three groups (LSD post hoc tests, all $p < .05$).

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$F(1, 30) = 18.51$, $p < .001$, $\eta^2 = .38$) but not the memory for the positive and negative events (both $F(1, 30) < 1$, both $p > .57$, both $\eta^2 < .02$; see Fig. 2).

Importantly, the age of the memories did not differ between groups (main effect group and group × emotionality interaction: both $F < 0.49$, both $p > .80$, both $\eta^2 < .03$). The described memories were on average 14.1 (SEM: 0.5) days old on experimental day 2.

Overall, memories were narrated from the first person perspective suggesting that they reflected indeed experiences of participants' own past. Furthermore, the inspection of the described memories verified that the mentioned events were negative, positive or neutral, according to the presented adjective.

3.4. Mood

MDBF did not differ between groups on experimental days 1 and 2 (all $F < 1.72$, all $p > .16$, all $\eta^2 \leqslant .07$), thus mood effects cannot explain our results.

4. Discussion

Our results demonstrate for the first time that stress can have detrimental effects on the reconsolidation of autobiographical memories in humans. This finding is in line with recent rodent studies showing impairing effects of stress and GCs on memory reconsolidation in mice and rats (Cai et al., 2006; Maroun & Akirav, 2008; Wang et al., 2008). Stress without prior memory reactivation did not affect later memory, thus ruling out any unspecific effects of the stressor. Furthermore, memory reactivation that was not followed by stress did not alter subsequent memory performance.

Although some studies suggest that stress (hormones) might impair memory consolidation for neutral information (Payne et al., 2007; Wolf, 2009), the vast majority of the studies on stress and consolidation demonstrate an enhancing effect of stress on memory consolidation (Beckner, Tucker, Delville, & Mohr, 2006; Cahill et al., 2003; Preuss & Wolf, 2009; for a review see Roozendaal et al., 2009). Thus, together with the previous rodent data, the present findings suggest that the effect of stress on reconsolidation differs from the stress effects on consolidation. This may be seen as another indication that the reconsolidation process after memory reactivation is not an exact copy of the original consolidation process after new learning (Alberini, 2005; Mactutus, Riccio, & Ferek, 1979; see also Nader, Hardt, & Wang, 2005). For instance, using c-fos as a marker of neuronal activation it has been shown that different brain circuits are involved in consolidation and reconsolidation (Tronel & Sara, 2002). Moreover, the molecular mechanisms mediating consolidation and reconsolidation differ at least partly. Memory consolidation is dependent on brain-derived neurotrophic factor (BDNF) but not on the transcription factor zif268 whereas reconsolidation involves zif268 but not BDNF (Lee, Everitt, & Thomas, 2004). In addition, consolidation but not reconsolidation requires expression of the transcription factor C/EBPβ in the hippocampus (Taubenfeld, Milekic, Monti, & Alberini, 2001). Stress and GCs have different effects on the neuroplasticity of different brain areas (Diamond, Campbell, Park, Halonen, & Zoladz, 2007) and exert various effects on transcription factors that are associated with memory processes (Beato & Sanchez-Pacheco, 1996). Understanding how the molecular and neuronal differences between consolidation and reconsolidation contribute to the opposite effects of stress on these two processes is a challenge for future research.

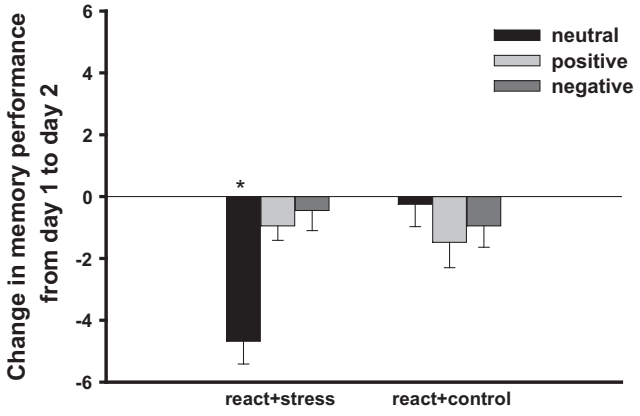

Fig. 2. Changes in the number of remembered details for the neutral, positive and negative events from day 1 to day 2 in the *react + stress* and *react + control* groups. Memory performance declined in all participants from day 1 to day 2. For neutral events, this decline was significantly more pronounced when participants were exposed to stress after memory reactivation on day 1 ($^*p < .05$). There was no influence of stress on the memory change for positive and negative events. Data represent means ± s.e.m.

In addition to the direction of the stress effect, the present data suggest that another difference between stress effects on consolidation and reconsolidation might be the emotionality of the material that is primarily affected. Stress enhances mainly the consolidation of emotionally relevant material (Buchanan & Lovallo, 2001; Cahill et al., 2003), whereas stress impaired in the present study selectively the reconsolidation of neutral memories. One possible explanation for the lack of a stress effect on emotional memory reconsolidation might be that the emotional memories were stronger (i.e. better consolidated; (Christianson, 1992) and therefore less susceptible to reconsolidation effects (Suzuki et al., 2004). However, there are several studies in rodents and humans showing that pharmacological manipulations that lead to significant alterations in stress response systems may impair also the reconsolidation of drug-related, trauma or fear (i.e. strong) memories (Brunet et al., 2008; Cai et al., 2006; Kindt et al., 2009; Wang et al., 2008). Thus, it appears rather unlikely that stress, in general, cannot affect the reconsolidation of emotionally arousing memories.

Alternatively, it could be argued that our brief stressor was not potent enough (e.g., did not cause a large enough cortisol increase) to affect the reconsolidation of emotional memories. The physiological changes observed here in response to the socially evaluated cold pressor test were of course significantly lower than those changes that are usually observed after pharmacological manipulations (e.g. Buchanan & Lovallo, 2001; Kindt et al., 2009). In addition to the mentioned pharmacological studies, however, there is also one recent study that assessed the effect of psychosocial stress on the reconsolidation of neutral and drug-related words in heroin addicts (Zhao, Zhang, Shi, Epstein, & Lu, 2009). This study showed, same as the present study, a disruptive effect of stress on memory reconsolidation. Though, in this study the effect was also found for emotional material (i.e. heroin-related words). Interestingly, the increase in cortisol was in this study more pronounced than in the present study suggesting that stronger stress (hormone) responses may be indeed required to alter the reconsolidation of emotional memories.

Recently, different approaches have been developed for the treatment of trauma memory in post traumatic stress disorder (PTSD) that are aimed at modulating reconsolidation processes by pharmacological manipulation of GC levels. In a rodent model, it has been shown that a fear memory can be persistently attenuated when GC receptors are inactivated after fear retrieval (Tronel & Alberini, 2007). In humans, GCs have been administered before retrieval to attenuate memory reactivation and reconsolidation (for a review see de Quervain & Margraf, 2008). Although these approaches might appear contradictory at first glance, they are based

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on the idea that both very low and very high GC concentrations impair memory processes (Lupien & McEwen, 1997). Future studies are needed to assess whether the stress effect observed here may be mimicked by pharmacological GC elevations. If higher GC levels after reactivation would disrupt also the reconsolidation of emotional memories, the post-retrieval administration of GCs may be another therapeutic approach to PTSD.

In addition, future studies could also measure the vividness or salience of the recalled memories because such factors might well have contributed to the observed differences in the stress effects on neutral and emotional memories. Furthermore, although participants described in the present study mainly specific details, an explicit separation of factual ("external" or semantic) details and those that pertain directly to the event that is described ("internal" or episodic) might be useful (Levine, Svoboda, Hay, Winocur, & Moscovitch, 2002).

In summary, we show here that stress after memory reactivation interferes with memory reconsolidation in humans. Although reconsolidation processes enable us to integrate novel information into existing memory traces (Hupbach et al., 2007), the disruptive effect of stress on this adaptive updating mechanism could prove useful as it could open another door to the treatment of psychiatric disorders, such as PTSD, that are characterized by overly strong memories.

Acknowledgment

We gratefully acknowledge the assistance of Valerie Kinner, Melanie Führer and Florentina Flack during data collection.

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