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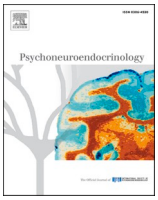

# Reappraisal enhances memory formation for a stressful episode

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ABSTRACT

Emotion regulation strategies have been shown to modify the physiological response to stress, yet whether these strategies can modulate also cognitive responses to stress is largely unknown. A prominent cognitive response to stress is the enhanced memory formation for the stressful event, which is an adaptive mechanism to prepare for similar events in the future. Thus, the present study aimed to investigate whether emotion regulation strategies impact the memory formation for a stressful episode. In a two-day study, participants (n = 124) underwent an enriched stressful episode or a control episode. Critically, before the exposure to the stressor, they were instructed to use a suppression or reappraisal strategy during the stressful episode. One week later, participants completed a memory test for central and peripheral details of this episode. Our results show that reappraisal enhanced not only the cortisol response to the stressor but also the memory formation for central features of the stressful episode. This reappraisal-related boost of memory for the stressor was particularly pronounced in participants' with high working memory capacity. These findings show that reappraisal may not only impact the physiological response to a stressful event but also the cognitive representation of this event in memory.

## 1. Introduction

Stress is ubiquitous in modern societies and may have a major impact on our health and wellbeing. Exposure to stressors triggers a number of physiological response systems that stimulate the release of numerous hormones, peptides and neurotransmitters, including catecholamines and glucocorticoids (Joëls and Baram, 2009; Ulrich-Lai and Herman, 2009). These stress mediators act on the brain, alter affective and cognitive processes and may ultimately contribute to stress-related mental disorders (de Kloet et al., 2005; McEwen, 1998; O'Connor et al., 2021). However, individuals differ substantially in their responses to stressors (Kudielka et al., 2009), and some individuals are more vulnerable to harmful influences of stressful events than others. One relevant source of these individual differences in stress responses may be the individual emotion regulation capacity, i.e., individuals' capacity to exert control over their emotional state (Gross, 1998b). Two major emotion regulation strategies have been in the spotlight of the literature: reappraisal and suppression (Gross and John, 2003; John and Gross, 2004). While reappraisal is an antecedent-focused strategy that aims to change the meaning and impact of the situation that elicits the emotion, suppression is a response-focused strategy directed at the modification of the facial expression of the emotion (Gross, 1998a). Several studies suggested that these emotion regulation strategies have different consequences on positive affect, social functioning, and mental health (Chervonsky and Hunt, 2017; Gross and John, 2003; John and Gross, 2004). Most importantly, there is also evidence linking emotion regulation strategies to the endocrine stress response (Carlson et al., 2012; de Veld et al., 2012; Krkovic et al., 2018; Lam et al., 2009; Lin et al., 2021).

While many studies on emotion regulation and stress reactivity have been correlational in nature, there are also a few studies that manipulated the emotion regulation strategy that individuals employed under stress. These experimental studies suggest that reappraisal enhances the physiological response to stress, including the cortisol response to the stressor (Denson et al., 2014; Jamieson et al., 2013, 2012; Jentsch and Wolf, 2020). Although one might assume that reappraisal as an adaptive emotion regulation strategy should attenuate the stress response (Gross and John, 2003), it has been argued that while long-term reappraisal training reduces the neuroendocrine reactivity to stress (Gaab et al., 2003), a short-term reappraisal manipulation may increase the Hypothalamus-pituitary-adrenal (HPA) axis activation due to increased task effort (Denson et al., 2014). Importantly, previous research on the impact of emotion regulation strategies on stress responses focused

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mainly on the physiological stress response. Few studies reported a beneficial effect of stress reappraisal on academic performance and attentional bias (Jamieson et al., 2010, 2012, 2016). Further, a recent study tested whether reappraisal might attenuate stress effects on memory retrieval but yielded inconclusive results, as this study did not obtain an influence of stress per se on retrieval (Marr et al., 2021). Therefore, whether emotion regulation strategies may also modulate the effects of stress on memory processes remains largely unknown. However, modulatory effects of emotion regulation on stress-induced changes in memory would be of utmost importance because these changes are thought to be an integral part of the adaptation to stressors but may also be a driving force for stress-related psychopathologies (de Kloet et al., 2005; Vogel et al., 2016).

One of the most prominent cognitive responses to stress is the enhanced memory formation for the stressful episode (Joëls et al., 2006; Kalbe et al., 2020; McGaugh, 2015; Sandi and Rose, 1994; Schwabe et al., 2022; Vogel and Schwabe, 2016; Wiemers et al., 2013). This memory enhancement is attributed to the action of catecholamines and glucocorticoids on prefrontal and medial-temporal lobe areas, including the amygdala and hippocampus (Joëls et al., 2006; Roozendaal et al., 2006; Schwabe et al., 2022; Schwabe et al., 2012). The superior memory for stressful events is generally an adaptive mechanism to prepare the organism for similar threatening situations in the future Joëls et al., 2011, 2006; Schwabe et al., 2022; Vogel et al., 2016). However, overly strong memory of a stressful event can be maladaptive and contribute to the aberrant memory in anxiety disorders or post-traumatic stress disorder (PTSD; de Quervain et al., 2017; Pitman et al., 2012). Importantly, stress does not strengthen memory for all aspects of an episode, but it appears to promote in particular the storage of central elements of the stressful episode, whereas the memory for peripheral details may be even reduced (Kalbe et al., 2020; Kensinger et al., 2007; Wiemers et al., 2013). This finding is consistent with a recent meta-analysis suggesting that stress enhances memory encoding only if the encoded information is directly related to the stressor (Shields et al., 2017). Despite the critical relevance of memory formation under stress for both adaptation to stressful events and the development of stress-related mental disorders such as PTSD, it remains completely unknown whether emotion regulation strategies can modify the memory formation for a stressful episode.

Thus, the present study aimed to test whether emotion regulation strategies can alter, in addition to the physiological stress response, the memory formation for the stressful event. To this end, we exposed participants to a stress or control manipulation that was embedded in an enriched episode including several sub-events and contextual details. Critically, before the stress manipulation, participants were instructed to use a reappraisal or suppression strategy during the stressful encounter. One week later, memory for the stressful (or control) episode was tested. In addition, we included a working memory assessment on Day 1 because working memory has been shown to modulate both cortisol reactivity to stress (Lin et al., 2020) and cognitive functions under stress (Otto et al., 2013; Quaedflieg et al., 2019). Since previous research suggested that reappraisal enhances physiological stress responses (Denson et al., 2014; Jamieson et al., 2013, 2012), we hypothesized that reappraisal (but not suppression) would increase sympathetic and neuroendocrine responses to stress, which would then contribute to enhanced memory for (central elements of) the stressful episode (McGaugh, 2015; Schwabe et al., 2022). Moreover, in light of the previously reported association between working memory capacity and stress responses (Lin et al., 2020; Otto et al., 2013), we further hypothesized that baseline working memory would modulate memory formation under stress.

## 2. Methods

### 2.1. Participants

We recruited 124 healthy volunteers (58 males and 66 females; Mean age ± SD: 24.60 ± 4.45 years; Age range: 18 ~ 36 years; Mean BMI ± SD: 22.67 ± 2.77 kg/m$^2$) through online advertisements on a local job portal and flyers on campus and in student dormitories. This sample size was based on an a priori power analysis using the software G*power 3 (Faul et al., 2007), which suggested that a total of 112 participants would be sufficient to detect a medium-sized group × item type interaction effect of $f = 0.2$ in a mixed-design ANOVA with a power of 0.95. We recruited 124 participants to ensure sufficient statistical power after a drop-out rate of up to 10 %. The following inclusion criteria were checked through a standardized screening interview: (1) Command of German on a native speaker level; (2) Women should neither be pregnant nor taking hormonal contraceptives; (3) No history of any neurological or mental disorders; (4) No consumption of nicotine or any illicit drugs; (5) No intake of any prescribed medication. Three participants did not show up for the memory assessment on day 2, thus leaving a final sample of 121 participants for memory analysis. All participants gave their informed consent before participation and received a monetary compensation of 40 €. The study was approved by the ethics committee of the University of Hamburg.

### 2.2. Experimental procedure

In this two-day study, participants came to the laboratory on two afternoons exactly one week apart (Fig. 1). Participants were randomly assigned to one of four groups: non-stress control group (C; 15 female, 16 male), standard stress group (S; 19 female, 12 male), stress-reappraisal group (S-Re; 15 female, 16 male), and stress-suppression group (S-Su; 17 female, 14 male). We did not include control groups with different emotion regulation strategies because the present study focused on how emotion regulation may alter memory formation for a stressful episode and not an effect of emotion regulation per se. Furthermore, in the control condition there was no emotionally relevant situation the emotion regulation could be directed at.

**Day 1.** After participants had provided written informed consent, we measured participants' baseline working memory performance with an n-back task (see Section 2.5.). Thereafter, participants were led to another room and received instructions for the TSST/Control task. Importantly, before the TSST, the two emotion regulation (ER) groups (S-Re and S-Su) also received the instruction to use a reappraisal or suppression strategy (see Section 2.3.). After participants had indicated that they understood the above instructions and had correctly retold the ER strategy they were supposed to use, they entered the TSST room to undergo the TSST/control manipulation (see Section 2.4.). Subjective, autonomic and cortisol stress measures were assessed at several time points before and after the TSST/control manipulation.

**Day 2 (one week after Day1).** Participants first reported on their sleep quality and duration as well as the amount of rehearsal of experimental Day 1, as sleep (Diekelmann and Born, 2010) and rehearsal (Karpicke and Roediger, 2008) have been shown to influence memory formation. We then collected another saliva sample for later cortisol analysis, recorded their blood pressure and pulse, and assessed their subjective mood. Next, participants performed a free recall task and then a recognition task to assess the memory of the Day 1 experience (see Section 2.6). Finally, participants were debriefed at the end of the experiment.

### 2.3. Working memory measurement

Working memory was assessed with a two-level numerical n-back task (Kirchner, 1958). In this task (see Fig. 1), a series of black digits (0−9) appeared one after another on the center of a gray screen.

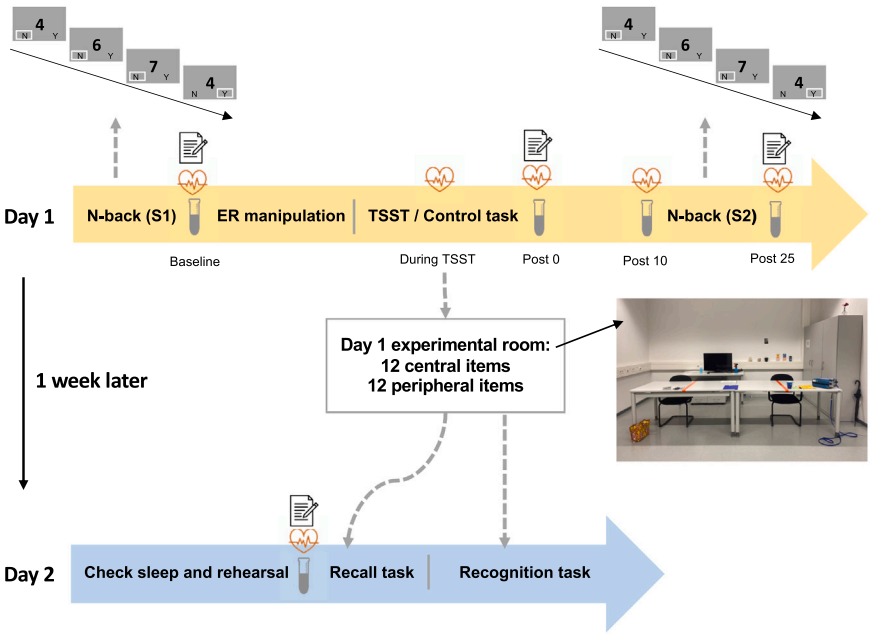


**Fig. 1.** Overview of the experimental procedure. In a two-day study, participants were exposed to the Trier Social Stress Test (TSST) or a control manipulation on Day 1. Both tasks were embedded in an enriched episode. Importantly, some of the participants received additional emotion regulation (ER) instructions related to reappraisal or suppression strategies before they underwent the TSST. Baseline (S1) and post-treatment (S2) working memory were assessed with an n-back task (n = 3 or 4). One week after Day 1, participants completed a free recall task and a recognition task to assess their memory of *central* and *peripheral* items experienced during the treatment episode on Day 1. *Central items* were items that the panel members interacted with during the stress/control procedure, whereas *peripheral items* were items that were not touched by the panel members and therefore were not part of the stress/control procedure.

Participants were instructed to indicate whether each number was identical to the one that appeared *n* (*n* = 3 or 4) trials before or not by pressing the left or right arrow key on a keyboard (left = "no", right = "yes"). To give participants feedback and remind them of the key-response association, a white rectangle appeared around the selected answer if the response was within the response window. In each trial, the stimulus was presented for 500 ms, with a max of 1500 ms response window (including the stimulus presentation time), followed by a 1500 ms inter-trial interval. At the beginning of the baseline session, participants practiced the n-back task until they reached 60% accuracy of both 3-back and 4-back. The formal task consisted of 4 blocks of 30 trials, and the sequence was either 3–4–3–4 or 4–3–4–3, which was counterbalanced across participants. After each block, there was a 13-s break with a "+ " on the screen, followed by a 5-s prompt of the n-back level of the upcoming block. Fifteen minutes after the stress/control manipulation, participants completed a post-stress session of the n-back task, which was identical to the baseline session (without practice phase) and served to test for a potential modulation of the previously reported working memory impairment after stress (Bogdanov and Schwabe, 2016; Schoofs et al., 2009) by reappraisal.

### 2.4. Emotion regulation manipulation

Participants in S-Re and S-Su groups were given the following additional instructions before the TSST, which were designed to be as close as possible to standard reappraisal and suppression instructions (Gross, 1998a):

***Reappraisal:*** "For the upcoming task, please try to keep in mind that this is only an experiment. The scenario is just a temporary situation and has no influence on the rest of your life. Try to think about the upcoming event objectively, by always keeping in mind that it is just an experiment."

***Suppression:*** "For the upcoming task, please try to not show any expression of emotion, but keep a neutral expression on your face. No one should be able to read your emotions from your face. Try to not trigger a strong reaction to the upcoming event, but to react neutrally to it, by always suppressing your emotions and maintaining a neutral facial expression."

Participants were required to correctly summarize the respective emotion regulation strategy in their own words before they proceeded into the TSST room.

### 2.5. Trier social stress test and control manipulation

Participants in the three stress groups (S, S-Re, and S-Su) underwent the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), which is an established protocol to induce psychosocial stress. The TSST included three phases: preparation, free speech, and mental arithmetic. In the preparation phase, participants were asked to prepare for a presentation on why they are the ideal candidate for a job tailored to their interest. Participants were then requested to give this presentation as 5-min free speech without notes. In the subsequent mental arithmetic task (5 min), participants were asked to count backwards in steps of 17 from 2043, and they were asked to restart from 2043 if they made a mistake. During the speech and mental arithmetic tasks, participants stood in front of a TSST panel of two serious and cold experimenters in white lab coats who were introduced as experts in behavioral analysis. Moreover, participants were videotaped and could see their own performance on a big screen placed behind the panel.

Participants in the non-stress control group (C) interacted with a panel of two friendly experimenters. Instead of a stressful mock job interview, participants had a 5-min casual talk about topics of their interest such as hobbies or journeys. Next, they did an easy 5-min counting game together with the two experimenters, skipping numbers including "7" or multiples of "7". Other than in the TSST, experimenters in the control task did not wear white coats and acted friendly, and the participants were not videotaped.

The TSST and control procedure were carried out in the same experimental room that was decorated for the purpose of the Day 2 memory test (see Section 2.6.). We placed 12 *central items* and 12 *peripheral items* in the room. The *central items* were items that the panel interacted with according to a predefined script including specific actions at a specific time, such as sharpening a pencil or binding paper with a stapler (Kalbe et al., 2020). In contrast, *peripheral items* were items that were not touched by the panel, such as a bag and an umbrella.

To assess the effectiveness of the stress manipulation and evaluate group differences in stress responses, we recorded both subjective and physiological measures before and after the TSST/control manipulation. Subjective measures were assessed with a German mood questionnaire (Steyer et al., 1994) before, immediately and 25 min after the TSST/control manipulation. Saliva samples were collected using Salivette collection devices (Sarstedt) before, immediately after, 10 min, and 25 min after the TSST/control task to measure cortisol concentrations.

The samples were stored at − 18 ℃ before cortisol analysis using a luminescence assay (IBL International, Hamburg, Germany). Blood pressure and pulse were measured using a Dinamap system (Critikon Inc.) before, during, immediately after, 10 min, and 25 min after the TSST/control manipulation.

### 2.6. Memory assessment on day 2

Memory was assessed with a free recall and a recognition task. In the free recall task, participants were instructed to recall (and write down) as many items as possible that they saw during the treatment episode on Day 1. Two independent raters counted the number of correctly listed items at the end of data collection. The interrater agreement was high (about 85 %) and discrepant ratings were discussed until an agreement was reached. In the recognition task (see Fig. 1), 24 old items (12 *central* and 12 *peripheral*) from the Day 1 episode and 24 similar new items (12 *central* and 12 *peripheral*) were randomly presented on a computer screen. Each trial began with a fixation cross presented for 1–2 s at the center of the screen, followed by a picture of an old or new item. Participants were instructed to indicate for each item whether they saw the exact same item during the treatment episode on Day 1, by selecting either "very certain old," "certain old," "rather old", "rather new", "certain new", or "very certain new" (Kalbe et al., 2020). The recognition test was self-paced.

### 2.7. Data analysis

For subjective mood measures, seven participants (3C, 1S-Re, 3S-Su) had missing values. For salivary cortisol, there were two missing values at baseline (1 C, 1 S), four missing values at post 0 (1S, 1S-Re, 2S-Su), three missing values at post 10 (1C, 1S, 1S-Su), and three missing values at post 25 (1 S, 2S-Su) due to little saliva. For blood pressure and pulse, one participant (C) had one time point missing during the non-stress control task due to technical errors. In the analysis of baseline working memory, we excluded participants with accuracy below 2.5 SD or reaction time above 2.5 SD across groups, which left 116 participants for the following analysis relevant to baseline working memory.

Subjective and physiological parameters were analyzed with mixed-design ANOVAs with group (C vs. S vs. S-Re vs S-Su) as between-subjects factor and time point of measurement as within-subject factor. For working memory, we focused on accuracy and reaction time data and performed mixed-design ANOVAs with group (C vs. S vs. S-Re vs. S-Su) as a between-subjects factor, and measurement session (baseline vs. post-stress) and task load (n = 3 vs. n = 4) as within-subject factors. For free recall performance, we analyzed the percentage of *central* and *peripheral* items that participants correctly recalled. For recognition performance, we included only the high-confidence responses ("very certain", "certain") when we calculated hits and false alarm rates, in line with a previous study on memory formation under stress from our lab (Kalbe et al., 2020), as high-confidence responses are assumed to be a better indicator of actual memory (Yonelinas, 1994). Recognition accuracy was calculated as difference between hits and false alarm rates (Kalbe et al., 2020). These memory parameters were subjected to a mixed-design ANOVAs with group (C vs. S vs. S-Re vs. S-Su) as between-subjects factor and item type (central vs. peripheral) as a within-subjects factor. Because cortisol is known play a key role in memory formation for stressful episodes (Joëls et al., 2011; Roozendaal et al., 2006; Schwabe et al., 2022; Sandi et al., 1997), we ran also an explorative analysis testing whether the cortisol response (high--responders vs low-responders, based on a median split on the baseline-to-peak difference) would modulate participants' memory for central elements of the treatment episode.

Furthermore, as previous studies suggested that baseline working memory also affects stress responses (Lin et al., 2020) and cognition under stress (Otto et al., 2013; Quaedflieg et al., 2019), we were interested in whether baseline working memory would interact with our treatment on cortisol responses and memory formation. Therefore, we entered baseline working memory (calculated by the average reaction time across 3-back and 4-back loads) as a covariate in mixed-design ANCOVAs with group (C vs. S vs. S-Re vs. S-Su) as between-subjects factor, and time point of measurement as a within-subject factor in the analysis of the cortisol responses and item type (central vs. peripheral) as a within-subjects factor in the analysis of memory performance. High vs. low baseline working memory performance comparison was based on a median split for the average reaction time of the 3-back and 4-back tasks. The number of participants in each subgroup was as follows: C (21 high vs. 10 low); S (14 high vs. 12 low); S-Re (12 high vs. 17 low); S-Su (11 high vs. 20 low).

Data analysis was performed using R (version 4.1.2) with packages afex (1.0–1) and emmeans (1.7.1–1). All reported *p*-values are two-tailed. The significance level was set at 0.05. Greenhouse–Geisser correction was applied in the case of violation of the sphericity assumption. In the post hoc analysis of main significant main or interaction effects, *p*-values were Bonferroni-corrected when indicated ($p_{corr}$).

## 3. Results

### 3.1. Reappraisal is associated with increased cortisol response to stress

As expected, the TSST led to significant increases in subjective and physiological stress parameters (see Table 1). For subjective mood, the mixed-design ANOVA revealed a significant group × time interaction for negative mood ($F(5.55, 208.89) = 9.992$, $p < 0.001$, $\eta_p^2 = 0.210$), restlessness ($F(5.23, 197.08) = 7.347$, $p < 0.001$, $\eta_p^2 = 0.163$), and tiredness ($F(5.85, 220.31) = 5.158$, $p < 0.001$, $\eta_p^2 = 0.121$). Post-hoc comparisons revealed that all three stress groups (S, S-Re, S-Su) showed elevated stress levels immediately after the treatment [bad mood: $t_s > 4.500$, $p_s < 0.001$; restlessness: $t_s > 4.173$, $p_s < 0.001$; tiredness: $t_s > 3.104$, $p_s < 0.01$], without any differences between the three stress groups ($p_s > 0.317$). For sympathetic arousal measures, the mixed-design ANOVA revealed a significant group × time interaction for pulse ($F(5.63, 223.51) = 5.270$, $p < 0.001$, $\eta_p^2 = 0.117$). Post-hoc comparisons revealed that the pulse of the three stress groups was elevated during the TSST compared with non-stress control group ($t_s > 2.227$, $p_s < 0.028$), without differences between the three stress groups ($p_s > 0.230$). For diastolic blood pressure ($F(9.06, 359.30) = 1.94$, $p = 0.045$, $\eta_p^2 = 0.047$), the S-Re and S-Su showed higher scores than the control group during the TSST ($t_s > 2.550$, $p_s < 0.012$), while the S group was not different from the control group ($t = 2.550$, $p = 0.129$). For systolic blood pressure, there were no significant group differences ($F(8.04, 319.04) = 0.090$, $p = 0.544$, $\eta_p^2 = 0.022$). The relative lack of significant group differences in blood pressure is most likely due to the fact that participants of all groups were interacting with other people during the manipulation and in line with previous findings using a similar task version (Wiemers et al., 2013). Moreover, the TSST led also to a significant increase in cortisol, which was not see in response to the control manipulation (group × time interaction: $F(5.19, 197.73) = 7.636$, $p < 0.001$, $\eta_p^2 = 0.166$). Post-hoc comparisons revealed that the TSST significantly elevated cortisol immediately after [S vs. C: $t(115 = 2.500$, $p = 0.014$, $d = 0.777$; S-Re vs. C: $t(115) = 2.759$, $p = 0.007$, $d = 0.857$], 10 min after [S vs. C: $t(115) = 2.587$, $p = 0.011$, $d = 1.109$; S-Re vs. C: $t(115) = 4.559$, $p < 0.001$, $d = 1.954$; S-Su vs. C: $t(115) = 1.887$, $p = 0.062$, $d = 0.816$] and 25 min after the TSST [S vs. C: $t(115) = 1.989$, $p = 0.049$, $d = 0.695$; S-Re vs. C: $t(115) = 4.228$, $p < 0.001$, $d = 1.478$; S-Su vs. C: $t(115) = 2.021$, $p = 0.046$, $d = 0.713$] compared to the control condition, whereas stress groups did not differ from the control group at baseline [S vs. C: $t(115) = 1.647$, $p = 0.102$, $d = 0.323$; S-Re vs. C: $t(115) = 0.002$, $p = 0.999$, $d = 0.000$; S-Su vs. C: $t(115) = 0.369$, $p = 0.713$, $d = 0.073$]. Critically, the results also revealed that in particular reappraisal enhanced the cortisol response (see Fig. 2.), compared to other two stress groups at 10 min after [S-Re

**Table 1**
Physiological and subjective responses to the stress and control manipulations.

| | control | | stress- standard | | stress- reappraisal | | stress- suppression | |
|---|---|---|---|---|---|---|---|---|
| Variables | Mean | SD | Mean | SD | Mean | SD | Mean | SD |
| Pulse | | | | | | | | |
| Baseline | 68.23 | 12.14 | 69.13 | 11.56 | 69.32 | 11.30 | 71.89 | 11.00 |
| During TSST | 84.45 | 21.82 | 96.26* | 20.60 | 102.60*** | 17.92 | 101.60** | 22.24 |
| Post 0 min | 70.18 | 17.50 | 70.86 | 11.36 | 72.76 | 11.60 | 75.02 | 14.02 |
| Post 10 min | 70.98 | 18.08 | 70.76 | 12.06 | 72.10 | 11.25 | 72.89 | 11.02 |
| Post 25 min | 68.05 | 10.21 | 69.90 | 11.01 | 71.42 | 10.86 | 73.03 | 10.08 |
| Systolic blood pressure | | | | | | | | |
| Baseline | 130.33 | 20.06 | 134.52 | 21.79 | 130.29 | 13.46 | 131.92 | 20.06 |
| During TSST | 147.77 | 21.76 | 152.92 | 21.76 | 157.53 | 20.34 | 151.16 | 28.09 |
| Post 0 min | 129.93 | 21.47 | 136.50 | 19.96 | 138.81 | 15.98 | 136.02 | 20.05 |
| Post 10 min | 127.93 | 19.59 | 132.92 | 20.05 | 134.50 | 15.28 | 132.48 | 17.37 |
| Post 25 min | 128.20 | 17.17 | 132.69 | 20.57 | 131.61 | 12.69 | 129.24 | 18.48 |
| Diastolic blood pressure | | | | | | | | |
| Baseline | 69.33 | 11.00 | 70.61 | 9.10 | 71.44 | 7.32 | 69.82 | 8.76 |
| During TSST | 87.12 | 11.12 | 91.92 | 10.44 | 95.13 | 9.12 | 96.34 | 16.90 |
| Post 0 min | 72.90 | 10.41 | 76.24 | 9.63 | 79.18 | 8.07 | 76.63 | 8.18 |
| Post 10 min | 70.20 | 9.08 | 73.58 | 10.81 | 75.32 | 8.52 | 76.55 | 9.09 |
| Post 25 min | 70.97 | 10.72 | 71.94 | 8.07 | 73.37 | 8.55 | 72.60 | 8.01 |
| Salivary cortisol (nmol/l) | | | | | | | | |
| Baseline | 4.43 | 2.88 | 5.60 | 3.15 | 4.43 | 2.89 | 4.16 | 1.87 |
| Post 0 min | 5.01 | 2.98 | 7.82* | 4.78 | 8.11** | 5.76 | 5.76 | 3.25 |
| Post 10 min | 5.05 | 2.95 | 9.06* | 5.60 | 12.12*** | 9.02 | 8.00 | 4.71 |
| Post 25 min | 3.98 | 2.11 | 6.49* | 3.73 | 9.32*** | 7.86 | 6.56* | 3.93 |
| Good vs. bad mood | | | | | | | | |
| Baseline | 32.81 | 6.17 | 33.29 | 5.39 | 32.30 | 4.91 | 32.46 | 5.34 |
| Post 0 min | 34.61 | 4.43 | 26.50*** | 8.80 | 25.90*** | 6.70 | 24.64*** | 7.26 |
| Post 25 min | 34.07 | 5.07 | 31.11 | 6.01 | 28.27*** | 6.87 | 28.68*** | 6.35 |
| Calmness vs. restlessness | | | | | | | | |
| Baseline | 30.61 | 6.15 | 30.71 | 5.95 | 30.20 | 6.00 | 30.04 | 5.24 |
| Post 0 min | 31.55 | 5.26 | 24.04*** | 8.68 | 24.20*** | 6.91 | 23.29*** | 6.37 |
| Post 25 min | 33.45 | 5.03 | 29.96* | 5.62 | 29.23** | 6.63 | 27.96*** | 6.33 |
| Alertness vs. tiredness | | | | | | | | |
| Baseline | 30.13 | 5.77 | 28.61 | 6.00 | 28.60 | 6.95 | 28.21 | 6.17 |
| Post 0 min | 32.48 | 4.64 | 26.36*** | 6.62 | 27.87** | 5.76 | 26.36*** | 6.15 |
| Post 25 min | 30.97 | 5.17 | 26.43** | 7.06 | 24.93*** | 7.21 | 24.11*** | 6.70 |

Note: $^{*}p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ (vs. the control group).

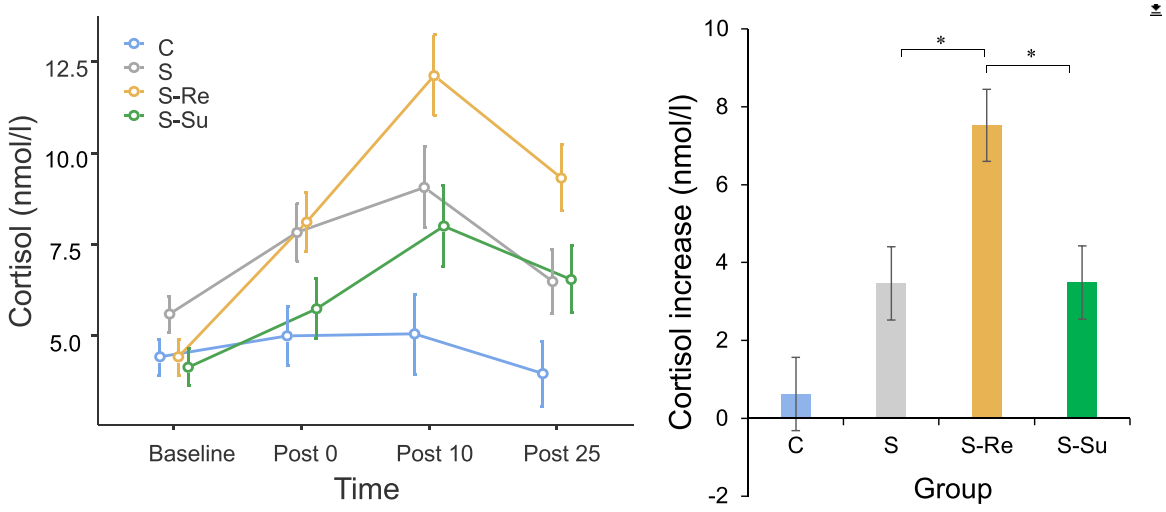


**Fig. 2.** Cortisol responses among four groups. Left panel shows the original salivary cortisol values. Right panel shows the baseline-peak cortisol increases. Data bars show means, and error bars show standard errors. $^{*}p < 0.05$ (vs. S-Re group). [C: control group; S: stress-standard group; S-Re: stress-reappraisal group; S-Su: stress-suppression group].

vs. S: $t(115) = 1.972$, $p = 0.051$, $d = 0.845$; S-Re vs. S-Su: $t(115) = 2.632$, $p = 0.010$, $d = 1.138$] and 25 min [S-Re vs. S: $t(115) = 1.972$, $p = 0.051$, $d = 0.845$; S-Re vs. S-Su: $t(115) = 2.632$, $p = 0.010$, $d = 1.138$] after TSST. Further analysis found that the baseline-to-peak increase was significantly different between the four groups: $F(3, 117) = 9.270$, $p < 0.001$, $\eta_p^2 = 0.192$. Post-hoc tests further revealed that the S-Re group showed a higher cortisol increase than both the S group [$t(117) = 3.075$, $p_{corr} = 0.016$, $d = 0.788$] and S-Su group [$t(117) = 3.059$, $p_{corr} = 0.017$, $d = 0.783$], see Fig. 2 (right panel).

### 3.2. Reappraisal enhances the memory for central features of a stressful episode

On Day 2, the four groups did not differ in terms of salivary cortisol concentration [$F(3, 111) = 0.917$, $p = 0.435$, $\eta_p^2 = 0.024$], blood pressure [systolic: $F(3, 110) = 1.366$, $p = 0.257$, $\eta_p^2 = 0.036$; diastolic: $F(3, 110) = 0.889$, $p = 0.449$, $\eta_p^2 = 0.024$], pulse [$F(3, 110) = 1.257$, $p = 0.293$, $\eta_p^2 = 0.033$], negative mood [$F(3, 111) = 1.886$, $p = 0.136$, $\eta_p^2 = 0.048$], and restlessness [$F(3, 111) = 1.165$, $p = 0.327$, $\eta_p^2 = 0.031$]. Participants of the S-Re and S-Su groups reported less alertness than the control group on Day 2: $F(3, 111) = 3.397$, $p = 0.020$, $\eta_p^2 = 0.084$, but there were no differences among the three stress groups ($p_s > 0.365$). In addition, groups did not differ in sleep quality or duration after the Day 1 experience ($p_s > 0.627$). For rehearsal, they did not differ in how often they thought or talked about the Day1 experience ($p_s > 0.126$), but the three stress groups all reported higher strain after the Day 1 experience than the control group ($p_s < 0.01$), confirming again the successful stress manipulation through the TSST.

In order to analyze the modulatory effect of emotion regulation strategies on memory formation for a stressful episode, the main question of the present study, we focused on the free recall test on Day 2. A group × item type ANOVA on the free recall data revealed a significant main effect of item type, $F(1, 117) = 151.433$, $p < 0.001$, $\eta_p^2 = 0.564$, indicating overall better memory for central items than for peripheral items. More importantly, however, this analysis revealed also a significant group × item interaction, $F(3, 117) = 3.697$, $p = 0.014$, $\eta_p^2 = 0.087$. Follow-up analysis showed that groups differed in the free recall performance for central items, $F(3, 117) = 2.693$, $p = 0.049$, $\eta_p^2 = 0.065$, with only the S-Re group showing significantly better memory than group C (Fig. 3; $t(117) = 2.707$, $p_{corr} = 0.047$, $d = 0.680$). As shown in Fig. 3, the S and S-Su groups tended to show enhanced memory for central features of the treatment episode as well, yet these differences relative to the control group did not survive corrections for multiple comparisons (both $p_{corr} > 0.227$). For peripheral items, however, groups did not differ in their recall performance, $F(3, 117) = 1.965$, $p = 0.123$, $\eta_p^2 = 0.048$.

Considering the significant role of cortisol on memory formation reported by previous studies (Joëls et al., 2011; Roozendaal et al., 2006; Schwabe et al., 2022), we also conducted a cortisol response × item type ANOVA on recall performance. This analysis was based on the whole sample and the number of participants in each group was as follows: C (7 high vs. 23 low); S (16 high vs. 14 low); S-Re (22 high vs. 9 low); S-Su (16 high vs. 14 low). The result revealed a significant interaction between cortisol change level and item type, $F(1, 116) = 5.994$, $p = 0.016$, $\eta_p^2 = 0.049$. Post-hoc comparisons showed that high-responders had higher recall performance than low-responders for central items [Fig. 4; $t(116) = 2.195$, $p = 0.030$, $d = 0.388$], but there were no differences in recall performance for peripheral items [$t(116) = -1.272$, $p = 0.206$, $d = -0.065$]. Notably, when we ran this analysis only in the stress group, there was no significant difference between cortisol high- vs. low-responders ($F(1, 87) = 0.409$, $p = 0.402$, $\eta_p^2 = 0.004$), which might be due to a lack of statistical power.

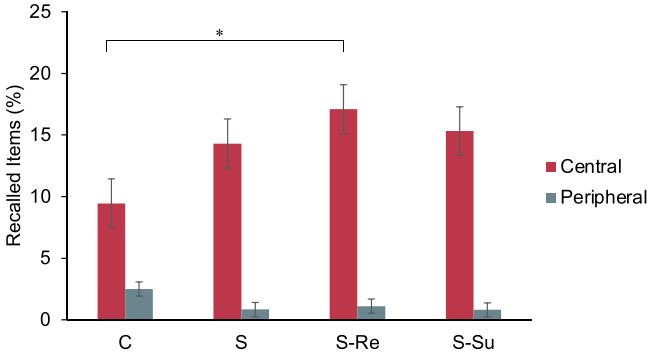


**Fig. 3.** Free recall performance. Data represent means, and error bars show standard errors. *$p < 0.05$. [C: control group; S: stress-standard group; S-Re: stress-reappraisal group; S-Su: stress-suppression group].

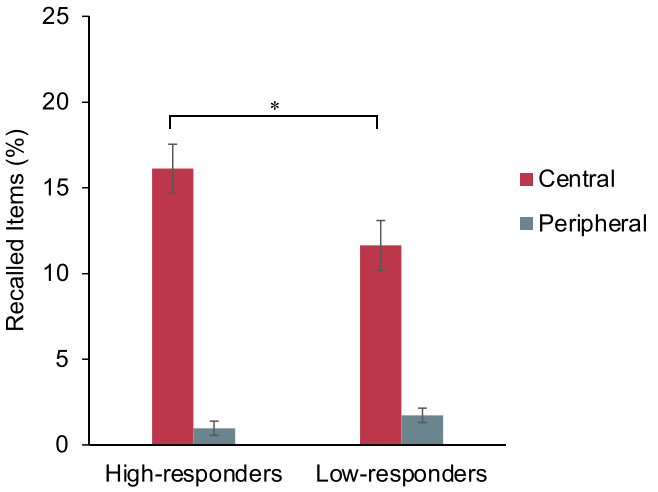


**Fig. 4.** Free recall performance comparison between high-responders and low-responders. Data represent means, and error bars show standard errors. *$p < 0.05$. The number of participants in each subgroup was as follows: C (7 high vs. 23 low); S (16 high vs. 14 low); S-Re (22 high vs. 9 low); S-Su (16 high vs. 14 low). [C: control group; S: stress-standard group; S-Re: stress-reappraisal group; S-Su: stress-suppression group].

Recognition memory performance was overall rather high (see supplementary tables S1 and S2). The group × item type ANOVA on accuracy revealed a higher accuracy for central compared to peripheral items [$F(1, 117) = 174.302$, $p < 0.001$, $\eta_p^2 = 0.598$], there was, however, no interaction between group and item type [$F(3, 117) = 0.205$, $p = 0.892$, $\eta_p^2 = 0.005$]. This pattern of results did not change when focusing not only on high-confidence hits but including all responses, irrespective of confidence (see supplemental material).

### 3.3. Baseline working memory effects on cortisol response and memory

Because previous studies suggested that baseline working memory capacity can modulate the physiological and cognitive response to stress (Lin et al., 2020; Otto et al., 2013; Quaedflieg et al., 2019), we further tested whether participants' baseline working memory capacity affected the cortisol response to and memory formation of the stressor. A group × time × baseline working memory (as a continuous covariate) ANOVA on salivary cortisol revealed a significant main effect of baseline working memory, $F(1, 107) = 14.349$, $p < 0.001$, $\eta_p^2 = 0.118$, as well as a significant baseline working memory × time interaction, $F(1.74, 186.17) = 5.309$, $p = 0.008$, $\eta_p^2 = 0.047$. Further analysis revealed that individuals with low baseline working memory (split by the median) showed higher cortisol increases in response to the treatment, $t(112) = 2.551$, $p = 0.012$, $d = 0.478$.

In a next step, we asked whether baseline working memory capacity was also involved in memory formation for the stressful episode. A group × item type × baseline working memory (as a continuous covariate) ANOVA on recall performance revealed a significant baseline working memory × group interaction, $F(3, 106) = 3.442$, $p = 0.019$, $\eta_p^2 = 0.089$, as well as a trend for a baseline working memory × group × item type interaction, $F(3, 106) = 2.623$, $p = 0.054$, $\eta_p^2 = 0.069$. Follow-up analyses revealed that the group difference was only significant for individuals with high baseline working memory, $F(3, 106) = 2.868$, $p = 0.040$, $\eta_p^2 = 0.075$, but not for individuals with low baseline working memory, $F(3, 106) = 0.166$, $p = 0.919$, $\eta_p^2 = 0.005$. For individuals with high baseline working memory (see Fig. 5), the group S-Re had better recall performance for central items ($F(3, 106) = 3.820$,

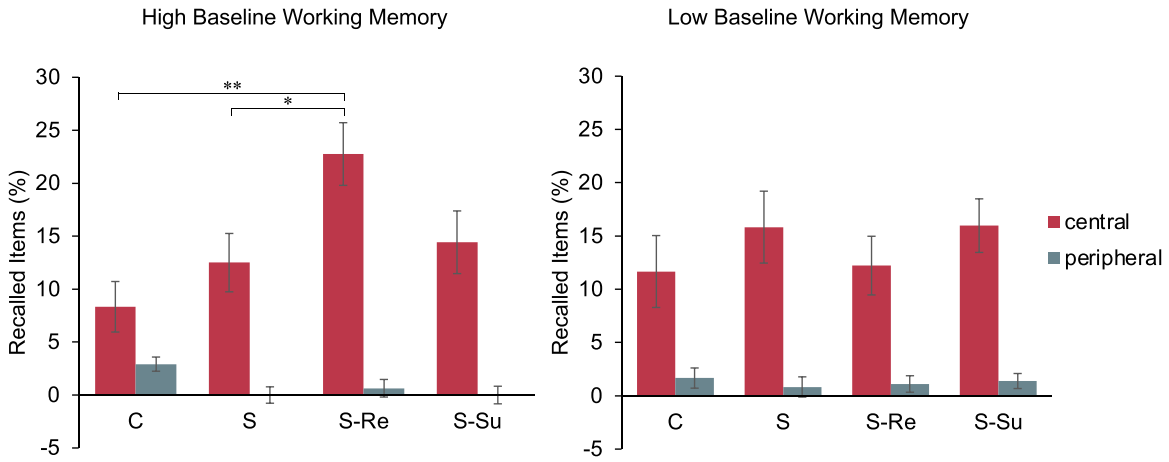


**Fig. 5.** Modulatory effects of baseline working memory on recall performance for the stressful episode. High vs. low baseline working memory performance distinction was based on a median split for the average reaction time of the 3-back and 4-back tasks. Data represent means and standard errors of the mean. $^{*}p < 0.05$; $^{**}p < 0.01$. The number of participants in each subgroup was as follows: C (21 high vs. 10 low); S (14 high vs. 12 low); S-Re (12 high vs. 17 low); S-Su (11 high vs. 20 low). [C: control group; S: stress-standard group; S-Re: stress-reappraisal group; S-Su: stress-suppression group].

$p = 0.012$, $\eta_p^2 = 0.098$) than both group C [$t(106) = 3.351$, $p = 0.001$, $d = 1.231$] and group S [$t(106) = 2.100$, $p = 0.038$, $d = 0.828$; vs. S-Su: $t(106) = 1.557$, $p = 0.122$, $d = 0.650$].

Notably, working memory perse was not affected by stress or emotion regulation strategy (all $p_s > 0.121$, see supplementary table S3).

## 4. Discussion

Previous research linked emotion regulation strategies to the physiological response to stress (Denson et al., 2014; Jamieson et al., 2012). However, whether emotion regulation further modulates fundamental cognitive responses to stress, such as the typically enhanced memory formation for stressful events, remained largely unknown. Thus, the present study aimed to investigate whether emotion regulation strategies, in particular reappraisal (vs. suppression), may modulate the memory formation for the stressful episode. Our findings show that reappraisal not only enhanced the cortisol response to the stressor but also boosted the subsequent recall of this stressor, in particular for its central elements. This reappraisal-related memory enhancement for the stressful episode was specifically observed in individuals with high working memory performance. We further show that those individuals that showed the highest cortisol response to the treatment showed also the best memory for the treatment episode.

Our endocrine data indicates that reappraisal enhances the cortisol response to stress, which is generally in line with the findings of previous studies (Denson et al., 2014; Jamieson et al., 2012). Although an enhanced physiological stress response after reappraisal may be counterintuitive at first glance, there are several potential explanations for this finding. First, since the reappraisal manipulation did not involve an extensive training, reappraisal may have been challenging to participants, requiring them to exert extra task effort during the stress task when using reappraisal. The finding that only reappraisal but not suppression increased the cortisol responses suggests that the increased physiological response reflects particularly an increased effort during the *appraisal* phase and less so during the *response* phase. In addition, this extra task effort could be related to the involvement of cognitive control, which is involved in two kinds of emotion regulation, i.e., attentional control and cognitive change (Ochsner and Gross, 2005). This idea would be consistent with the finding that apart from reappraisal, attentional training also increased the neuroendocrine reactivity to stress (Pilgrim et al., 2014). Moreover, it has been suggested that reappraisal increases self-consciousness (Denson et al., 2014) and that reappraisal of bodily arousal increases perceptions of available resources, promoting experiences of challenge instead of threat (Jamieson et al., 2012). Together, these factors may increase both effort and physiological activation during a stressful event, at least in the short-term. Long-term reappraisal, in turn, has been related to attenuated stress responses (Gaab et al., 2003).

Critically, while our endocrine data corroborate earlier findings that reappraisal may increase the cortisol response to a stressor, we show here for the first time that reappraisal may also modulate a central cognitive response to a stress, the building of strong memories for the stressful encounter. More specifically, the present findings show that the adoption of a reappraisal strategy during a stressful encounter may result in enhanced memory formation for the stressful event, particularly in individuals with high working memory capacity (see below). This result and the finding that higher cortisol responses to the treatment were directly linked to a better subsequent memory thereof are generally in line with the idea that sympathetic arousal in combination with increased glucocorticoid activity – which was particularly high in the stress-reappraisal group – drives the memory enhancement for stressful events (Joëls et al., 2011; Roozendaal et al., 2006; Schwabe et al., 2022). In addition to the increased cortisol response to the stressor, reappraisal may have been also related to an enhanced recruitment of prefrontal circuits that are relevant for reappraisal (Ochsner and Gross, 2005; Wager et al., 2008) but may also promote memory encoding (Blumenfeld and Ranganath, 2007). For instance, an event-related fMRI study found that reappraisal enhanced memory for negative pictures compared with suppression and passive viewing, and this successful encoding during reappraisal was predicted by robust prefrontal cortex activity and co-activation of the left inferior frontal gyrus and medial temporal lobe, including the hippocampus and amygdala (Hayes et al., 2010).

Importantly, the memory boost and its modulation by reappraisal were only observed for central elements of the stressful episode. This finding dovetails with earlier reports that memory is typically enhanced for central but not for peripheral details of a stressful episode (Kalbe et al., 2020; Kensinger et al., 2007; Wiemers et al., 2013). This may be due to a shift in large-scale neural networks towards the salience network that is thought to prioritize emotionally salient information (Hermans et al., 2014, 2011), which may be highly adaptive during a stressful encounter. Notably, the memory enhancement for the stressful episode was observed in a free recall test but, other than in previous studies (Kalbe et al., 2020; Wiemers et al., 2013), not in a recognition test. Recall and recognition represent two distinct memory processes: recall performance relies primarily on the episodic system, whereas recognition replies strongly on both episodic and semantic systems. Furthermore, recall performance is mainly supported by a search

process of recollection, whereas recognition performance is also heavily supported by familiarity (Yonelinas, 2002). Therefore, recognition performance is typically better than free recall performance, as observed in the present study. The more variable performance in the free recall test and its more episodic nature may have rendered this test more sensitive to stress effects than the recognition test.

Our data further show that the link between emotion regulation and memory for the stressful episode was critically modulated by baseline working memory. Specifically, reappraisal enhanced memory for central items only in individuals with high baseline working memory capacity. This result extends previous findings suggesting that high working memory capacity buffers the stress-induced shift towards more inflexible responses (Otto et al., 2013) by showing that high working memory capacity may further be beneficial for the enhancing effects of stress on cognitive functions. Working memory is typically associated with general intellectual and executive capacities (McCabe et al., 2010) as well as prefrontal cortex functioning (Braver et al., 1997; Curtis and D'Esposito, 2003), which in turn are critically involved in reappraisal (Ochsner and Gross, 2005). Thus, it might be argued that higher baseline working memory capacity may have generally had beneficial effects on encoding processes and the implementation of the reappraisal strategy, resulting in the reappraisal-related increase in memory formation for the stressor specifically in individuals with high working memory capacity. At this point, it is important to note that our emotion regulation included a rather short and simple instruction. Future studies could test whether more detailed instructions or explicit training in the emotional regulation strategy reduces the modulatory influence of individual working memory capacity on the effect of reappraisal on the response to a stressor.

Notably, individuals with lower working memory showed a more pronounced cortisol response to the stressor, which suggests that these individuals experienced the performance-related stressor elements (free speech, mental arithmetic) as more demanding and more stressful. Moreover, this finding shows that an increased cortisol response per se is not sufficient for the enhanced memory formation. The increase in cortisol was delayed and only present when the stressful episode was already over. Hence, cortisol could only affect the consolidation of the previously encoded material. We assume that individuals with lower working memory capacity may have encoded less information during the stressful episode than those with higher working memory capacity, thus leaving less material that could subsequently benefit from the enhancing effects of cortisol on consolidation. At this point, it is also important to note that, in contrast to other studies (Bogdanov and Schwabe, 2016; Schoofs et al., 2009), we did not observe a stress-induced impairment in working memory performance or a modulation thereof by the employed emotional regulation strategy. The absence of such a stress-induced working memory impairment might be due to several reasons, such as the time lag between the stress manipulation and the working memory assessment, the strength of the cortisol response to the acute stress, and the working memory load itself, as previous studies on the impact of stress on working memory also reported enhancement or no-effect outcomes (Oei et al., 2006; Qin et al., 2009; Smeets et al., 2006).

Finally, our study included healthy young adults, many of them being university students. This raises the question of the generalizability of the results to the broader population. For instance, there is evidence that working memory declines with age (Gajewski et al., 2018), which might be relevant for the present results because we observed that the reappraisal effect on memory was modulated by working memory capacity. Moreover, the current study excluded women taking hormonal contraceptives due to the potential influence of hormonal contraceptives on stress responsivity. However, given the fact that many women in the general population are using hormonal contraceptives, this methodological choice might also affect the generalizability of the present results. Future studies are requested to test the influence of reappraisal on physiological and cognitive responses to stress in samples that are more representative of the general population to assess the generalizability of the present findings.

To conclude, we show here that reappraisal enhances not only the cortisol response to a stressor but also the memory formation for the stressful episode, in particular in individuals with high working memory capacity. These findings significantly extend previous research on emotion regulation strategies by showing that these strategies modify not only the physiological response to a stressful event but also its cognitive representation in memory. Recent data show that post-encoding arousal may reverse the systems consolidation process and hence promote the long-term vividness of emotional memories (Atucha et al., 2017; Krenz et al., 2021). In light of these data, a key question associated with the present findings relates to the nature of the enhanced memory for the stressful episode. Does reappraisal during a stressful episode result in a more detailed memory that includes also contextual details or in a vivid but more generalized memory of the stressful event? Understanding the nature of the reappraisal-related memory enhancement for a stressful event may have relevant implications for fear- or trauma-related disorders that are characterized by aberrant memory for emotionally arousing events.

## Conflict of interest

The authors have no conflict of interest to disclose.

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## Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.psyneuen.2022.105924.

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