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Stress-induced cortisol responses, sex differences, and false recollections in a DRM paradigm

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Abstract

The current studies investigated whether acute stress potentiates false recollections (so-called ''false memories'') in a Deese–Roediger–McDermott (DRM) paradigm, and whether sex differences modulate these effects. Participants were assigned to either a stress (trier social stress test) or a control group. Subsequently, they were subjected to DRM word lists and probed for recall and recognition. Results showed no differences between the stress and control group on measures of false recollections (Study 1; $N = 60$). Even though correct recall was impaired by acute stress, there were no differences between high or low cortisol responders and controls on false recall or recognitions rates (Study 2; $N = 92$). These results suggest that cortisol responses do not directly potentiate false recollections. Neither in Study 1 nor in Study 2 did we find any evidence that the effects of cortisol on false recollections are different in men and women, although there was an indication that independent of stress men produced more commission errors.

Keywords: Acute stress; Cortisol; Sex differences; False recall; False recognition

1. Introduction

Using a large number of different settings and paradigms, so-called *false memories* have been shown to be fairly easy to elicit and bear clinical as well as legal implications. One paradigm intended to elicit false recall and recognition 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, snooze, doze, nap). After the list has been presented, participants are given a free recall task. Subsequently, a test probing for recognition of presented words (e.g., dream), non-presented unrelated distracter words (e.g., rain), and a semantically related non-presented word termed the ''critical lure'' (in this case the word sleep) follows. Typically, people often falsely recall and/or recognize semantically related but non-presented words (i.e., the critical lures) as having been presented to them in the list learning task (e.g., McDermott, 1996; Roediger and McDermott, 1995; Roediger et al., 1996; Roediger et al., 2001). Thus, the DRM paradigm has the capacity to reliably elicit striking memory illusions (see, for further examples, Blair et al., 2002; Roediger and McDermott, 2000).

Recent findings from Payne and co-workers (Payne et al., 2002) suggest that stress may increase false recognition rates in the DRM paradigm. After they were exposed to the trier social stress test (TSST; Kirschbaum et al., 1993) or a filler task, participants 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. Payne et al. (2002, p. 5) concluded that ''When stress disrupts hippocampal and/or prefrontal cortex processing, false recognition increases dramatically. [. . .] Whatever the precise localisation of stress effects on false recognition, this study has demonstrated that moderate psychological stress renders subjects unable to distinguish between ''true'' and ''false'' memories in the DRM paradigm.'' This conclusion implies that people under stressful circumstances are more vulnerable to specific memory distortions, due to secretion of glucocorticoid stress hormones.

In line with animal research (e.g., de Kloet et al., 1999; McGaugh and Roozendaal, 2002), studies in humans have shown that acute glucocorticoid administration can have

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enhancing as well as disruptive effects on memory, depending on several modulatory variables (for reviews, see Het et al., 2005; Lupien and Lepage, 2001; Wolf, 2003). Furthermore, glucocorticoids are known to facilitate memory formation (e.g., Buchanan and Lovallo, 2001) while impairing retrieval (e.g., Wolf et al., 2004). Also worth mentioning here is that recent work suggests that emotional valence might influence the memory effects glucocorticoids can have (e.g., Kuhlmann et al., 2005; Tops et al., 2003; Wolf et al., 2004).

Animal (e.g., Conrad et al., 2004; Luine, 2002; Wood and Shors, 1998; Wood et al., 2001) as well as human studies have found evidence for sex differences in the effects of stress hormones (e.g., cortisol) on memory functioning. For example, Seeman et al. (1997) found that higher basal levels of cortisol were associated with impaired memory performance among elderly women, but not among men. In line with these results, Wolf et al. (1998) noted that exposure to the TSST following dehydroepiandrosterone (DHEA) treatment significantly impaired memory performance in elderly women, but not in elderly men. In a follow-up study, Wolf et al. (2001) found that within a group of young adults exposed to the TSST, cortisol increases were negatively correlated with memory performance. Further analysis, however, revealed that this effect was carried by a strong negative correlation in men while no such correlation was found among women.

Taken together, human studies suggest that stress-induced cortisol increases may have a corrupting effect on memory performance and that sex may modulate the effects of stress on memory, but that the precise form that this modulation may take is far from clear. Looking at potential sex differences in memory performance after stress is important given the mixed results in this domain of research. With this in mind, the current studies were carried out. More specifically, using equal numbers of men and women distributed over stress and control groups, we sought to replicate the results of Payne et al. (2002). Furthermore, we examined whether the modulating effect of sex on memory functioning also translates into commission errors during free recall (i.e., falsely recalling non-presented words not related to the study words). Additionally, we obtained objective cortisol sampling data to investigate whether there is an interaction between sex, stress-induced cortisol responses, and false recall and recognition of the critical lures following a DRM paradigm.

2. Study 1

2.1. Methods

2.1.1. Participants

Our sample consisted of 60 young healthy undergraduate students (30 men and 30 women). Their mean age was 19.91 years (S.D. = 3.32). Participants were asked whether they suffered from any cardiovascular diseases, endocrine disorders, or asthma. If so, they were excluded from the study. In Study 1 no attempt was made to control for menstrual cycle or the use of oral contraceptives. All 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 given a small financial compensation (€ 10; approximately US$ 12.5 dollars) for completing the experiment.

2.1.2. Materials

2.1.2.1. Profile of mood states. Subjective stress was measured using the anger–hostility and tension–anxiety subscales of the profile of mood states-short form (POMS; McNair et al., 1992). The POMS is a self-report measure that is widely used as a measure of typical and persistent mood reactions to current life situations. Participants indicate to what extent they agree with adjectives describing their current mood or feelings on 5-point scales (anchors: 0 = not at all, 4 = extremely). Adjectives include "annoyed", "angry", and "grumpy" for the subscale anger–hostility and "nervous", "tensed", and "panicky" for the tension–anxiety subscale. The POMS has excellent psychometric properties (see for example, Lezak, 1995; McNair et al., 1992; Shacham, 1983). The present experiment used a Dutch version of the POMS that has been proven to be valid and reliable (de Groot, 1991; Wald and Mellenbergh, 1990). For practical consideration and following Van Honk et al. (2003), we did not use the total POMS.

2.1.2.2. Deese–Roediger–McDermott paradigm (DRM). We used a version of the DRM paradigm (Roediger and McDermott, 1995) in which participants were read out 12 semantically related word lists. After each list they were given a free recall test. After all lists had been presented, participants were probed for recognition memory using a 72 item recognition task consisting of 36 studied words and 36 non-studied words. Non-studied words included 12 critical lures and 24 unrelated distracter words. Participants had to indicate whether the words were presented to them ("old") or not ("new"). Five dependent measures were derived from the DRM paradigm: (1) the number of commission errors (i.e., confabulated extra list words not related to the study words) on the free recall task, (2) proportion correct recall of presented words, (3) proportion falsely recalled critical lure words, (4) proportion correct recognition (i.e., recognizing presented words as "old" and recognizing distracter words as "new"), and (5) proportion falsely recognized critical lures (i.e., non-presented critical lures judged as "old").

2.1.2.3. Trier social stress test (TSST). The trier social stress test (Kirschbaum et al., 1993) is a psychosocial challenge test which basically consists of a 5 min preparation period, a 5 min free speech, and a 5 min mental arithmetic task in front of an audience while being videotaped. The TSST is a valid and reliable procedure to induce physiological stress responses in children, young as well as elderly adults (e.g., Kirschbaum et al., 1992; Kudielka et al., 2004a,b). In a recent meta-analysis, the TSST was found to provoke the most robust physiological stress responses (i.e., cortisol stress responses) relative to various other laboratory stress tasks (Dickerson and Kemeny, 2004). The current study closely followed the TSST protocol as described by Kirschbaum et al. (1993).

2.1.3. Saliva sampling and biochemical analyses

Cortisol data were obtained using cotton Salivettes (Sarstedt®, Nümbrecht, Germany). The uncentrifuged saliva samples were stored at −40 °C immediately upon collection. Salivary free cortisol levels were determined in duplicate by direct radioimmunoassay (RIA; University of Liège, Belgium), including a competition reaction between ^{125}iodohistamine-cortisol and anti-cortisol serum made against the 3-CMO–BSA conjugate. After overnight incubation at 4 °C of 100 μl of saliva, separation of free and antibody-bound ^{125}iodohistamine-cortisol was performed via a conventional 'second antibody' method. In order to reduce sources of variability, all four samples taken from each participant (see below) were analyzed in the same assay. Mean intra- and inter-assay coefficients of variation were less than 4.3 and 8.3%, respectively.

2.1.4. Design

Participants were quasi-randomly assigned to one of two groups. Half of the participants (i.e., 15 men and 15 women) were exposed to the TSST (Kirschbaum et al., 1993) serving as the stress group, while the other half were assigned to a control group that included a filler task. The two groups did not differ with respect to age (stress group $M = 19.97$ years, S.D. = 4.20; control group $M = 19.86$ years, S.D. = 2.18). Thus, the experiment was designed according to a 2 (group: stress versus control) × 2 (sex: men versus women) set-up.

2.1.5. Procedure

Experimental sessions were run between 14 and 17 h when basal cortisol levels are low and stable. Participants were tested individually. To allow for objective-controlled cortisol sampling all participants were deprived of food, drinks, smoking, and heavy exercise at least one hour prior to the test phase. After arrival in the laboratory, they were informed about the memory tests and subsequently signed a consent form. A first cortisol sample (pre-stress) was then obtained using a Salivette (Sarstedt®, Nümbrecht, Germany) and the POMS was administered a first time. In order to eliminate anticipatory stress reactions (e.g., Kirschbaum et al., 1992) which could affect pre-stress cortisol measurement, participants were told about the stress or filler task they would have to perform subsequently after the pre-stress cortisol measurement had been obtained. Participants in the stress group were exposed to the TSST, while the control group was given a filler task that consisted of filling out some questionnaires and playing a computer card game or minesweeper. TSST and filler task had a similar duration.

Subsequent to the TSST or filler task, a second cortisol sample ($t + 20$) was obtained and the POMS was again administered. Afterwards, participants were exposed to a DRM task consisting of 12 DRM lists and free recall tests followed by a recognition task. A third cortisol sample ($t + 35$) was taken after the sixth DRM list (i.e., 15 min after the TSST) and a fourth and final sample ($t + 50$) was obtained at the end of the test session. All memory measures were completed after about 40 min and total time of the entire session did not exceed 1 h. Participants were debriefed, paid, and thanked for their participation.

2.1.6. Statistical analyses

A 2 (group) × 2 (sex) × 2 (time: pre- versus post-manipulation) analysis of variance (ANOVA) with time as repeated factor was used to evaluate feelings of subjective stress (POMS). Cortisol responses were analyzed using a 2 (group) × 2 (sex) × 4 (time: pre-stress versus $t + 20$ versus $t + 35$ versus $t + 50$) ANOVA, with cortisol sample as repeated factor. Additionally, delta increases in cortisol (i.e., cortisol responses) defined as peak cortisol level ($t + 20$, $t + 35$, or $t + 50$) after the TSST or filler task minus pre-stress cortisol level were computed for each participant individually. Delta responses were analyzed using a 2 (group) × 2 (sex) ANOVA. A responder rate of participants showing a cortisol increase larger than 2.5 nmol/l (see, for example, Kirschbaum et al., 1993, 1996; Schommer et al., 2003), which is thought to reflect a cortisol secretory episode (Van Cauter and Refetoff, 1985), was calculated. For each dependent measure a 2 (group) × 2 (sex) ANOVA was employed. Finally, for participants exposed to the TSST Spearman rho correlations were computed between false recall and recognition rates and delta cortisol increases. When sphericity assumptions were violated, Greenhouse-Geisser corrected p-value are reported. Alpha was set at 0.05 unless specified otherwise, and adjusted (Bonferroni) for multiple comparisons where necessary.

2.2. Results

2.2.1. Manipulation check

Table 1 shows sum scores on the anger–hostility and tension–anxiety POMS subscales indicating feelings of subjective stress. As expected, a significant group × time interaction was found [$F(1, 54) = 12.25$; $p = 0.001$] for the anger–hostility subscale. No other main or interaction effects were found. For the tension–anxiety subscale, ANOVA yielded significant effects of time [$F(1, 54) = 23.87$; $p < 0.001$], group [$F(1, 54) = 12.31$; $p = 0.001$], and the anticipated group × time interaction [$F(1, 54) = 70.42$; $p < 0.001$]. All other main or interactive effects remained non-significant. Both ANOVA's indicate that men as well as women in the stress group experienced more stress than did control participants after the TSST or filler task.

2.2.2. Pre-stress cortisol analyses

Two participants indicated having violated the non-smoking/eating/drinking prohibition and were therefore excluded from the data set, leaving 14 women and 15 men in the stress group and 15 women and 14 men in the control group. Stress and control group did not differ with regard to pre-stress cortisol levels (means being 3.57 nmol/l; range: 1.65–7.06 nmol/l; and 4.37 nmol/l; range: 1.30–10.04 nmol/l, respectively).

2.2.3. Cortisol stress responses

As anticipated, the ANOVA showed a significant main effect of time [$F(3, 162) = 6.49$; $p = 0.008$] and a significant group × time interaction [$F(3, 162) = 9.76$; $p = 0.001$], in the absence of additional significant main or interaction effects involving sex, time, or group. The group × sex interaction just fell short of being significant [$F(1, 54) = 4.23$; $p = 0.054$]. Increases in cortisol levels throughout the experiment can be seen in Fig. 1.

Men in the stress group showed a mean delta increase in salivary free cortisol of 4.61 (S.D. = 7.14) nmol/l, while women showed a mean increase of 1.53 (S.D. = 2.12) nmol/l. Control participants showed minor to no increase in response to the filler task, mean increases being −0.16 (S.D. = 0.93) nmol/l for men and 0.24 (S.D. = 0.85) nmol/l for women. An ANOVA on delta increases in cortisol yielded a main effect of group [$F(1, 54) = 9.06$; $p = 0.004$]. No other main or interaction effects were detected. Eleven out of 29 participants (i.e., 38%; eight men and three women) could be classified as showing a straightforward cortisol response.

2.2.4. Memory performance

Descriptive statistics for the DRM recall task and the DRM recognition task are shown in Table 2. ANOVA showed a main

Table 1
Study 1 sum scores on subscales anger–hostility and tension–anxiety of the profile of mood states before (initial) and after (post-test) experimental manipulation or control filler task for men and women separately

	Men/control ($n = 14$)	Women/control ($n = 15$)	Men/stress ($n = 15$)	Women/stress ($n = 14$)
Initial anger–hostility	0.79 (0.89)	0.60 (1.59)	0.27 (0.80)	0.57 (1.09)
Post-test anger–hostility[a]	0.21 (0.43)	0.40 (0.74)	1.40 (1.99)	1.86 (2.21)
Initial tension–anxiety	5.57 (2.90)	5.53 (2.00)	5.00 (2.30)	4.64 (2.17)
Post-test tension–anxiety[a]	3.43 (2.53)	5.27 (2.15)	9.27 (2.28)	9.50 (4.40)

Standard deviations are given in parentheses.
[a] Stress > control, $p < 0.01$.

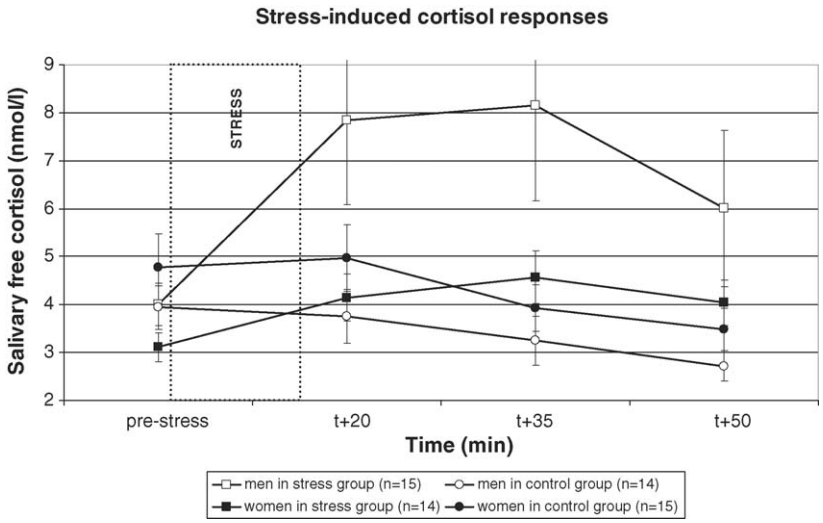

Fig. 1. Study 1 mean free salivary cortisol levels (nmol/l) in the stress and control group for men and women separately. Data points indicate cortisol levels before stress/filler manipulation (pre-stress), immediately after the stress (TSST) or filler task ($t + 20$), 15 min after stress or filler task ($t + 35$), and after testing was completed ($t + 50$).

effect of sex [$F(1, 54) = 4.27$; $p = 0.044$] and a significant group × sex interaction [$F(1, 54) = 4.59$; $p = 0.037$], but no main effect of group for the number of commission errors in the free recall task. Post hoc tests showed that the interaction effect was due to men making more commission errors, but only so in the control group [$t(27) = -2.60$; $p = 0.015$]. There were, however, no significant main effects or interactions involving sex or group for proportion correct recall, proportion of falsely recalled critical lure words, proportion correct recognition, or proportion of falsely recognized critical lures. Delta cortisol increases were not significantly related to false recall ($r = -0.08$) or false recognition ($r = -0.23$) rates among participants exposed to the TSST.

2.3. *Discussion*

The main results of Study 1 can be summarised as follows. First, we replicated the standard effect obtained in numerous other DRM studies (e.g., Roediger and McDermott, 1995; Roediger et al., 1996; Stadler et al., 1999) in that both participants in the stress and the control group exhibited false recall and/or recognition of the critical lure. Second, false recall and false recognition rates did not differ between the stress and the control group. To some extent, this latter finding contradicts previous work of Payne et al. (2002), who found elevated false recognition rates in participants exposed to the TSST. One possible explanation for these divergent findings is that in our study stress responses in terms of cortisol remained at the low end of the continuum. It may well be that false recollections in a DRM paradigm are heightened only when cortisol levels are substantially elevated. Indeed, studies using acute psychosocial stressors like the TSST to activate the hypothalamus–pituitary–adrenal (HPA) axis have noted differential effects of stress on memory performance for high and low cortisol responders (e.g., Elzinga and Roelofs, 2005; Takahashi et al., 2004). Also worthy of note is that Kirschbaum et al. (1999) found that for women, phase of menstrual cycle plays an important role in the responsiveness to acute psychosocial stressors. No attempt was made to control for menstrual cycle in Study 1. Keeping in mind the limitations of Study 1 we conducted a second study that sought to further delineate the effects of acute cortisol responses on rates of false recall and recognition in a DRM paradigm.

3. Study 2

Study 2 increased the sample size so as to include 34 men and 34 women in the stress group. This gave us the opportunity

Table 2
Study 1 mean number of commission errors, proportion correct recall, and proportion false recall (i.e., critical lure words) on the free recall task and proportion correct (i.e., "old" words recognized as "old" and "new" words recognized as "new") and false (i.e., incorrectly recognizing critical lures as "old") recognition for men and women in the stress and control group

	Men		Women	
	Control ($n = 14$)	Stress ($n = 15$)	Control ($n = 15$)	Stress ($n = 14$)
Number of commissions[a]	5.14 (4.45)	2.80 (2.86)	1.93 (1.71)	2.86 (1.79)
Proportion correct recall	0.69 (0.11)	0.68 (0.07)	0.70 (0.08)	0.68 (0.05)
Proportion false recall	0.42 (0.18)	0.38 (0.18)	0.32 (0.19)	0.41 (0.19)
Proportion correct recognition	0.76 (0.04)	0.75 (0.05)	0.73 (0.05)	0.76 (0.02)
Proportion false recognition	0.69 (0.25)	0.62 (0.28)	0.61 (0.25)	0.70 (0.22)

Standard deviations are given in parentheses.
[a] Men in control group > men in stress group, $p < 0.05$.

to differentiate between high cortisol and low cortisol responders whilst maintaining a reasonable number of participants per group. Furthermore, only women in their late luteal phase of the menstrual cycle were included in Study 2.

3.1. Methods

3.1.1. Participants

Ninety-two healthy undergraduate students (46 men and 46 women) participated in Study 2. Their mean age was 19.74 years (S.D. = 1.87). Participants were excluded in case they suffered from any cardiovascular diseases, endocrine disorders, or asthma, or used any medication. All 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 given a small financial compensation (€ 10; approximately 12.5 US$ dollars) for completing the experiment. As cortisol responses are influenced by menstrual cycle, only women who self-reportedly were in their luteal phase (i.e., days 21–25 of the menstrual cycle) were included. During the late luteal phase stress-induced cortisol responses of women appear to be similar to those of men (Kirschbaum et al., 1999).

3.1.2. Materials

Materials (i.e., POMS and TSST) used were the same as those employed in Study 1. The DRM task differed somewhat from Study 1 in that Study 2 relied on eight rather than 12 DRM lists, which had to do with practical considerations. A 48-item recognition task was construed analogous to the recognition task used in Study 1. Saliva sampling and biochemical analyses were performed similar to those in Study 1.

3.1.3. Design

Participants were assigned to one of two groups. Thirty-four men and 34 women served as the stress group and underwent the TSST, while 12 men and 12 women were assigned to a control group that included a filler task. The two groups did not differ with respect to age (stress group $M = 19.69$ years, S.D. = 1.75; control group $M = 19.87$ years, S.D. = 2.23). Thus, as was the case in Study 1, Study 2 was designed according to a 2 (group: stress versus control) × 2 (sex: men versus women) set-up.

3.1.4. Procedure

Study 2 basically followed the procedure of Study 1 (cf. supra). In Study 2, the filler task consisted of reading a neutral text. The POMS was administered only once (i.e., after the TSST or filler task). All memory measures were completed after about 40 min and total time of the entire test session never exceeded 50 min.

3.1.5. Statistical analyses

A 2 (group) × 2 (sex) ANOVA was used to evaluate feelings of subjective stress (POMS). Cortisol levels were analyzed as in Study 1. Delta cortisol increases were computed and analyzed analogous to Study 1. We then looked at high responders (i.e., a delta increase in cortisol > 2.50 nmol/l; see Study 1) versus low responders versus controls. For each dependent measure, a 3 (responder group: high responders versus low responders versus controls) × 2 2 (sex) ANOVA was employed. Spearman rho correlations between false recall and recognition rates and delta cortisol increases were calculated for participants in the stress group. Greenhouse-Geisser correction was used where appropriate. Alpha was set at 0.05 and adjusted (Bonferroni) for multiple comparisons where necessary.

3.2. Results

3.2.1. Manipulation check

Mean scores on the anger–hostility and tension–anxiety POMS subscales indicating levels of subjective stress are shown in Table 3. Main effects of group were found for the anger–hostility [$F(1, 88) = 16.11$; $p < 0.001$] as well as the tension–anxiety [$F(1, 88) = 26.42$; $p < 0.001$] subscale scores, with participants in the stress group indicating feeling more stressed than their control counterparts. For both anger–hostility as well as tension–anxiety, no other main or interaction effects were found.

Table 3
Study 2 sum scores on subscales anger–hostility and tension–anxiety of the profile of mood states for men and women separately

	Men/control ($n = 12$)	Women/control ($n = 12$)	Men/stress ($n = 34$)	Women/stress ($n = 34$)
Anger–hostility[a]	0.17 (0.39)	0.25 (0.62)	3.53 (3.87)	2.32 (2.56)
Tension–anxiety[a]	3.25 (1.81)	5.08 (2.23)	8.12 (3.22)	7.68 (3.44)

Standard deviations are given in parentheses.
[a] Stress > control, $p < 0.01$.

3.2.2. Pre-stress cortisol analyses

None of the 92 participants indicated having violated the non-smoking/eating/drinking exclusion criteria. There were no differences between the stress and control group with regard to pre-stress cortisol levels, means being 7.40 nmol/l (range: 1.76–25.44 nmol/l) and 5.86 nmol/l (range: 2.65–25.51 nmol/l), respectively).

3.2.3. Cortisol stress responses

As expected, we found significant main effects of time [$F(3, 264) = 7.49$; $p = 0.001$] and group [$F(1, 88) = 7.02$; $p = 0.01$], as well as a significant group × time interaction [$F(3, 264) = 5.94$; $p = 0.002$]. No other main or interaction effects involving sex, time, or group were observed.

Delta increases in cortisol differed significantly between groups for men as well as women, as indicated by a main effect of group [$F(1, 88) = 11.05$; $p = 0.001$] in the absence of additional main or interactive effects. Because of the high variance in cortisol responses to the TSST, a Post hoc split between high and low cortisol responders was conducted. Twenty-one of all men and 13 of all women exposed to the TSST responded with a clear-cut cortisol response. Mean delta increases for high ($N = 34$) and low ($N = 34$) responders and controls can be found in Table 4. As anticipated, an ANOVA confirmed that delta responses differed significantly between responder groups [$F(1, 86) = 78.06$; $p < 0.001$], while no other main or interactive effects were found. Post hoc tests on delta increases proved that high responders showed larger increases than low responders and controls ($p < 0.001$). Low responders,

Table 4
Study 2 mean delta cortisol responses (nmol/l) for high responders, low responders, and controls for men and women separately

	Men	Women
High cortisol responders[a,b]	8.82 (3.97; $n = 21$)	8.52 (4.92; $n = 13$)
Low cortisol responders[a]	−0.23 (1.96; $n = 13$)	−0.28 (2.69; $n = 21$)
Controls[b]	0.44 (2.50; $n = 12$)	0.25 (0.96; $n = 12$)

Standard deviations and sample sizes are given in parentheses.
[a] High cortisol responders > low cortisol responders, $p < 0.01$.
[b] High cortisol responders > controls, $p < 0.01$.

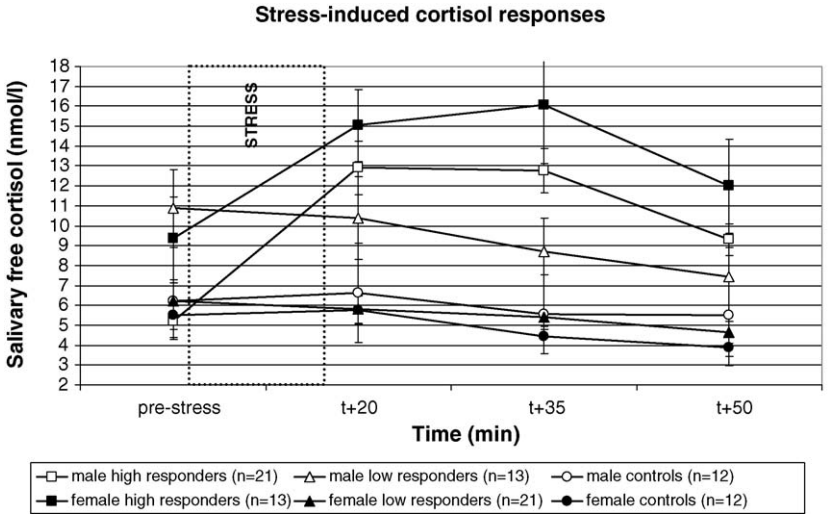

Fig. 2. Study 2 mean free salivary cortisol levels (nmol/l) for male and female high responders, male and female low responders, and male and female controls. Data points indicate cortisol levels before stress/control manipulation (pre-stress), immediately after the stress (TSST) or filler task ($t + 20$), 15 min after stress or filler task ($t + 35$), and after testing was completed ($t + 50$).

Table 5
Study 2 mean number of commission errors, proportion correct recall, and proportion false recall (i.e., critical lure words) on the free recall task, and proportion correct (i.e., ''old'' words recognized as ''old'' and ''new'' words recognized as ''new'') and false (i.e., incorrectly recognizing critical lures as ''old'') recognition

	Men			Women		
	Controls ($n = 12$)	Low responders ($n = 13$)	High responders ($n = 21$)	Controls ($n = 12$)	Low responders ($n = 21$)	High responders $n = 13$)
Number of commissions[a]	2.75 (2.38)	2.77 (1.17)	3.43 (3.01)	1.00 (0.74)	2.62 (1.66)	1.92 (1.19)
Proportion correct recall[b,c]	0.71 (0.11)	0.65 (0.07)	0.62 (0.05)	0.71 (0.07)	0.67 (0.07)	0.68 (0.08)
Proportion false recall	0.39 (0.23)	0.34 (0.16)	0.35 (0.21)	0.27 (0.21)	0.31 (0.21)	0.37 (0.18)
Proportion correct recognition	0.91 (0.08)	0.86 (0.07)	0.87 (0.07)	0.88 (0.07)	0.88 (0.05)	0.89 (0.07)
Proportion false recognition	0.68 (0.32)	0.65 (0.21)	0.70 (0.30)	0.57 (0.26)	0.71 (0.17)	0.67 (0.24)

Descriptive results are provided for male and female high responders, low responders, and controls separately. Standard deviations are given in parentheses.
[a] Men > women, $p < 0.01$.
[b] Controls > high cortisol responders, $p < 0.01$.
[c] Controls > low cortisol responders, $p < 0.01$.

however, did not differ from controls. Fig. 2 shows increases in cortisol levels throughout the experiment for male and female high responders, low responders, and controls.

3.2.4. Memory performance[1]

Table 5 gives descriptive results from the DRM recall and DRM recognition task. For the number of commission errors, ANOVA showed a main effect of sex [$F(1, 86) = 7.11$; $p = 0.009$] devoid of other main or interactive effects. As can be seen in Table 5, this had to do with men committing more errors of commission than women. An ANOVA performed on correct recall showed a significant main effect of responder group [$F(2, 86) = 4.48$; $p = 0.014$], while no other main or interaction effect was found. Post hoc tests showed that controls showed superior correct free recall performance in comparison to low [$t(56) = 2.18$; $p = 0.034$] and high [$t(56) = 3.07$; $p = 0.003$] cortisol responders. There were, however, no differences between low and high responders. For falsely recalled critical lures, proportion correct recognition, and false recognition (i.e., recognizing a critical lure as ''old'') rates, no significant main or interactive effects were found.

Delta increases were not significantly related to false recall ($r = 0.02$) or false recognition ($r = 0.08$) rates among participants exposed to the TSST. Even when data from studies 1 and 2 was collapsed and then correlations were computed, these r's remained non-significant ($N = 97$; $r = -0.02$ and 0.02 for false recall and recognition, respectively).

[1] Note that in this case, a median split would result in the same high and low responder groups. To check whether other ways to split the present data would lead to similar results, we also conducted a tertile split. For each of the dependent variables, 4 (responder group: high responders; $n = 23$ versus medium responders; $n = 22$ versus low responders; $n = 23$ versus controls; $n = 24$) × 2 (sex) ANOVA's yielded highly similar results, with the only significant results being an effect of sex [$F(3, 84) = 6.35$; $p = 0.014$] for number of commission errors and a main effect of responder group for correct recall [$F(3, 84) = 2.99$; $p = 0.036$].

3.3. Discussion

The main results of Study 2 can be summarised as follows. To begin with, there were significant differences between high cortisol responders, low cortisol responders, and controls with respect to correct recall of presented words. More specifically, exposure to the TSST resulted in impairments in correct free recall in comparison to control participants. High cortisol responders, however, did not differ from low cortisol responders in correct free recall rate. Neither did we find evidence for the idea that the effect of stress on memory is modulated by sex, although overall men made more commission errors than women. Taken together, our data fit well with studies demonstrating the undermining effects of acute stress on recall of neutral words (e.g., Jelicic et al., 2004; Kirschbaum et al., 1996; Tops et al., 2003). Second, despite its undermining effect on correct free recall exposure to the TSST did not result in heightened levels of false recall of the critical lures. This was true for low as well as for high cortisol responders. Moreover, false recognition rates also did not differ between low or high cortisol responders and controls.

4. General discussion

To summarise, participants in both studies 1 and 2 falsely recalled and recognized the non-presented critical lures at rates similar to those reported elsewhere (e.g., Roediger and McDermott, 1995; Stadler et al., 1999). Results from Study 1 showed that stress and control group did not differ with respect to false recall and false recognition of critical lures. Study 2 showed that high cortisol responders did not demonstrate elevated levels of false recall or false recognition in comparison to low cortisol responders or controls. There were, however, significant impairments in correct recall of presented words for high and low cortisol responders compared to their control counterparts (Study 2). No significant correlations between false recall and recognition rates and delta increases in cortisol were revealed.

The finding that stressed (Study 1) and high and low cortisol responders (Study 2) do not differ from controls in their rate of false recollection, to some extent contradicts the work of Payne et al. (2002). It should be noted, though, that there are substantial differences in methodology between the Payne et al. study and the current studies. That is, the present studies drew upon a free recall task following each DRM list and an overall recognition task after all DRM lists had been presented, while the Payne et al. study employed a single recognition task subsequent to each DRM list.

A recent meta-analysis by Het et al. (2005; p. 780) looking at administered acute cortisol and memory performance concluded that for recognition memory ‘‘[. . .] the effect sizes were on average descriptively smaller – almost zero – than the effect sizes for free or cued recall performances. This finding may indicate that recognition memory performance is less suitable to uncover effects of cortisol on memory.’’ Thus, assuming that acute cortisol elevations were responsible for the heightened rate of false recognition in the Payne et al. (2002) study, one would have predicted that these effects would have been even larger with recall tasks of the type used in the current studies.

Meanwhile, glucocorticoids (e.g., cortisol) may yield very different results for recall and recognition tasks as these tasks seem to tap different neural structures involved in memory (e.g., Brown and Aggleton, 2001; Buckner and Wheeler, 2001). Recognition performance on DRM-related tasks has been investigated by means of event-related potentials (e.g., Curran et al., 2001; Fabiani et al., 2000), positron emission tomography (Schacter et al., 1996), and functional magnetic resonance imaging (e.g., Cabeza et al., 2001). Collectively, these studies indicate that while the hippocampus, together with several cortical regions, contributes to false recognition in the DRM paradigm, rates at which stimuli are falsely recognized may be under regulation from monitoring processes of the prefrontal cortex (for a thorough review, see Schacter and Slotnick, 2004).

Animal, but also human research shows that glucocorticoids may differentially affect the various memory phases (e.g., Roozendaal, 2002). Specifically, while glucocorticoids like cortisol have been shown to enhance memory formation (e.g., Buchanan and Lovallo, 2001), there have also been reports of an impairing effect of glucocorticoids on retrieval (e.g., Wolf et al., 2004). Roozendaal (2002) suggests that when triggered by stress, the basolateral amygdala turns the brain into a memory-consolidation state, thereby resulting in strong consolidation for ongoing events while at the same time undermining future attempts at retrieval. This might be important given the fact that a DRM task inherently comprises only one session in which DRM lists are studied by research participants who subsequently, but during the very same session are probed for recall and/or recognition. Indeed, from a theoretical viewpoint it is conceivable that facilitating effects of cortisol increases on memory formation might overshadow the detrimental effects of cortisol on retrieval processes.

In addition, several studies involving acute psychological stressors including, but not limited to the TSST, have shown a sex effect in HPA axis stress responses (for a comprehensive review, see Kudielka and Kirschbaum, 2005). In particular, Kudielka and Kirschbaum (2005, p. 117), note that ‘‘[. . .] most psychological stress studies revealed that there are (a) no significant sex differences or (b) higher cortisol responses in young men than in young women [. . .]’’. In both studies 1 and 2, delta cortisol increases were indeed larger for men than for women, although this difference fell short of being significant. This is especially important given that, at least in Study 1, cortisol responses to the TSST were relatively small. Furthermore, recent studies suggest that individual differences in cortisol reactivity may very well reflect genetic predispositions (e.g., Wüst et al., 2004), anticipatory cognitive appraisal (e.g., Gaab et al., 2005; Rohrmann et al., 1999), polymorphism in sensitivity of glucocorticoid receptor tissue (e.g., Rohleder et al., 2003), or even variation in lifetime cortisol exposure (e.g., Lupien et al., 2002a).

In addition to animal studies, research involving humans has occasionally found evidence suggesting that sex differences may modulate the effects of glucocorticoids on memory

functioning. In contrast, no differences between men and women on memory performance after exposure to the TSST were found in the present studies. However, we did observe that relatively independent of stress, men produced more commissions (i.e., confabulated extra list words) on the free recall task. Another factor that might be of relevance when studying the effects of cortisol on memory performance is that cortisol levels in the morning appear to lead to impairing effects, while generally yielding no or slightly enhancing effects when tested in the afternoon (Lupien et al., 2002b). From their meta-analysis, Het et al. (2005) concluded that studies performed in the afternoon on average yielded an effect size that was smaller and in the opposite direction than the effect size found by studies performed in the morning. Therefore, it awaits to be seen whether our failure to detect an effect of cortisol increase on false recall can be generalized to other times of day.

Some notes on the methodological limitations of the present studies are in order. First, the current studies used only a limited number of cortisol measurements to characterize the stress responses. Hence, it is possible that we missed the peak level of cortisol in a substantial number of our participants. Secondly, it can be argued that our studies used only a single session memory performance task, which makes it impossible to differentiate between learning and recall effects following acute psychosocial stress. Thus, it cannot be excluded that beneficial effects of cortisol on memory formation were reversed through the impairing effect of cortisol on memory retrieval resulting in a net effect that did not differ from controls. Also, it must be acknowledged here that, especially in Study 1, cortisol responses to the TSST were not as large as in some other studies (e.g., Kirschbaum et al., 1992; but see Elzinga and Roelofs, 2005, for similar cortisol responses among high and low responders to the TSST). Future research should take these limitations into account.

In sum, then, our results suggest that cortisol elevations alone might be insufficient to increase false recall or recognition rates in a DRM paradigm. The fact that heightened false recognition rates following acute stress do not seem strongly related to cortisol responses or to a modulating effect of sex indicates that factors other than HPA axis responses may be responsible for the detrimental effects of acute stress on false recollections reported elsewhere (e.g., Payne et al., 2002). A likely candidate for this would be the brain's noradrenergic system that controls autonomic output (Sved et al., 2002). Clearly, this issue warrants further study. What can be said with some confidence is that in the present studies, exposure to an acute psychosocial stressor – even when this reduced correct recall – did not necessarily promote false recall and recognition.

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