

Acute Stress Impairs Recognition for Positive Words—Association with Stress-induced Cortisol Secretion

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Some studies suggest that stress-induced effects of cortisol on memory are modulated by the valence of the stimuli to be learned and retrieved. The present study investigated the effect of acute stress-induced cortisol secretion on acquisition and retrieval of pleasant, unpleasant and neutral words.

Sixty healthy men were randomly assigned to one of the three experimental groups. Participants were either exposed to a standardized laboratory stressor (the Trier Social Stress Test) before learning a wordlist, or before retrieval, or were not stressed. Free recall and recognition were tested 24 h later.

Free recall was not affected by stress exposure. For recognition, there was no main effect of the stressor, but a main effect of valence and a valence by group interaction emerged: recognition for positive words was significantly impaired when subjects were stressed before retrieval. In addition, a positive correlation between the cortisol response and errors of commission was found.

The results suggest that acute stress impairs memory for positive stimuli and that stress-induced cortisol secretion interferes with accuracy of memory retrieval, i.e. the ability to discriminate true memories from false ones.

Keywords: Psychosocial stress; Verbal memory; Stimulus valence; Cortisol; Recognition memory

INTRODUCTION

Acute stress is well known to trigger several hormonal alterations in animals and humans, including marked increases of glucocorticoid concentrations in blood and saliva (Mason, 1968; Kirschbaum *et al.*, 1993).

A large body of animal research indicates that acute elevations of circulating glucocorticoids may enhance memory storage (Roosendaal, 2000). The memory enhancing effect of glucocorticoids has been shown repeatedly in aversive conditioning paradigms and water-maze experiments, which are associated with a marked increase in emotional arousal, suggesting an interaction of arousal and glucocorticoids on memory formation (Roosendaal, 2000; Okuda *et al.*, 2004). There is now considerable evidence that this interaction takes place at the level of the basolateral amygdala, with circulating glucocorticoids modulating the effect of central noradrenergic mechanisms (McGaugh *et al.*, 1996; McGaugh, 2000; Roosendaal *et al.*, 2002). The literature on human studies is less consistent: some human studies

have shown beneficial effects of post-learning stress (Cahill *et al.*, 2003) and stress hormones (e.g. epinephrine, Cahill and Alkire, 2003), while others showed the opposite (Rimmele *et al.*, 2003) or were not able to detect interactions of cortisol and arousal on memory (Abercrombie *et al.*, 2003).

In accordance with the animal data, Buchanan and Lovallo (2001) reported a memory enhancing effect of cortisol in humans, which was specific for emotionally arousing material. They found that a single dose of 20 mg cortisol enhanced long-term memory for arousing pictures when given orally 1 h prior to picture presentation. Interestingly, the memory enhancing effect appeared to be independent of the valence of the arousing stimuli, since data for the arousing stimuli was collapsed over pleasant and unpleasant pictures (Buchanan and Lovallo, 2001). These results extend previously conducted experiments in animals suggesting that glucocorticoids interact with arousal in enhancing memory for emotionally relevant stimuli in humans.

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On the other hand, stress and glucocorticoids have been reported to impair memory in both animals and humans under certain conditions (Wolkowitz *et al.*, 1990; Kirschbaum *et al.*, 1996; Lupien and McEwen, 1997; Newcomer, *et al.*, 1999; Lupien *et al.*, 2002). De Quervain *et al.* reported that rats which were trained in a water-maze showed impaired memory of the platform location when stressed or administered corticosterone (the main glucocorticoid in rodents) before the test trials, suggesting a specific effect on memory retrieval (de Quervain *et al.*, 1998). In two studies with human subjects, de Quervain *et al.* were able to show a similar effect with verbal material. Subjects who had received 25 mg of cortisone showed impaired memory performance for a word list, only when the drug was administered before retrieval, but not when the drug was given before or immediately after learning (de Quervain *et al.*, 2000, 2003; see also Wolf *et al.*, 2001a, for consistent results). Thus, paying regard to the phase within the memory process shows that exogenous as well as endogenous glucocorticoids do not seem to impair memory acquisition but rather retrieval of already acquired memories.

Although it has been suggested that endogenous stress-induced increases in cortisol secretion should have memory effects similar to those reported after administration of exogenous glucocorticoids, the empirical evidence in humans is not consistent. Data from studies with elderly subjects show that a brief stressor can reduce declarative memory performance when subjects are exposed to the stressor immediately before learning (Wolf *et al.*, 1998) or retrieval (Lupien *et al.*, 1997). In contrast, three other studies were not able to detect such effects using a similar laboratory stressor (Wolf *et al.*, 2001b, 2002; Domes *et al.*, 2002). The inconsistency may be caused by differences in timing of the stressor and valence of the employed verbal material.

Although it has been suggested that arousal is the critical factor in emotional modulation of memory storage at the level of the amygdala (Cahill and McGaugh, 1998; McGaugh, 2000; Cahill *et al.*, 2003), a few studies suggest that valence specific enhancement of memory storage is independent of arousal and does not depend on amygdaloid structures (Phelps *et al.*, 1997, 1998). Hence, it might be speculated that stimulus valence is also a modulating factor for the effect of stress and stress-induced cortisol secretion on memory function.

Support for this hypothesis comes from observations in mood disorders. Clinical depression is often accompanied by high endogenous cortisol secretion (Plotsky *et al.*, 1998) and has been associated with chronic stress (de Kloet, 2003). Moreover, dysregulation of cortisol secretion has been proposed to play a crucial role in depressive pathology and associated cognitive disturbances, including memory impairment (Dinan, 1994; Ehler *et al.*, 2001; Gold and Chrousos, 2002). Interestingly, both clinical depression and depressive mood in normal populations have been associated with impaired recall for positive information, i.e. both verbal

and pictorial (Matt *et al.*, 1992; Burt *et al.*, 1995). Taken together, the aforementioned effects of memory phase and stimulus valence might suggest that stress-induced cortisol secretion mainly interferes with memory retrieval of positively valenced material, leaving retrieval of neutral or negative material unaltered.

Thus, the present experiment was designed to investigate whether psychosocial stress and the accompanying increase in endogenous cortisol secretion differentially affects acquisition and retrieval of words differing in valence.

First, according to the results reported by de Quervain *et al.* (2000), we expected a negative effect of acute psychosocial stress on retrieval rather than on acquisition.

Second, we more specifically hypothesized that acute stress-induced elevations of cortisol secretion would lead to impaired retrieval of long-term memory for pleasant words, but not for neutral or unpleasant words.

Third, the valence-specific impairment in memory retrieval should be associated with the stress-induced cortisol response, i.e. subjects showing marked increases of cortisol secretion in response to a stressor should exhibit more pronounced impairment of retrieval of positively valenced memories.

METHODS

Subjects

Male subjects were recruited using campus advertisement and were pre-screened by a telephone interview. Based on the telephone interview subjects were not included if they had experienced serious physical or mental illness in the previous 12 months. In addition, all subjects with habitual nicotine consumption were not allowed to participate in the study. A male population was chosen to rule out potential effects of gender and endogenous oestradiol on cortisol secretion reactivity to stress (Kirschbaum *et al.*, 1999). Sixty healthy non-smoking male volunteers aged 18–42 years (mean \pm SD, 25.3 \pm 6.6 years), with a body-mass-index ranging 17.1–28.3 (mean \pm SD, 23.0 \pm 2.4 years) were recruited and randomly assigned to one of the three experimental conditions (see below). Three subjects for the control condition had to be excluded from cortisol analysis because saliva volume was too low. All other analyses were conducted on the whole sample of 60 subjects.

The study protocol was approved by the local ethics committee. All subjects gave informed written consent and were paid for participation.

Experimental Protocol

The experimental sessions were conducted on two consecutive days. All sessions began between 14:00 and 16:00 h and lasted approximately for 2 h.

On the first day, subjects learned a list of German nouns, which were presented on a computer screen

(for details see below). On the second day, 24 h after learning, memory for these words was tested with a free recall and a recognition task.

Subjects were randomly assigned to one of the three conditions: one group was exposed to a brief laboratory stressor before word presentation on the first day (pre-learning stress; $n = 20$); the second was exposed to the stressor before retrieval on the second day (pre-retrieval stress; $n = 20$); and the third served as a control group without exposure to stress (no stress; $n = 20$).

Memory Testing

Two equivalent word lists (*A,B*) were assembled consisting of 20 pleasant (e.g. joke, friend, love), 20 neutral (e.g. paper, hall, reason) and 20 unpleasant (e.g. fear, poison, damage) nouns each. All words were taken from a standardized list of German words (Schwibbe *et al.*, 1994). The word lists were tested for emotional equivalence using two-way ANOVA (valence \times list) with the valence ratings reported by Schwibbe *et al.* (1994) as the dependent variable. There was no significant effect of list, $F(1, 114) = 0.71$, $p = 0.41$, or list by valence interaction $F(2, 114) = 2.79$, $p = 0.066$, indicating emotional equivalence of the two lists. Presentation of the lists was balanced across groups, i.e. List *A* served as the learning list on the first day for one half of each experimental group, whereas it served as the distractor list on the second day for the other half. The same was true for List *B*.

On the first day, one of the word lists was presented to the subjects, the second list served as distractor for later recognition testing. The words of the learning list were separately presented on a computer screen for 4 s each, with the instruction to learn them for delayed recall.

On the second day, free recall of words was tested. Participants were asked to write down all words they spontaneously remembered. Thereafter, recognition memory was tested. Therefore, the previously learned words were mixed with the 60 nouns of the non-learned list and presented in random order on a computer screen. Subjects were asked to decide as fast as possible if the word was old (previously learned) or new (not previously learned). According to a two-high-threshold model, the sensitivity index (P_r ; hit rate minus false alarms rate) was calculated as a measure of recognition performance (Snodgrass and Corwin, 1988).

Psychosocial Stress

To induce psychosocial stress, we employed the Trier Social Stress Test (TSST—Kirschbaum *et al.*, 1993). The TSST consists of two parts, an unprepared speech and a mental arithmetic task performed standing in front of an audience. Both tasks lasted for 5 min. The TSST has been shown to reliably induce significant increases in cortisol concentration in blood and saliva (Kirschbaum *et al.*, 1999; Heinrichs *et al.*, 2001; Domes *et al.*, 2002). The control condition was introduced to the

subjects as an investigation of the effects of posture on stress-hormone secretion. The subjects were asked to stand still for the same length of time as the TSST.

Salivary samples were collected before the onset of the TSST (-20 min) and four times ($+1$, $+10$, $+25$, $+65$ min) after cessation of the stressor. Saliva was collected using the Salivette sampling device (Sarstedt, Rommelsdorf, Germany). Salivary samples were immediately frozen and stored at -20°C until biochemical analysis with a time-resolved fluorescence immunoassay (Dressendorfer *et al.*, 1992). Inter- and intra-assay coefficients of variance were below 10%.

Assessment of Mood, Effort and Distress

Changes in mood due to the TSST were assessed with a standardized 6-item 5-point Likert-scale. The scale is a part of a three-dimensional subjective state questionnaire that has been approved for good reliability and sensitivity in the assessment of momentary mood changes (Steyer *et al.*, 1997). The mood scale was given before and after the stressor. Mood change indices were calculated by subtracting post-stress values from pre-stress values, to compare experimentally induced changes in mood on both experimental days.

At the end of the TSST, subjects were asked to rate adjectives describing their effort and distress during the stressor on two 5-item 5-point-Likert scales. The two scales showed good consistency (Cronbach's Alpha; effort: $r = 0.89$; distress: $r = 0.91$) and did not correlate substantially ($r = 0.16$).

Data Analysis

Linear correlations between demographic factors (age, education, body mass index [BMI]) and cortisol concentrations were tested using multiple regression analysis.

For validation of the stressor, we compared the cortisol response to the experimental manipulation on the two days. Two separate ANOVA were calculated, with the between factor "group" and the within factor "time" for the different saliva samples. To test group-differences for the different sampling times, multiple single comparisons were conducted using *t*-tests with adjusted levels of significance (Bonferroni correction).

Group differences concerning memory performance were tested for statistical significance using separate two-way ANOVA with the factors "group" and "valence" for free recall and recognition performance and different types of recognition errors.

For correlation analysis, the stress response for salivary cortisol (peak cortisol concentration) was calculated subtracting the pre-stress (baseline cortisol concentration) value from the $+10$ min post-stress value. Pearson's correlations between peak-cortisol levels and memory performance were calculated and tested for statistical significance using *t*-tests for correlations.

Significance level for all analyses was set to $p < 0.05$.

RESULTS

Sample Characteristics and Subjective Responses to the Stressor

The three experimental groups were compared for *a priori* differences in several demographic variables. There were no significant differences for any of the demographic variables tested (Table I, upper panel).

In addition, we tested differences in mood changes due to the psychosocial stressor. Mood change indices (see above) were compared for the two days. As a result, the groups did not differ in mood changes, as indicated by non-significant group effects in the ANOVA (Table I, lower panel). Furthermore, the two groups that were exposed to the stressor did not differ in self-reported stress or effort.

Finally, prediction of individual cortisol secretion (baseline and peak-cortisol levels) by demographic factors (age, education, BMI) was tested using multiple regression analysis. No significant beta was observed (all $p < 0.05$), the overall explained variance was $R^2 = 0.134$ for baseline-cortisol and $R^2 = 0.120$, for peak-cortisol levels. Furthermore, individual cortisol secretion could not be predicted by stress-induced changes in mood, as indicated by non-significant linear correlations (all $r < 0.20$). Also, self-reported stress and effort had no predictive power for cortisol secretion (all $r < 0.25$).

Stress-induced Cortisol Secretion

On the first day, the pre-acquisition stress group showed a marked elevation of salivary free cortisol concentration in response to the stressor, while the other two groups did not show such an increase. On the second day, a significant reaction of the pre-retrieval stress group was observed. In accordance, the two ANOVA with the cortisol data revealed significant interaction effects of group and time on both days, $F(8, 208) = 5.86$; $p < 0.001$ and $F(8, 208) = 13.72$; $p < 0.001$, respectively (Fig. 1). For both days, multiple single comparisons indicated significant differences between the stress group of

the respective day and the other two groups at all sampling times except for the pre-stress value.

In addition, we explored differences in the magnitude of the stress response in the two stress groups. The two groups did not differ with regard to the post-stress cortisol maximum concentration (mean \pm SD: pre-learning: 16.7 ± 9.7 nmol/l, pre-retrieval: 18.5 ± 13.1 nmol/l; $t(38) = -0.49$; $p = 0.63$), but the pre-retrieval stressed group showed a significantly greater *relative increase* in salivary cortisol concentration in response to the stressor (percent from pre-stress value; mean \pm SD: pre-learning: $193 \pm 118\%$, pre-retrieval: $394 \pm 360\%$; $t(23.05) = -2.37$, $p < 0.05$).

Effects of Stress on Memory Acquisition and Retrieval

For the free recall task, despite a significant main effect of valence, $F(2, 114) = 12.94$, $p < 0.001$, neither a main effect of group, nor a group by valence interaction was observed, $F(2, 57) = 0.31$, $p = 0.73$, and $F(4, 114) = 0.25$, $p = 0.91$ (Fig. 2, left panel).

Consistently, word recognition was significantly affected by stimulus valence, $F(2, 114) = 8.79$, $p < 0.001$, but stress had no significant effect in terms of a main effect of group, $F(2, 57) = 0.37$, $p = 0.69$. Conversely, a significant valence by group interaction emerged, $F(4, 114) = 2.49$, $p < 0.05$, with the two stress groups showing impaired recognition of stimuli with positive valence (Fig. 2, right panel). A *post-hoc* single comparison analysis for the positive stimuli revealed impaired recognition for the pre-retrieval stressed group, $t(38) = 2.36$, $p < 0.025$, and a trend towards impaired recognition in the pre-learning stressed group, $t(38) = 1.93$, $p = 0.062$, both compared to the non-stressed control group.

Additionally, we tested specific effects of the stressor on different types of recognition errors (errors of omission vs. errors of commission). Separate two-way ANOVA (group \times valence) for each error type were calculated. For the *misses* (errors of omission) a strong valence effect emerged, $F(2, 114) = 21.63$, $p < 0.001$. No group effect, $F(2, 57) = 0.07$, $p = 0.92$, and no group by valence

TABLE I Demographic variables and measures of mood, distress and effort in response to the experimental stressor of the three experimental subgroups

	Group (Time of stressor)						ANOVA
	Pre-learning		Pre-retrieval		Control		
	<i>m</i>	SD	<i>m</i>	SD	<i>m</i>	SD	
Age	27.8	9.0	26.4	5.4	27.8	8.6	$F(2, 59) = 0.21$; n.s.
Education (years school)	12.3	1.3	12.5	1.2	12.1	1.4	$F(2, 59) = 0.08$; n.s.
BMI	22.6	2.1	22.9	2.6	23.7	2.3	$F(2, 59) = 0.64$; n.s.
Mood difference post-pre stress, day 1	-2.4	3.6	-1.3	3.7	0.1	2.0	$F(2, 59) = 2.85$; n.s.
Mood difference post-pre stress, day 2	0.4	2.2	-0.1	2.4	0.3	1.4	$F(2, 59) = 0.23$; n.s.
Effort	18.9	2.6	18.5	3.7	-	-	$t(38) = 0.45$; n.s.
Distress	12.7	4.9	13.0	4.1	-	-	$t(38) = -0.18$; n.s.

Means were compared using one-way ANOVA and student's *t*-test.

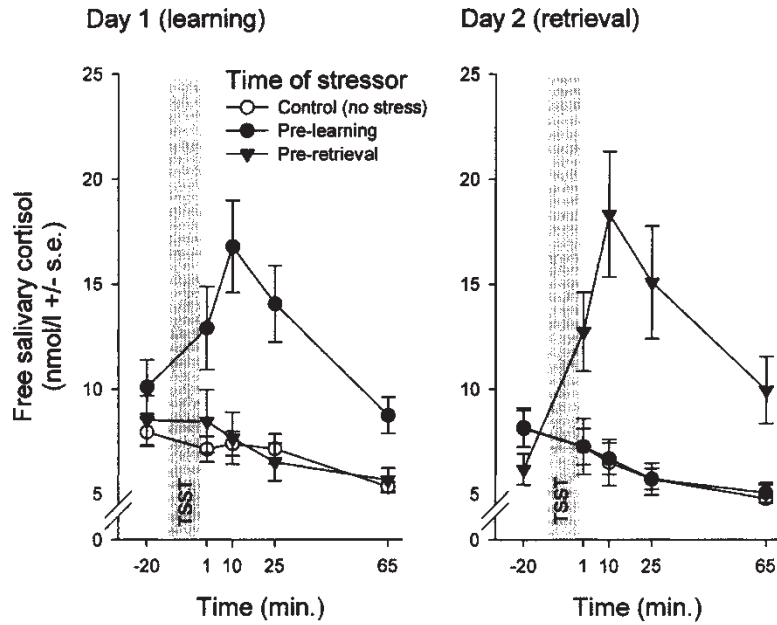


FIGURE 1 Cortisol secretion on the first and the second day for the different experimental groups. The graph shows mean salivary free cortisol concentration before the onset of the Trier Social Stress Test (TSST)/control condition (-20 min) and at four sampling times after cessation of the stressor/control condition ($+1$, $+10$, $+25$, $+65$ min); error bars represent SEM. The pre-learning stressed subjects ($n = 20$) showed elevated cortisol concentrations after the stressor on day 1, while the pre-retrieval stressed subjects ($n = 20$) had elevated cortisol concentrations on day 2 compared to the control group ($n = 20$), as indicated by significant group differences for the sampling points after cessation of the stressor (for details see text).

interaction was observed, $F(4, 114) = 0.92$, $p = 0.46$. For the false alarms (errors of commission) also a significant valence effect emerged, $F(2, 114) = 3.40$, $p < 0.05$. There was no group effect, $F(2, 57) = 0.58$, $p = 0.56$, but there was a trend towards a group by valence interaction, $F(4, 114) = 2.09$, $p = 0.09$. (Fig. 3). More specifically, *post-hoc* single comparison analysis revealed a significantly greater false alarms rate in the pre-learning stressed group, $t(38) = -2.05$; $p < 0.05$, and a trend in the same direction for the pre-retrieval stressed group, $t(38) = -1.77$; $p < 0.10$ both compared to the control subjects.

Finally, we inspected the data for an association between the individual cortisol stress-response and memory performance, calculating Pearson's correlations for peak cortisol concentrations and different indices of memory performance (Table II). Regardless of stimulus valence and time of the stressor, subjects showing high cortisol concentrations in response to the stressor showed significantly more errors of commission, as indicated by positive Pearson's correlations between peak cortisol concentrations and false alarm rates. No other correlation reached significance (Table II). We furthermore inspected the distribution with

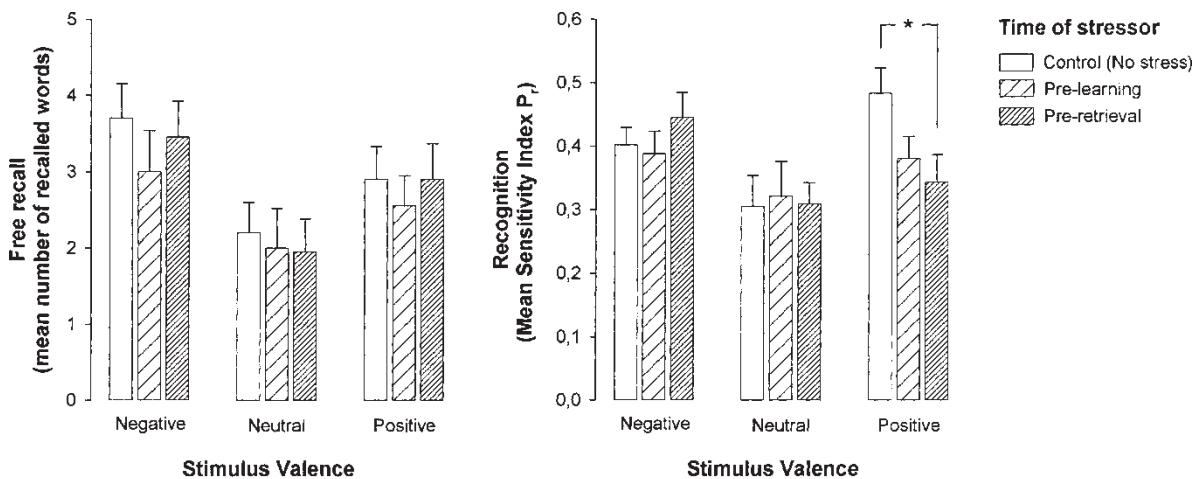


FIGURE 2 Free recall and recognition performance as a function of stimulus valence and timing of the stressor. The bars show mean number of spontaneously recalled words (free recall), and the mean sensitivity index P_r (recognition); error bars represent SEM. Using separate two-way ANOVA, (valence \times group), a significant interaction was confirmed for recognition memory, $F(4, 114) = 2.49$, $p < 0.05$: the pre-retrieval stressed group showed impaired recognition compared to the control group, $t(38) = 2.36$, $p < 0.025$, the pre-learning stressed group showed a trend in the same direction, $t(38) = 1.93$, $p = 0.062$. * $p < 0.05$.

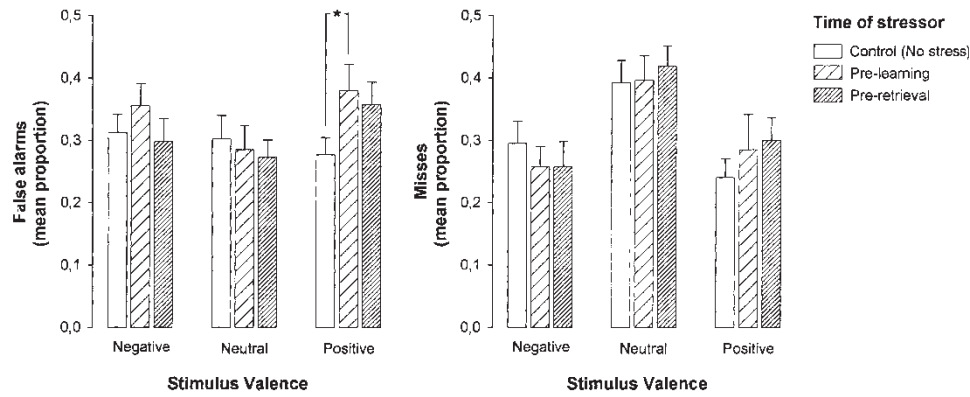


FIGURE 3 Recognition errors as a function of stimulus valence and timing of the stressor. The bars show mean proportions of false alarms (errors of commission, type II) and misses (errors of omission, type I); error bars represent SEM. Using separate two-way ANOVA (valence \times group), a trend towards an interaction between group and valence was confirmed for the false alarm rate, $F(4, 114) = 2.09, p = 0.09$: the pre-learning stressed group showed more false alarms compared to the control group, $t(38) = -2.05, p < 0.05$, the pre-retrieval stressed group showed a trend in the same direction, $t(38) = -1.77, p = 0.083$. * $p < 0.05$.

a scatterplot (mean false alarm rate vs. cortisol response) and could identify one subject scoring high on both variables (Fig. 4). Accordingly, using Spearman's rank correlations the association between the cortisol response and false alarm rate was weaker, but still significant for the negative stimuli ($r = 0.44; p < 0.05$). For the positive stimuli, there was still a trend in the same direction ($r = 0.41; p = 0.07$).

DISCUSSION

In sum, there was no global effect of the psychosocial stressor on both free recall and recognition memory, when stimulus valence was not taken into account. This contrasts with previously reported negative effects of exogenously administered glucocorticoids on long-term memory (de Quervain *et al.*, 2000, 2003; Wolf *et al.*, 2001a). In turn, the results are consistent with recently published data, which did not show global memory impairment due to stress-induced cortisol secretion (Wolf *et al.*, 2001b, 2002; Domes *et al.*, 2002). It should be mentioned that cortisol concentrations

in pharmacological studies were significantly greater than those obtained in the present study. Since it is well known from animal studies that the association between cognition and glucocorticoids follows an inverted-U function (Lupien and McEwen, 1997), concentration differences might explain the inconsistency between behavioural and pharmacological studies. In line with this are the results of Abercrombie *et al.* (2003), who showed a non-linear relation between cortisol concentration and memory in humans.

However, the present findings support our hypothesis that the effect of acute psychosocial stress on long-term verbal memory depends on stimulus valence and timing of the stressor. Consistent with our hypothesis, the psychosocial laboratory stressor specifically impaired recognition performance for positive stimuli. Furthermore, the impairment was more pronounced in subjects stressed before retrieval compared to those stressed before acquisition. A large body of experiments suggests that memories are more easily retrieved when their valence is congruent with the momentary mood of the subject (Blaney, 1986; Matt *et al.*, 1992), i.e. subjects with low mood are expected to show enhanced memory for

TABLE II Pearson's correlations (Spearman's rank correlations) between the individual stress-induced cortisol response and different indices of memory performance as a function of time of stressor and stimulus valence

	Time of stressor (group)					
	Pre-learning			Pre-retrieval		
	Negative words	Neutral words	Positive words	Negative words	Neutral words	Positive words
Free Recall	-.10 (-.04)	.04 (.02)	.13 (.10)	.18 (.33)	.00 (.19)	.29 (.27)
Recognition (Pr)	-.14 (-.08)	-.17 (-.09)	.18 (-.02)	-.18 (-.06)	-.16 (.00)	-.13 (-.18)
False Alarms (Errors of commission)	-.14 (-.11)	.03 (.06)	-.21 (-.19)	.61 (.44)	.50 (.28)	.46 (.41)
Misses (Errors of omission)	.30 (.32)	.23 (.22)	.14 (.13)	-.39 (-.35)	-.28 (-.35)	-.31 (-.16)

Note. Correlations printed in bold are significant at $p < 0.05$.

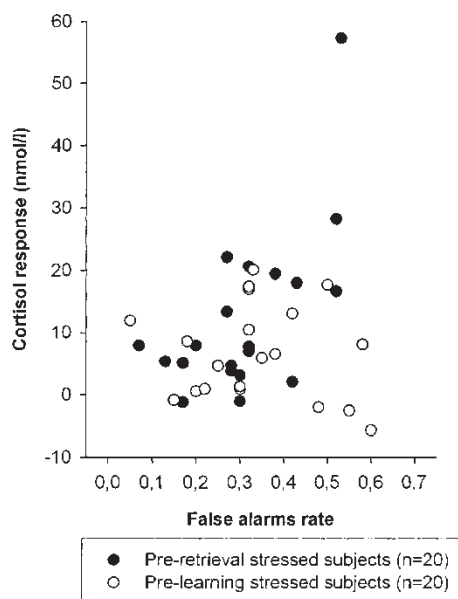


FIGURE 4 Scatterplot showing the bivariate distribution of cortisol responses (relative peak values) vs. the false alarm (errors of commission) rate.

negative stimuli and impaired memory for positive stimuli. Because acute psychosocial stress normally induces negative affective states, the observed impairment in recognition of positive stimuli could be explained in terms of a mood-congruency effect. But in contrast, there was no enhancement of recognition for the negative material. In addition, there was no significant effect of the stressor on self-reported mood in both the stressed groups. Hence, mood-congruency does not seem to be a sufficient explanation for the observed results.

An alternative explanation is that stress-induced cortisol concentrations interfered with memory for the positive stimuli. Empirical support for this hypothesis comes from a study which most recently showed that exogenous cortisol specifically impaired memory for positive and neutral words, leaving memory for negative material unaffected (Tops *et al.*, 2003). It should be mentioned that processes of altered attention and/or motivation after stress exposure may also have affected memory.

With regard to the underlying neuronal mechanisms, it has been suggested that dopaminergic mechanisms in the prefrontal cortex (PFC) might play a role in both acute stress and memory function (Lupien *et al.*, 1999). Accordingly, acute stress has been associated with altered dopamine activity in the PFC, an area that specifically expresses a high density of glucocorticoid receptors (Joels and de Kloet, 1994; Sanchez *et al.*, 2000). Furthermore, dopamine function has been associated with affective processes, especially positive affect (Ashby *et al.*, 1999). Following these assumptions, glucocorticoid released under acute stress may have affected memory through modulation of prefrontal dopaminergic function. Using functional magnetic-resonance imaging, Kensinger and Corkin (2004) has shown that effects of stimulus valence on memory encoding seem to be mediated by

a PFC-hippocampal network, whereas effects of arousal appear to be dependent on an amygdalar-hippocampal network. Thus, it might be speculated, albeit not concluded, that the observed selective effects of acute stress on memory for positive stimuli are at least partially mediated by prefrontal dopaminergic mechanisms.

The hypothesis of an endocrine mediation of the interaction between acute psychosocial stress and memory performance would predict that significant correlations between the cortisol response and memory performance should occur. In agreement, we found that the individual cortisol response to the stressor was significantly associated with recognition performance, more specifically, subjects with a greater cortisol response (high-responders) showed more errors of commission (false alarms) in recognition, compared to the low-responders. Contrary to our hypothesis, the association was not specific for positive stimuli. However, the results are consistent with the study by Payne *et al.* (2002), which showed enhanced false recognition in response to a laboratory stressor. Our findings extend these results, as we show that at least some aspects of stress-induced recognition impairment seem to be linked to cortisol secretion. Taken together, the pattern of correlations support the hypothesis that specific aspects of memory performance are impaired under high cortisol concentrations, while other aspects are unaffected. They are also consistent with the results from de Quervain *et al.* (2000), which suggest that retrieval is more sensitive to the effects of cortisol. It should be noted that the stress-induced increase in cortisol concentration was remarkably greater in the pre-retrieval stress group compared to the pre-learning stress group. Hence, it is conceivable that a stronger cortisol response in the pre-learning stress group would have led to a more pronounced memory impairment. Further experiments should be aimed to differentiate more precisely the memory effects of acute stress in cortisol high- and low-responders, while carefully controlling for the subjective reaction (e.g. negative affect, stress and effort).

To summarize, the results of the present study do not support the idea of a general impairment of memory due to stress or stress-induced HPA-axis activation, but merely suggest that acute stress impairs memory for positive stimuli and that stress-induced cortisol secretion specifically interferes with the accuracy of memory retrieval, i.e. the ability to discriminate memories from false memories.

With respect to replication, the results of the present experiment have important implications. First, a large body of research focuses on the problem of false memories in testimony, including errors of commission in the retrieval of stressful past events. Elaborate models have been put forward to explain these memory problems in terms of disturbances in memory acquisition (for review, see Christianson (1992)). However, the present results indicate that stress-induced cortisol concentrations during retrieval probably also contributes to the observed memory inaccuracies. Clearly, more research is needed on this topic to further explore the association between acute stress endocrinology and the memory process.

Second, the present results might serve as a possible explanation for the inconsistent literature regarding stress, cortisol and memory. Differences in memory testing, i.e. differences in the emphasis that is put on errors in recall and recognition might contribute to the inconsistency in the current literature. Also, inconsistencies in valence of the employed stimuli might account for the non-uniform results of previous studies.

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