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# A non-arousing test situation abolishes the impairing effects of cortisol on delayed memory retrieval in healthy women

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**Abstract**

Animal and human studies have repeatedly shown that stress hormones influence memory. Glucocorticoids (GCs) enhance memory consolidation but impair memory retrieval. Studies in rodents indicate that adrenergic activation is necessary for GC induced effects on memory. We have shown, in two previous placebo-controlled double-blind experiments, that memory retrieval is significantly impaired after oral cortisol (30 mg) treatment in healthy young women. Here, we changed the experimental setting before and during the retrieval testing, so that the participants ($n = 31$) experienced a more relaxed test situation. The learning material, the timing and the tester used were identical to the two previous studies. In the relaxed condition no effect of cortisol on memory retrieval occurred ($p = 0.84$). The results indicate that the experimental setting can influence the effect of cortisol on memory. Our findings suggest that glucocorticoid effects on memory retrieval require testing-associated arousal in humans.


*Keywords:* Glucocorticoids; Stress; Adrenergic activation; Memory retrieval; Humans

Stress leads to a cascade of physiological reactions, one of which is the release of glucocorticoids (GCs) and catecholamines (epinephrine and norepinephrine). These ensure appropriate adaptation to the changed internal or external environment [11]. A large body of studies with GCs have shown that they can have an impairing as well as an enhancing effect on memory [10,16,21]. Most researchers report impaired memory retrieval after stress or GC treatment in animals and humans (e.g. [12,13,15,19,20]). In contrast, cortisol seems to enhance memory consolidation, especially for emotional material (e.g. [2,3,14]).

Catecholamines also modulate memory performance for arousing or emotional contents. Unlike GCs, epinephrine cannot cross the blood brain barrier. However, adrenergic activation of the vagal afferents terminate in the nucleus of the solitary tract and the locus coereleus. These regions then release norepinephrine in the brain [21]. In addition, catecholamines also act locally as neurotransmitters in the brain [17]. Extensive evidence indicates that memory is modulated via noradrenergic activation in the basolateral amygdala (BLA) [21]. Rodent studies reported that post training intra BLA infusions of epinephrine or a beta-adrenoceptor agonist enhances memory [21]. Besides, human participants receiving a beta blocker before learning did show impaired emotional memory when tested 1 week later [4]. Pharmacological human neuroimaging studies have recently shown in vivo that adrenergic activation in the amygdala is a prerequisite for emotional memory enhancement [24,25].

Experiments in rodents indicate that GCs modulate the action of norepinephrine in the brain. On the one hand GCs affect adrenoceptors. On the other hand the noradrenergic brain stem cell groups contain glucocorticoidreceptors and can thus be modulated by GCs [21]. Additionally, there seems to be another connection between epinephrine and GCs. Several experiments in rodents indicate that the presence of adrenergic stimulation is necessary for the memory modulating effects of GCs. For example, infusion of beta-adrenoceptor antagonists into the BLA before the training blocks the enhancing effects of glucocorticoid receptor (GR) agonists [21]. Similar effects in rodents have been achieved by the modulation of the learning situation. In this study, one half of the rats were habituated to the learning environment and the other half were not. After acquisition, rats from both groups received GC or vehicle injections. Only the non-habituated group displayed enhanced object recognition whereas no effect occurred in the habituated group. Okuda and colleagues concluded that training-induced emotional arousal is essential for positive as well as nega-

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tive GC effects on memory [18]. The interdependent effects of GCs and epinephrine on memory retrieval were examined in another recent experiment of Roozendaal et al. [22]. In the study, rats which had learned to locate the platform position in a watermaze paradigma, received intrahippocampal infusions of a GR agonist before retrieval testing. Here too, as in previous results, their retrieval was impaired. Parallel injections of a beta-adrenoceptor antagonist into the hippocampus or the BLA blocked the negative effects of GR agonist injections on retrieval. These results indicate that beta-adrenergic activation in limbic regions is necessary for GC effects on memory retrieval [22].

A recent human stress study investigated the interaction between cortisol and adrenergic activation induced by the testing condition [6]. In this experiment effects on working memory were explored. Participants in the Trier social stress test (TSST) were post hoc separated in responders (cortisol increase in response to stress) and non-responders. Working memory was tested within the stress context and during the recovery period. Cortisol responders showed poorer performance than the non-responders in the arousing context only. The authors suggest that the absence of adrenergic activation in the recovery period leads to absent effects of the stress induced cortisol elevations. Another investigation observed that the cortisol reactivity to a social speech stressor immediately after learning is associated with enhanced memory consolidation for negative pictures only in subjects who reported emotional arousal (increase in negative affect) because of the speech stressor [1]. This study therefore also supports the notion that arousal and cortisol interact in modulating memory in humans.

The absence of studies investigating the interdependent effects of a pharmacological GC administration and arousal of the learning situation on declarative memory retrieval in humans encouraged us to conduct the following study. We tried to modulate arousal by modifying the testing conditions with a procedure that parallels the strategy used by Okuda et al. [18].

The newly reported data of the current study will be contrasted with two previous studies from our laboratory, in which we used identical study design and material [12,15]. In addition the same experimenter (S.K.) conducted the cognitive testing in all three studies. Only naturally cycling women (not using hormonal contraception) were studied. All reported a regular menstrual cycle between 26 and 32 days. None of the women had acute or chronic diseases or were taking medications. Subjects were not obese (BMI < 25, BMI: weight in kg/height in $m^2$) and were between 20 and 35 years old.

The study was conducted in accordance with the declaration of Helsinki (http://www.wma.net). It was approved by the national ethic committee of the German Psychological Association (Deutsche Gesellschaft fuer Psychology; DGPs) and all subjects provided written informed consent.

The relaxed group comprised 31 women. Sixteen of them were tested during the menses phase (2nd to 4th day of bleeding) and 15 in the follicular phase (17th to 21st days before the onset of the new menstrual cycle).

The first reference group comprised 16 free cycling women in the first half of their menstrual cycle [12].

The second group comprised 27 women. Thirteen of them were tested in the menses phase (2nd to 4th day of bleeding) and 14 in the luteal phase (4th to 8th days before the onset of the new menstrual cycle) [15].

The one way ANOVA showed no significant differences between the groups in age ($F(2,73) = 1.98$; $p = 0.15$) or BMI ($F(2,73) = 1.90$; $p = 0.16$).

In a double-blind crossover placebo controlled fashion participants were orally administered with either 30 mg hydrocortison (Hoechst, Germany) or placebo. Treatment order was randomized. Upon arrival (between 10:00 and 11:00 am) the subjects learned a list containing 15 neutral and 15 negative words (details below). Four hours later they received either cortisol or placebo. After a delay of 1 h memory retrieval was tested (details below).

This relaxed group differed in the environmental conditions during the afternoon from the other two previously published groups. In the relaxed testing condition subjects spent the 1 h waiting between treatment intake and memory retrieval testing together with the experimenter in her quiet office. Subjects were allowed to read while the experimenter worked on her desk. Sometimes the experimenter and the subjects engaged in short small-talk like conversations. The memory retrieval testing also took part in the office room, without an explicit prior announcement that cognitive testing would soon commence.

The two reference groups [12,15] spent the 1 h waiting after the treatment in an open room with other participants and/or in a waiting area in the hallway. They were also allowed to read. There was often some disturbance like the ringing of the phone, conversation between the experimenter and other persons, and or movements from subjects entering or leaving the rooms. For the memory retrieval testing the subjects were lead to another testing room located in another floor of the building (approximately 400 m distance). In addition, during the walk to the test room, they were explicitly informed that their memory would soon be tested.

A word list (with two parallel versions available) containing 15 negative and 15 neutral words were presented to the subjects on a piece of paper [12]. There were no differences between neutral and negative words or between the two lists with respect to word length or word frequency. Subjects were given 2 min to learn the list with immediate free recall being tested. The participants were informed that their memory for the words would be tested once again in the afternoon. This procedure was directly repeated leading to a total of two learning trials. In the afternoon (5 h after initial learning and 1 h after oral cortisol or placebo treatment) delayed free recall of the wordlist presented in the morning was tested. In order to account for within and between subject variance in initial learning, free recall performance in the afternoon was expressed as the percentage of words remembered in relation to the second (and last) learning trial in the morning [12]. After free recall, cued recall was assessed by presenting the first two letters of each learned word in a random order on a piece of paper. Again memory results were expressed as the percentage of words remembered in relation to the last learning trial.

An adjective checklist for the assessment of good versus bad mood, awake versus tired and calm versus restless was filled

Table 1
Learning and cued recall performance of the participants

| Testing condition | Relaxed ($n = 31$) | Formal: reference I [12] ($n = 16$) | Formal: reference II [15] ($n = 27$) | Formal groups (combined) |
|---|---|---|---|---|
| Learning performance placebo | 21.58 ± 0.61 | 20.75 ± 1.26 | 20.93 ± 0.74 | 20.86 ± 0.65 |
| Learning performance cortisol | 21.39 ± 0.66 | 21.06 ± 0.93 | 21.11 ± 0.67 | 21.09 ± 0.54 |
| Cued recall performance in % placebo | 79.29 ± 2.53 | 87.41 ± 5.34 | 79.23 ± 2.25 | 82.27 ± 2.48 |
| Cued recall performance in % cortisol | 78.13 ± 2.88 | 84.45 ± 4.41 | 77.80 ± 1.99 | 80.27 ± 2.09 |

Memory performance (mean ± S.E.) in initial learning and cued recall for the relaxed group, the two formal groups separately [12,15] and both formal groups combined. Cued recall performance was expressed as the percentage of words remembered in relation to the second (and last) learning trial in the morning.

out by the subjects shortly before the retrieval testing [12]. Each scale comprised eight adjectives, which had to be rated on a five-point scale (possible scores range being 0–40).

Saliva was collected using Salivette collection devices (Sarstedt, Nümbrecht, Germany). Samples were taken before treatment, 60 min (immediately before cognitive testing) and 90 min after treatment. Free cortisol was measured using an immunoassay (IBL, Hamburg, Germany). Inter-assay and intra-assay variations were <15%.

Cortisol levels increased in response to the treatment in the relaxed testing group. In baseline measures both conditions did not differ (7.31 ± 0.72 nmol/l in placebo condition versus 8.88 ± 1.25 nmol/l in cortisol condition). In the placebo condition the levels stayed low before (5.84 ± 1.09 nmol/l) and after the testing (4.76 ± 0.45 nmol/l). In contrast, in the cortisol condition, the level before testing was 145.64 ± 16.61 nmol/l and after testing was 147.11 ± 22.00 nmol/l. ANOVA with the factors treatment and time revealed a main effect of treatment ($F(1,30) = 83.58$; $p < 0.01$), time ($F(2,60) = 28.96$; $p < 0.01$) and a time by treatment interaction ($F(2,60) = 28.87$; $p < 0.01$). These effects occurred also in both formal conditions and similar cortisol levels were observed (for further results see [12,15]).

The three groups did not differ in initial learning in the morning (see Table 1). Univariate ANOVA with the factor group did not reveal a significant difference between the three groups on learning/acquisition on the placebo ($F(2,71) = 0.31$; $p = 0.74$) or cortisol ($F(2,71) = 0.06$; $p = 0.94$) day.

In contrast to the two previous studies participants in the relaxed testing condition displayed no cortisol induced retrieval impairment (For means and S.E.s see Fig. 1). An ANOVA with the factors treatment and test group was calculated. There was a main effect treatment ($F(1,71) = 9.15$; $p < 0.01$) but also a significant treatment by test group interaction ($F(2,71) = 3.76$; $p < 0.03$). Follow up analysis was done with bonferroni-adjusted paired $t$-tests. The test revealed that no effect of cortisol treatment occurred in the relaxed group ($t(30) = -0.21$, $p = 0.84$). In contrast cortisol had significantly ($t(15) = 3.67$; $p < 0.01$) impaired memory retrieval in the first reference group [12]. Similarly, in the second reference group [15], a significant cortisol induced retrieval impairment was detected ($t(26) = 3.65$; $p < 0.01$).

In both reference groups we had reported larger cortisol induced retrieval impairments for emotional (negative) than for neutral words [12,15]. In the relaxed group neither a main effect of emotionality ($F(1,30) = 1.44$; $p = 0.24$) nor a treatment by emotionality interaction ($F(1,30) = 1.28$; $p = 0.27$) occurred.

To examine if the effects of cortisol treatment differed depending on the treatment order (placebo first versus cortisol first) an ANOVA with the factors treatment and treatment order was calculated. There was no main effect of treatment order ($F(1,29) = 2.30$; $p = 0.14$) and, more importantly, no treatment by treatment order interaction ($F(1,29) = 0.99$; $p = 0.33$).

We calculated effect sizes for the retrieval effect in order to allow evaluation of the magnitude of the treatment effect and to allow the comparison with a recently published meta analysis of ours [9]. We used the formula provided by Hedges and Olkin [8] to calculate the effect size which is defined as the difference between the mean of the experimental group ($x/_{EG}$) and the control group ($x/_{CG}$) standardized by the pooled standard deviation [8]. Calculation was performed using the meta-analytic software program META [23]. According to Cohen [5] an effect size of 0.50 can be classified as moderate, while an effect size of 0.80 can be classified as large. The effect size for the first reference group was −0.66 and for the second reference group −0.85. In contrast, for the relaxed testing condition group the effect size was very small and positive (0.04).

In the cued recall condition no differences in the relaxed and two formal groups between the placebo and cortisol condition occurred (see Table 1). ANOVA with the factors treatment and test situation showed no main effect of treatment ($F(1,72) = 0.82$;

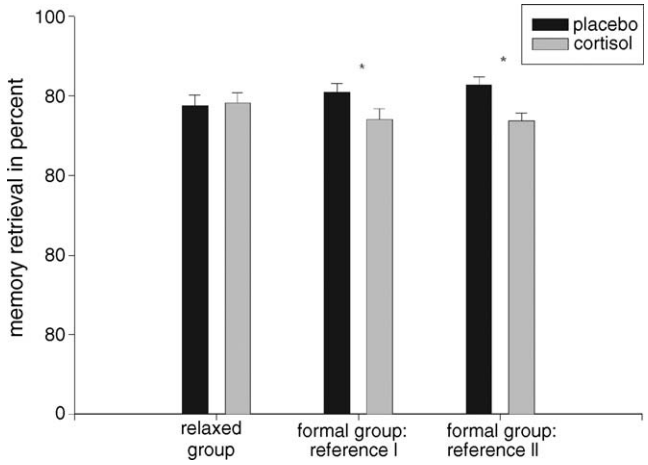


Fig. 1. Effects of oral cortisol treatment (30 mg) on memory retrieval (expressed in percent) in the relaxed testing group and the two formal testing reference groups. Results from the two reference groups are modified from two previous publications and are published with permission from the following references [12,15]. $^{*}p < 0.01$ difference in paired $t$-test. Error bars represent S.E.

$p = 0.37$) or treatment by situation interaction ($F(2,72) = 0.06$; $p = 0.94$) on cued recall performance.

As in the previous studies [12,15] cortisol treatment did not affect mood in the relaxed group (all $p$ values > 0.38). Therefore, we averaged the questionnaire results of the 2 test days. No significant differences between the relaxed group and the two formal groups occurred for the comparison good versus bad mood ($t(72) = 0.56$; $p = 0.58$), awake versus tired ($t(72) = 1.09$; $p = 0.28$) and calm versus restless ($t(72) = 0.17$; $p = 0.86$). On a descriptive level, subjects in the relaxed testing group reported to be less awake ($25.5 \pm 0.80$ compared to $26.9 \pm 0.93$).

The aim of the present study was to investigate whether a certain degree of arousal during the testing situation is necessary for the cortisol induced retrieval impairment. Consistent with our hypothesis we found that delayed memory retrieval was not impaired by cortisol in the relaxed testing condition whereas, as previously reported [12,15], in both formal conditions retrieval was significantly impaired. We were thus able to abolish the cortisol induced impairments in declarative memory retrieval by slight changes in the test setting. The findings show an interaction between arousal induced by the testing situation and the cortisol effects on delayed recall performance in humans. This might suggest that adrenergic activation is an essential step in mediating GC effects on memory retrieval in humans too.

The interpretation of non-significant results of course raises the issue of statistical power. The sample of the current study ($n = 31$) was substantially larger than that of most previous studies in this area [9]. In our two previous studies with the identical experimental timing we observed medium to large effect sizes ($-.66$ to $-.85$). We therefore calculated the power to detect such an effect with our current sample size using the program G-power [7]. This analysis revealed that our study had sufficient power (ranging from 82% for the 0.66 effect size to 95% for the 0.85 effect size).

Several investigations in rodents detected interdependent effects of corticosterone and adrenergic activation on memory performance. A recent study conducted by Roozendaal et al. [22] was able to show that injection of adrenoceptorantagonist in the BLA or the hippocampus prevented GC induced retrieval impairments. A further study observed similar effects with different handlings (habituation versus no habituation) of the animals during the testing situation [18]. Habituation of the animals prevented the GC effects on memory. This was interpreted as demonstrating that the level of arousal and accordingly adrenergic activation during training modulated effects of GCs on memory. The interpretation is similar to the conclusion drawn through our experiment: the arousal induced by the testing condition influences the GC dependent memory modulation.

Recent human stress studies examined the possible interaction between cortisol and arousal by investigating working memory during and after participation in a stress paradigm [6]. Cortisol stress responders showed impaired working memory performance during the stress paradigm only. Another recent stress study observed that stress induced cortisol elevations were associated with enhanced memory consolidation only in participants who reported negative affect [1]. These two studies, together with the current experiment, support the idea that emotional arousal is necessary for modulatory effects of cortisol on several memory systems in the human.

However, the present study has limitations which call for additional research. We have no direct proof for our assumption of higher adrenergic activation in the two formal previously published testing groups. We believe that the testing in a special lab and a specific cue like “we will test your memory performance now” causes higher arousal. In addition the somewhat noisy and busy waiting area ensured that the participants did not feel sleepy. However, mood assessment failed to reveal significant differences between the groups for the dimension awake versus tired, even though, on a descriptive level, subjects in the relaxed group reported being less wakeful.

In the relaxed testing condition the relationship between the experimenter and the women was more amicable because of the waiting time together in an office. Additionally, the office was less objectionable than the minimally furnished testing lab in the formal condition. Moreover, there was no explicit prior announcement that the memory testing would start soon and no walk towards the testing lab occurred. We do not know which of those modulations of the setting is most important for the differences between the relaxed and the formal groups. While we can argue that the arousal was lower in the relaxed group than in the formal groups a biological marker demonstrating this is missing in our study. Clearly the use of measures of sympathetic nervous system (SNS) activities is advisable in future studies on this topic. In the current study no such markers were collected.

In animals, novelty is an important factor influencing global arousal. As previously mentioned, habituation to the testing environment abolished the effects of GCs on memory in animals [18]. In our human study, novelty appeared to be a less important modulator, since the treatment order (cortisol first or placebo first) did not interact with the missing cortisol effects in this study as well as the occurring cortisol effects in the two previous studies [12,15].

Possible experimenter effects are another issue which have to be discussed briefly. In the current study, we once again used a double blind design, in order to prevent the possibility that the experimenter might involuntarily influence the subjects. Moreover, comparison of the average retrieval results and their variance, as well as performance in the other tests, revealed remarkably similar results within the three groups. This, in our view, disagrees with the notion that in the current study, the experimenter substantially influenced cognitive performance in general or retrieval performance in particular. Only in combination with the double blind applied cortisol-treatment was a specific effect of the test situation on delayed free retrieval apparent.

Our results on the impact of the testing condition might be able to explain the partly inconsistent results in human cortisol studies [9]. Even minor changes in experimental procedure appear to be able to blunt memory modulations after GC treatment. It seems likely that most researchers try to create a testing situation which is pleasing and relaxing for the participants to ensure that they experience the situation as less aversive as possible. It should be noticed that our results indicate that a very

calm situation might cause absent cortisol effects. We would encourage a careful description of the test setting for future pharmacological cortisol experiments since the circumstances might differ substantially between workgroups. This could be helpful for the explanation of divergent results.

We and others have shown that the emotional arousal of the learning material interacts with the effects of stress or cortisol treatment on memory retrieval [12,13] as well as memory consolidation [2,3,14]. It appears that the beneficial effects on consolidation as well as the impairing effects on retrieval are more pronounced for emotionally arousing material. Our present observation as well as those made by others in animals [21] and people [1,6] suggests that arousal induced by the testing condition and thus unrelated to the learning material itself also modulates the effects of GCs on memory.

In this study, we used an experimental design which, in two previous studies (both formal conditions), has been shown to reliably induce negative effects of cortisol on retrieval [12,15]. Again, only free cycling women were tested because a previous study of our workgroup showed that cortisol had only a minor effect on memory retrieval in women using oral contraceptives [15]. It remains to be shown whether subtle variations in the testing conditions also modulate the effects of cortisol on memory in men.

In sum, the present study documents that a more relaxed testing condition abolishes the negative effects of cortisol on memory retrieval in healthy young women. This finding might be explained by the reduced arousal of the participants of the relaxed testing condition probably leading to reduced noradrenergic activity. However, this assumption was not directly proven in our study. Future human studies should measure adrenergic activity and/or should pharmacologically influence beta receptors. Despite these limitations our study lends indirect supports to the hypothesis that adrenergic activation is a prerequisite for cortisol effects on memory in humans.

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