

induction via the Trier Social Stress Test. Following stress, participants reactivated previously seen neutral and emotional material. Recognition memory was tested 24 h later. Salivary cortisol and alpha-amylase responses to stress were assessed before, during and after stress induction.

Results: Dexamethasone and propranolol administration successfully suppressed the physiological stress response in participants. Memory reconsolidation following stress resulted in a better memory for neutral instead of emotional stimuli, while after pharmacological suppression of the stress response emotional and neutral memories were comparable.

Conclusions: Our results reveal the importance of context as it relates to social attention and memory formation, since the neutral faces of the audience during the TSST might have sensitized subjects to neutral faces during reconsolidation. These findings have important implications for understanding differential effects of situational and biological context (i.e. stress) on the underlying mechanisms associated with stress and its effects on attention and memory.

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Cortisol disrupts the neural correlates mediating extinction recall



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Extinction learning refers to the unlearning of a previously acquired association or response as a consequence of changing contingencies. However, extinguished responses may recover i.a. after a change in context indicating that extinction memory recall is sometimes prone to failure. Stress hormones have been implicated to modulate extinction processes, with mostly impairing effects on extinction recall. However, the neurobiological mechanisms mediating stress effects on extinction memory remain elusive.

In this functional magnetic resonance imaging study, we investigated the effects of cortisol administration on the neural correlates of extinction memory recall in a predictive learning task. In this task, participants were required to predict whether certain food stimuli were associated with stomach trouble when presented in two different contexts. A two-day renewal paradigm was applied in which an association was acquired in context A and subsequently extinguished in context B. On the following day, participants received either cortisol or placebo 40 min before extinction memory recall was tested in both contexts.

Behaviorally, cortisol impaired the retrieval of extinguished associations when presented in the extinction context. On the neural level, this effect was characterized by a reduced context differentiation for the extinguished stimulus in the ventromedial prefrontal cortex, but only in men. In the placebo group, ventromedial prefrontal cortex was functionally connected to the left cerebellum, the anterior cingulate and the right anterior parahippocampal gyrus to express extinction memory. This functional crosstalk was reduced under cortisol.

These findings illustrate that the stress hormone cortisol disrupts ventromedial prefrontal cortex functioning and its communication with other brain regions implicated in extinction memory recall.

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Cortisol levels in atypical and non-atypical depression using hair and saliva specimens



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Background: The identification of clear biological correlates in affective disorders has the potential to aid diagnosis and improve treatment stratification. Hypercortisolemia is a frequent finding in affective disorders, but there is not yet a clear consensus as to its utility as a biomarker in depression. Some inconsistency in previous results may have resulted from the use of different measures of cortisol, and a failure to differentiate between subtypes of depression. Thus, atypical depression is often found associated with hypocortisolemia rather than hypercortisolemia.

Aim: In this study, we measured cortisol levels in subjects with atypical (A-MDE) and non-atypical (NA-MDE) major depressive episodes and in controls, using both saliva and hair specimens – the latter representing a validated method for measuring long-term cortisol levels over the previous three months.

Methods: Clinical data, hair cortisol concentration (HCC) and six saliva specimens that were taken across the day for measuring four saliva measures were collected in a matched group of 40 controls, 27 A-MDE and 44 NA-MDE.

Results: A-MDE subjects reported a larger number of daily stressors and more symptoms overlapping with DSM Somatic Symptom Disorder clusters than NA-MDE (all $p < 0.05$). We did not find any difference in HCC between groups. However, A-MDE subjects showed reduced total daily salivary cortisol output (AUCg) compared to controls, 94.5 nmol/l, (s.d = 30.6 nmol/l) v/s 113.77 nmol/l, (s.d = 38.9 nmol/l), respectively, ($p = 0.04$).

Conclusions: Chronic cortisol levels measured using hair did not differentiate A-MDE or NA-MDE from each other or from healthy controls. The combination in A-MDE of low daily salivary cortisol output and normal hair cortisol could suggest low basal salivary cortisol but with a mid- to long-term neurobiological hyperreactivity producing transient generalized glucocorticoid hypersensitivity, the net result being normal “averaged” cortisol levels in hair. Atypical depression was found to share clinical features with somatic symptom disorders.

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