



Inverse relation between cortisol and anger and their relation to performance and explicit memory

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ABSTRACT

Cortisol has been found to increase in response to social evaluative threat. However, little is known about the cortisol response to induced anger. Thus, in the present study, we investigated the cortisol response to anger induction and its effects on performance and explicit memory. A variant of the Montreal Stress Imaging Task (MIST; [Dedovic et al., 2005](#)) was used to induce anger in 17 male and 17 female students. Consistent with previous observations, a significant decrease in cortisol was found from pre to post manipulation which was inversely related to increases in subjective anger. Moreover, whereas anger increase was related to impairments in performance, cortisol reduction was inversely related to cognitive performance and explicit memory (recall and recognition of persons' features in a social memory task). The adaptive value of an increase in cortisol in response to fear or uncontrollability and of a decrease in cortisol in response to anger will be discussed.

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Stress occurs in a situation in which a person needs to adapt to an increased situational demand so as to cope appropriately. In the case of a social evaluative threat, the hypothalamic-pituitary-adrenal (HPA) axis is activated and stress hormones, including cortisol, are released to supply the organism with additional energy to respond to the stressor ([Dickerson and Kemeny, 2004](#); [Sapolsky, 1992, 1996](#)). While social evaluation seems to be primarily associated with acute cortisol release, fear of physical injury or death seems to be predominantly associated with an increase in activity of the sympathetic nervous system (SNS) ([Herman et al., 2003, 2005](#)). However, not all negative emotions need necessarily be associated with an increase in activity of the stress systems. In fact, there is recent evidence that anger is related to reductions in the cortisol response rather than to increases in it ([Herrero et al., 2010](#); [Matheson and Anisman, 2009](#); but see [Moons et al., 2010](#)).

One possible reason for divergent effects of threat vs. anger on the cortisol response is that whereas threat elicits higher perceptions of risk and feelings of lack of personal control, anger may elicit optimistic perceptions of risk ([Lerner and Keltner, 2001](#)). Specifically, anger may trigger cognitive appraisals of certainty and control, which in turn lead to lower risk estimates. According to [Ortony et al. \(1989\)](#), anger is based on a cognitive interpretation that

an undesirable event has occurred whose cause is considered controllable and external. Results of EEG studies ([Harmon-Jones, 2003, 2007](#); see [Harmon-Jones et al., 2010](#), for a recent review) showing that anger activates frontal areas of the left cerebral hemisphere (a pattern of Alpha wave reduction in the EEG), which are associated with approach but not with avoidance behavior ([Harmon-Jones and Allen, 1998](#)), are compatible with this view. Likewise, [Carver \(2004\)](#) and [Carver and Harmon-Jones \(2009\)](#) consider anger to be a special type of negative emotion associated with the behavioral approach system (BAS), but not with the behavioral inhibition system (BIS).

The long-term effects of high cortisol levels on emotion regulation and cognitive performance (including declarative memory) are negative, especially when the level of stress is too high and not only mineralocorticoid but also glucocorticoid receptors in the hippocampus are engaged ([Dickerson and Kemeny, 2004](#); [Lupien et al., 2005](#)). These deficits have been attributed to lower efficiency of a chronically activated hippocampus, which is involved in endocrine stress regulation as well as in spatial and episodic memory ([Sapolsky, 1992](#)).

The effects of cortisol on emotion, cognition, and behavior, however, are complex, especially those related to momentary cortisol changes. For example, it has been proposed that chronically increased cortisol levels are maladaptive ([Dickerson and Kemeny, 2004](#); [Sapolsky, 1992](#)), given its adverse effects on cognitive performance and declarative memory ([Kirschbaum et al., 1995, 1996](#); [Wolf et al., 2004](#)). However, other research suggests that while extreme levels of cortisol (too much or too little) may be maladaptive, moderate levels may be considered adaptive (e.g., [de](#)

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Kloet et al., 1999). This notion reminds of the Yerkes–Dodson law of arousal–performance relationships and is also compatible with the hormetic theory of glucocorticoids on memory (Lupien et al., 2005). Specifically, the latter theory implies that increases or reductions in cortisol as a response to an acute stressor, within a given range, may be finely tuned to the requirements of the task at hand, be it in terms of physical or mental energy (i.e., effort). If this is the case, it is understandable that the short-term effects of high levels of cortisol as a result of stress may sometimes facilitate certain types of cognitive performance (de Kloet et al., 1999), such as remembering emotional information (Buchanan and Lovall, 2001; Cornelisse et al., 2011; Nater et al., 2007). If anger is related to cortisol reduction, it could be speculated that it may have a *negative* short-term effect on performance of complex cognitive tasks and declarative memory (see Nater et al., 2007). Accordingly, lower than average cortisol levels might be inversely related to cognitive performance and declarative memory and in turn, average levels of cortisol might improve this type of performance (de Kloet et al., 1999).

Although anger is considered a major stressor by some (see Spielberger and Sarason, 2005), very few studies have specifically investigated its effects on HPA axis activation and the cortisol response (e.g., Herrero et al., 2010; Matheson and Anisman, 2009). Matheson and Anisman (2009) investigated the effects of gender discrimination and mood priming of anger or shame on the cortisol response in two experiments with women. They found that whereas shame was associated with an increase of cortisol, anger was associated with a reduction of cortisol. More recently, Herrero et al. (2010) induced anger through a mood manipulation in a group of 30 men, and investigated its effects on cardiovascular, hormonal, and asymmetric brain activity. The anger manipulation consisted of reading descriptors of anger experiences (50 self-referent statements ranging from relatively neutral to those connoting irritability, hostility, rage and anger), recalling relevant personal memories, and evoking the mood suggested by the sentence/memory. They found that anger was associated with increased cardiovascular reactivity and with a reduction of cortisol levels. In addition, they also found that anger facilitated left-hemispheric processing as assessed by a dichotic listening test, which is consistent with the results of Harmon-Jones and associates (see Harmon-Jones et al., 2010).

In contrast to the results of the previous studies, Moons et al. (2010), using the Trier Social Stress Test (Kirschbaum et al., 1993), found that whereas post-stress anger predicted higher cortisol levels, post-stress fear predicted lower levels of cortisol. One objection to this study is that these authors did not directly manipulate anger or fear during the task, but only measured them retrospectively, using a post-stressor mood questionnaire. In addition, it is not clear whether the anger reported was “anger-in” (towards one-self for deficient performance) or “anger-out” (towards the judges or the experimenter). Finally, the absolute levels of anger were not reported which makes it difficult to evaluate the direct effect of anger on the cortisol response. In summary, with one exception, the previously performed studies indicate that the effects of fear and anger on the cortisol response might be diametrically opposed, with fear leading to increases in cortisol levels, and anger leading to decreases.

The aim of the current research is to investigate the relationship between anger and cortisol by first hypothesizing that anger is associated with reductions in the cortisol response. To induce anger, we used a modified version of the Montreal Imaging Stress Task (MIST; see Dedovic et al., 2005), which is described in Section 1. The MIST is normally applied to induce helplessness and evaluation anxiety, but it has the potential of inducing anger through bogus performance feedback. We also wanted to explore the effects of anger and its corresponding cortisol effects on cognitive performance and

declarative memory (recall and recognition), hypothesizing that it would be associated with lower performance.

1. Method

1.1. Sample and sociodemographic characteristics

Thirty-four undergraduate students, 17 women and 17 men (aged 19–45 years, $M = 24.5$; $SD = 5.8$), participated for course credit. The study was approved by the Ethics Committee of the University and all participants signed a written consent form before participating. One participant was excluded from the analysis because his baseline level of cortisol was 3 SD above the mean of the group on the experimental day. As assessed by self-report, none of them had hormonal problems or a depressive or other psychiatric disorder.

1.2. Materials

We measured affect using the Affect–Temperament Scales (ATS; Kuhl and Kazén, 2011; for validations studies, see Kazén, 2006 and Quirin et al., 2011), an analogous instrument to PANAS which measures the current mood using the term “Right now I feel:” via a Likert scale ranging from: (0) *not at all* to (3) *very much*. Each of the subscales has 3 items: *Anger* (annoyed, irritable, aggressive; Cronbach’s $\alpha = 0.79$); *Helplessness* (helpless, sad, gloomy; Cronbach’s $\alpha = 0.71$); *Distress* (strained, worried, tense; Cronbach’s $\alpha = 0.73$); *Relaxation* (calm, confident, relaxed; Cronbach’s $\alpha = 0.87$). We calculated sum scores for each scale (range: 0–9).

Cortisol values were assessed from saliva at the beginning of the experiment (baseline), and also at 25 and 45 min after the beginning of the MIST, by using the cotton swabs ‘Salivette’ (Sarstedt, Rommelsdorf, Germany) which participants chewed on and kept in their mouths for about 30 s. All saliva samples were frozen at -20°C until being assayed. The samples were sent to the laboratory of the Center for Psychobiological and Psychosomatic Research at the University of Trier in Germany. The cortisol levels were analyzed using a time-resolved immunoassay with fluorescence detection (for an exhaustive description, see Dressendorfer et al., 1992).

1.3. Procedure

The study took place in two sessions. In the first (group) session, participants filled out a battery of questionnaires. Participants were instructed to refrain from drinking alcohol or doing exercise on the day of the next (experimental) session, which would take place at least one week after the first session during the afternoon (from about 4:00 to 6:30 pm). They were also required not to eat, drink (except water), smoke cigarettes, or brush their teeth in the 2 h prior to participation. On the experimental day participants came in groups of 4–6, but were tested individually in isolated small cubicles furnished with a desk, a chair, and a computer. We recorded the time in which they started the session (“session time”) to control for circadian differences in cortisol level as a covariate. Participants were informed that they had to carry out a series of independent experimental tasks relating to different research questions, among them one on the effects of cognitive tasks on hormonal reactions measured via saliva samples.

The procedure of the second session is illustrated in Fig. 1. The experimenter instructed participants on how to sample saliva using the salivette, and asked them to chew on it for at least 30 s, and to deliver the first sample of saliva about 10–12 min before the beginning of the main task. After filling out the first mood ratings, participants had a social-memory encoding task. In this task, they were consecutively presented 16 photographs of unknown persons (50% women and 50% men, in a random sequence for each participant) with associated information (name, occupation, and age; e.g., John Smith, teacher, 30 years) for 10 s each. Participants were requested to pay attention to each person and learn not only the face but also all related information.

Next, participants carried out a modification of the MIST (see Dedovic et al., 2005) used for anger induction. Different from the original MIST, we did not use the rest or control conditions but only the experimental condition. As in the original version, participants consecutively carried out a series of 80 mental arithmetic tasks under time pressure (e.g., $(6 \times 4)/8 = ?$). Each solution consisted of a one-digit number. The correct solution to each task was given by pressing a key (from 0 to 9) on the keyboard. To induce time–pressure during task performance, the participant was shown a progress bar in the upper part of the monitor showing how much time was left to solve the task. When the allotted time ran out without a response, a message was shown (“Time Out”) and the participant was not allowed to respond anymore (cf. Dedovic et al., 2005, Fig. 1). We used percent correct calculations (i.e., number of correct responses/ 80×100) and frequency of time overshoots as performance outcomes.

This task has a social evaluative component because participants are asked to compare their performance with that of a comparison group and can see whether they performed below, equal to, or above average, as indicated by two colored arrows pointing to a performance feedback bar on top of the screen. In contrast to the original MIST, we manipulated the feedback given to participants. Specifically, they all received positive feedback during the first half of the tasks (1–40), which was relatively easy, and negative feedback during the second half of the tasks (41–80),

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