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Short communication

Exogenous cortisol causes a shift from deliberative to intuitive thinking

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ABSTRACT

People often rely on intuitive judgments at the expense of deliberate reasoning, but what determines the dominance of intuition over deliberation is not well understood. Here, we employed a psychopharmacological approach to unravel the role of two major endocrine stress mediators, cortisol and noradrenaline, in cognitive reasoning. Healthy participants received placebo, cortisol (hydrocortisone) and/or yohimbine, a drug that increases noradrenergic stimulation, before performing the cognitive reflection test (CRT). We found that cortisol impaired performance in the CRT by biasing responses toward intuitive, but incorrect answers. Elevated stimulation of the noradrenergic system, however, had no effect. We interpret our results in the context of the dual systems theory of judgment and decision making. We propose that cortisol causes a shift from deliberate, reflective cognition toward automatic, reflexive information processing.

1. Introduction

Have you ever jumped to an intuitive conclusion that later turned out to be wrong? If yes, the cognitive process you went through is well captured by the dual process theory, which has become a central framework in human judgment and decision-making research in recent decades (Kahneman, 2011). The dual system theory postulates the existence of two modes of information processing: one that is fast, intuitive and automatic, and another that is slow, analytical and reflective (Kahneman, 2011). As strong reliance on automatic processing is prone to cognitive biases and often leads to disadvantageous outcomes (Kahneman, 2011; Toplak et al., 2011), it is essential to determine which factors intensify its dominance. Here, we propose that endocrine stress markers tilt the balance of the two systems toward dominance of automatic processing.

Acute stress is characterized by parallel hypothalamic–pituitary–adrenal (HPA) axis and sympathetic nervous system activation, and elevated levels of neuromodulators cortisol (CORT) and noradrenaline (NA). Glucocorticoids (both externally administered and endogenous) are known to affect cognition by interfering with frontal-lobe-dependent functions, such as cognitive control, working memory, and selective attention and thereby weaken individuals' ability to discriminate between relevant and irrelevant information (al'Absi et al., 2002; Lupien and Mcewen, 1997; Lupien et al., 2007). The simultaneous action of exogenously administered NA and CORT has been shown to induce a shift from goal-directed toward habitual behavior (Schwabe et al., 2012), and evidence suggests that behaviorally induced stress alters decision making (Margittai et al., 2015; Starcke and Brand, 2012) for example by increasing susceptibility to decision biases (Porcelli and Delgado, 2009). Some of the reported findings are consistent with a stress-hormone induced bias toward automatic processing, however, such a causal link cannot be made without the employment of a task specifically designed to assess automatic versus deliberate processing, combined with a precise pharmacological manipulation of the two main stress neuromodulators.

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Using orally administered hydrocortisone and/or yohimbine (a drug that increases noradrenergic stimulation), we tested the extent to which endocrine stress markers impaired performance in the cognitive reflection task (CRT, Frederick, 2005), a well-established paradigm designed to quantify one's capacity to suppress intuitive, incorrect responses to simple arithmetic problems in favor of deliberate reasoning (Alter et al., 2007; Johnson et al., 2012; Pinillos et al., 2011; Toplak et al., 2011). CRT scores have been shown to predict performance in various areas of decision making and cognitive functioning (Campitelli and Labollita, 2010; Cokely and Kelley, 2009; Frederick, 2005; Hoppe and Kusterer, 2011; Liberali et al., 2012; Oechssler et al., 2009; Toplak et al., 2011).

Since increased levels of CORT and NA interfere with prefrontal functions responsible for cognitive control, required for deliberate reasoning, and upregulate limbic and subcortical mechanisms, such as amygdalar and striatal function, they are likely to increase dominance of automatic processes (Hermans et al., 2014). Based on these findings we expected that increased levels of stress modulators would impair performance on the CRT by causing a shift away from deliberate, toward intuitive thinking.

2. Method

2.1. Participants

Eighty-three men participated in this experiment (age: $M = 24.33$, SD = 5.94; see SOM for eligibility criteria, demographic and control measures. The four experimental groups did not differ in demographic and control measures, see SOM. We used an exclusively male sample in order to avoid differential HPA-axis activation caused by the intake of oral contraceptives and variations in menstrual cycle (Kirschbaum et al., 1999), thus our results are not generalizable to both genders and further research should be carried out in a female sample to be able to compare findings. Participants gave their informed consent. The experiment was approved by the medical ethics committee of the University Hospital Düsseldorf, and was carried out in line with the declaration of Helsinki.

2.2. Procedure

In a double-blind, placebo controlled between subjects design, participants were randomly assigned to one of four experimental groups: placebo ($N = 22$), placebo + yohimbine (an alpha2-adrenoreceptor blocker that stimulates noradrenergic activity; 20 mg, Chephlapharm, $N = 20$), placebo + hydrocortisone (cortisol agonist, 20 mg, Jenapharm, $N = 20$), or yohimbine + hydrocortisone (20 mg each, $N = 21$). After pill intake and a waiting period of 45 min (Schwabe et al., 2013a, 2012, 2010a), participants completed the CRT and two unrelated tasks (see SOM).

The CRT contains three short mathematical questions:

(1) A bat and a ball cost €1.10. The bat costs €1.00 more than the ball. How much does the ball cost?
(2) If it takes 5 machines 5 min to make 5 widgets, how long would it take 100 machines to make 100 widgets?
(3) In a lake, there is a patch of lily pads. Every day, the patch doubles in size. If it takes 48 days for the patch to cover the entire lake, how long would it take for the patch to cover half of the lake?

To take the example of the first question, the intuitive answer here is €.10, but the correct answer, requiring suppression of intuitive responding is €.05. Low CRT scores thus indicate increased difficulty in suppressing intuitive, incorrect answers.

2.3. Saliva sampling and further stress measures

Saliva samples were collected twice at baseline, from which an average was calculated as participants' baseline measure, and +30, +45 and +75 min after pill intake using Salivette devices (Sarstedt, Germany). Samples were frozen and stored at −20 °C until analysis for concentrations of salivary cortisol and alpha-amylase (a marker of noradrenergic activity) as reported by Rohleder et al. (2004). Values for unusable samples (8 out of 415) were replaced with the mean of the appropriate experimental group. Results remain significant even after exclusion of participants with one or more missing saliva samples ($N = 5$). Blood pressure was measured and subjective feelings of stress were assessed using visual analogue scales (VAS, 100 mm scale) at the same timepoints as the saliva samples.

3. Results

3.1. Manipulation check of the drug administration

Salivary cortisol increased after taking hydrocortisone, as shown by a mixed ANOVA (within-subject factor: timepoint, between-subjects factors: hydrocortisone (yes/no) and yohimbine (yes/no); main effect of hydrocortisone: $F_{1,79} = 103.85$, $p < .001$, $\eta^2 = .56$; timepoint x hydrocortisone interaction $F_{1.87,147.58} = 42.62$, $p < .001$, $\eta^2 = .26$). Yohimbine had no effect on cortisol levels, and the two substances showed no interaction ($p > .213$ Fig. 1A).

Salivary alpha-amylase concentrations were analyzed similarly, revealing increased levels in those participants who received yohimbine compared to those who did not (main effect of yohimbine: $F_{1,79} = 8.31$, $p = .005$, $\eta^2 = .095$; yohimbine x time interaction $F_{1.92,151.53} = 6.48$, $p = .002$, $\eta^2 = .067$). Hydrocortisone had no effect on alpha-amylase levels, nor was there an interaction between the two substances (both $p > .563$ Fig. 1B).

3.2. Blood pressure measures

We measured blood pressure using Sanitas SBC-41 wrist monitors at the same timepoints as the saliva samples. Due to device malfunction measures from one participant could not be obtained. Three separate mixed ANOVAs for the systolic, diastolic and pulse measures (using the same factors as detailed in the ANOVAs under saliva samples) revealed a main effect of timepoint on pulse ($F_{2.45,190.95} = 2.91$, $p = .046$, $\eta^2 = .017$), indicating that pulse increased somewhat in all groups over the course of the experiment, however neither pulse nor blood pressure were affected by either yohimbine or hydrocortisone (all $p > .106$).

3.3. Subjective feelings of stress

We measured participants' subjective feelings of stress using VAS scales at the same timepoints as the saliva samples and blood pressures. A mixed ANOVA with the same factors as reported above revealed significantly increased levels of subjective feelings of stress over time in those who received yohimbine (timepoint x yohimbine interaction: $F_{2.52,198.83} = 4.96$, $p = .004$, $\eta^2 = .060$). There was no effect of hydrocortisone manipulation, nor an interaction between the two substances on subjective feelings of stress (all $p > .130$), which is in line with reports indicating that cortisol administration often does not cause changes in subjective affect or mood (Putman and Roelofs, 2011).

3.4. Cortisol causes a shift from deliberate to intuitive thinking

To test how cortisol and noradrenaline actions impact cognitive reflection in isolation and by interaction, we employed a 2×2 ANOVA with hydrocortisone (yes/no) and yohimbine (yes/no) as

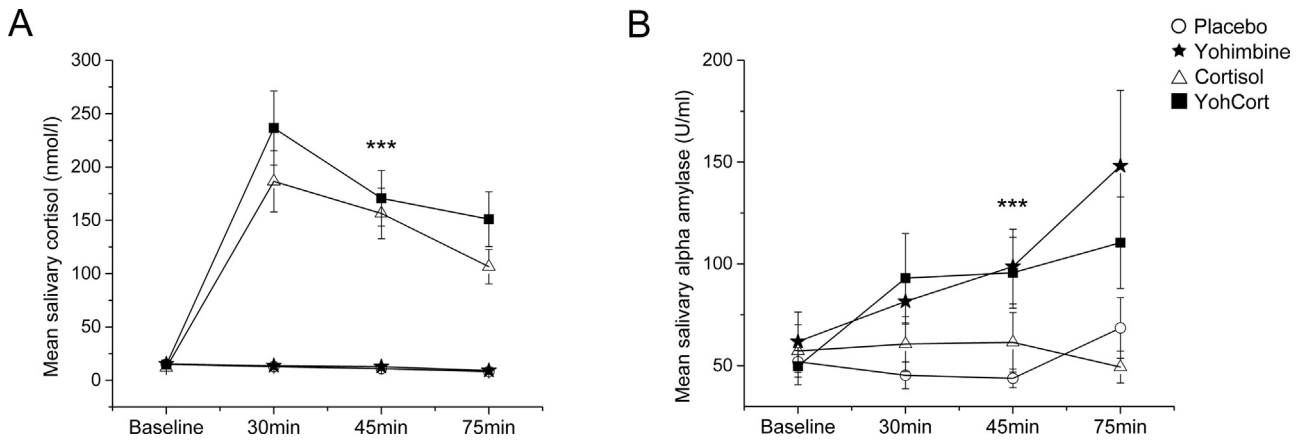

Fig. 1. Concentrations of salivary cortisol and alpha-amylase after hydrocortisone and yohimbine intake. The CRT task was performed between 45 and 75 min after pill intake. Error bars represent ±1 SE. (A) Participants taking hydrocortisone had significantly increased salivary cortisol concentrations after pill intake before the CRT task ($^{***}p < .001$). (B) Salivary alpha-amylase levels significantly increased after yohimbine intake. ($^{***}p = .005$).

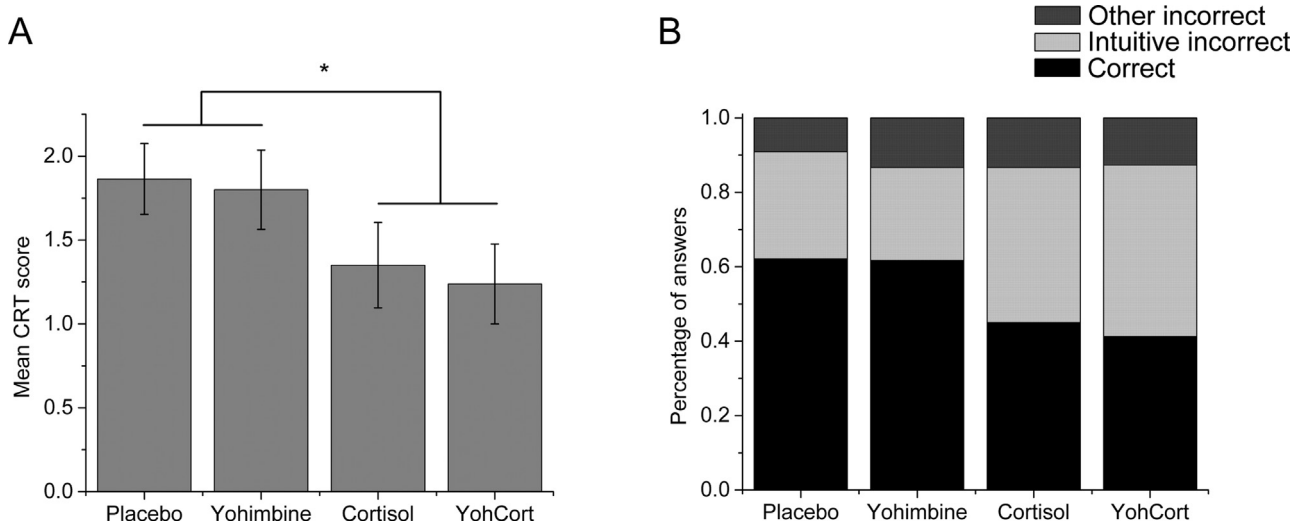

Fig. 2. CRT performance in the different treatment groups. (A) Individuals who received hydrocortisone showed decreased performance compared to those who did not (error bars represent ±1 SEM), $^{*}p < .05$. (B) Percentage of correct, intuitive incorrect and other incorrect answers in the experimental groups.

between subject factors. This analysis revealed that individuals who received cortisol had significantly lower CRT scores than those who did not (main effect of hydrocortisone, $F_{1,79} = 5.25$, $p = .025$, $\eta^2 = .062$, Fig. 2A). There was neither a significant effect of yohimbine, nor a significant interaction between the two substances on CRT scores (both $p > .709$).

Moreover, participants who received cortisol showed decreased rates of correct (=deliberate) answers, paralleled by increased rates of intuitive incorrect answers ($\chi^2(1, N = 219) = 9.21$, $p = .002$), while the proportion of other incorrect replies did not differ between conditions ($Z = -.46$, $p = .65$). This demonstrates that cortisol affected the CRT by inducing a genuine shift from deliberative to intuitive thinking, rather than by simply making the problem solving process noisier (Fig. 2B).

3.5. Belief about treatment

After the experimental tasks we asked participants to indicate whether they believe to have received placebo or an active substance during the experiment. The number of participants who believed to have been given placebo versus active substances differed between the four treatment conditions on trend-level ($\chi^2(3, N = 83) = 5.48$, $p = .061$, Cramer's $V = .30$). However adding individuals' belief about the treatment as a covariate in our main analysis does not alter our findings (main effect of CORT on CRT scores: $F_{1,78} = 5.15$, $p = .026$, $\eta^2 = .062$).

4. Discussion

We demonstrated that pharmacologically elevated cortisol, but not noradrenaline, levels impaired performance in the CRT by increasing reliance on intuitive over deliberate judgments. We thus provide direct, causal evidence that cortisol is likely involved in setting the balance between automatic and deliberate thinking. Our findings provide two novel insights that delineate the effects of stress neuromodulators on human judgment and decision-making. First, we used a task that is specifically designed to test the engagement of intuitive versus deliberate reasoning, allowing us to make a direct conclusion about the involvement of the two information processing systems. This complements prior studies that used more complex decision making, reasoning and cognitive tasks (Porcelli and Delgado, 2009; Putman et al., 2010) which only allowed indirect conclusions about the involvement of automatic versus deliberate processing as an explanation for their findings. Second, by employing a direct, pharmacological manipulation we were able to provide causal conclusions about how endocrine stress mecha-

nisms influence cognition while excluding confounding factors that accompany paradigms in which stress is induced behaviorally, such as the element of social evaluation or physical pain (Dickerson and Kemeny, 2004; Porcelli and Delgado, 2009).

Stress-related increases in cortisol and noradrenergic action follow distinct temporal profiles: while cortisol in concert with noradrenaline synergistically and transiently modulate neural activity and cognition during an initial fast-acting wave of stress-neuromodulators, noradrenergic action wears out within minutes after stress onset, and the brain is mainly influenced by slower cortisol effects alone in the aftermath (Joëls et al., 2011; Schwabe et al., 2012). Our experimental design allows dissociating the functional difference of the combined and isolated effects of cortisol and noradrenaline on cognitive reflection. Our results suggest that deliberate thinking is affected by cortisol alone, and that this effect is not moderated by noradrenergic activity. This extends recent findings indicating that cortisol biases the engagement of different cognitive systems (Schwabe and Wolf, 2013) and complement prior evidence that the pharmacological blockade of a receptor for cortisol abolishes the stress-induced shift from cognitive to habit memory in spatial and classification learning, pointing also to a crucial role of cortisol in the modulation of flexible cognition (Schwabe et al., 2013b, 2010a). By contrast, in instrumental learning, the stress-induced bias toward habit performance has been shown to require cortisol actions in concert with noradrenergic activity (Schwabe et al., 2010b). Our results complement these data by showing that higher-order cognitive reflection is cortisol-dependent, but, unlike reinforcement learning, it is independent of fast-acting noradrenergic action.

As response patterns in the CRT correlate with behavior in various domains of decision making and cognition (Campitelli and Labollita, 2010; Cokely and Kelley, 2009; Frederick, 2005; Hoppe and Kusterer, 2011; Liberali et al., 2012; Oechssler et al., 2009; Toplak et al., 2011), our findings have broader implications for the influence of cortisol on reasoning, decision making and cognitive function. For example, because the CRT is known to predict behavior in decisions under risk, our results may extrapolate to existing findings on the effects of stress-modulators on risky decisions (Coates and Herbert, 2008; Kandasamy et al., 2014; Pabst et al., 2013; Porcelli and Delgado, 2009; Putman and Roelofs, 2011; Van den Bos et al., 2009) and make a first step toward providing a common mechanism through which stress-modulators affect economic decisions. Additionally, as CRT performance correlates with performance on heuristics-and-biases tasks (Toplak et al., 2011), predicts susceptibility to behavioral biases, such as overconfidence, conservatism bias and endowment effects (Hoppe and Kusterer, 2011), and is associated with individual differences in probability judgement (Liberali et al., 2012) our findings generate novel testable behavioral hypotheses regarding the effects of cortisol on everyday decision and judgement fallacies.

The exogenous drug administration used in the present study carries the benefit of allowing causal conclusions about the involvement of the major stress mediators cortisol and noradrenaline in cognitive reflection. However, it is important to note that physiological reactions to pharmacological manipulations are not necessarily identical to those occurring after a natural stress situation, and might result in, for example, supraphysiological levels compared to natural stressors (Lupien et al., 1999), or might lack the affective response to threatening situations. Hence, it remains to be shown whether natural stressors have similar effects on deliberative thinking.

In conclusion, we demonstrated that exogenously administered cortisol impairs cognitive reflection and potentiates a shift from deliberate to intuitive information processing. We provide causal evidence of one mechanism through which stress impairs human judgement and decision-making that could explain several findings of how stress fosters decision biases in several areas of economic decision making.

Author contributions

All authors contributed to the study design and developing the concept. Z. Margittai performed testing and data collection. Z. Margittai, G. Nave, T. Strombach and T. Kalenscher performed the data analysis. Z. Margittai prepared the manuscript and all authors provided critical revisions. All authors approved the final version of the manuscript before submission. Z. Margittai and G. Nave are joint first authors of this article. T. Kalenscher supervised the project.

Conflicts of interest

The authors declare no conflict of interest.

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.psyneuen.2015.11.018.

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