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The effect of hydrocortisone administration on intertemporal choice☆

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ABSTRACT

Intertemporal choices – decisions involving trade-offs of outcomes at different points in time – are often made under stress. Stress activates the hypothalamic–pituitary–adrenal (HPA) axis, resulting in the release of corticosteroids. Recent studies provide evidence that corticosteroids can induce rapid non-genomic effects focused on immediate resolution of the stressful situation, followed by slower genomic effects focused on long-term recovery after stress. It remains unknown, however, how corticosteroids affect intertemporal choice. We randomly assigned healthy men to receive either 10 mg hydrocortisone or a placebo before measuring intertemporal choice. To target time-dependent effects, hydrocortisone was administered either 195 or 15 min before choice elicitation, while a placebo was administered at the other timepoint, in a double-blind design. Intertemporal choices were elicited by offering subjects decisions between small rewards available sooner vs. large rewards available later. We demonstrate a time-dependent effect of hydrocortisone administration on intertemporal choice: when tested 15 min after hydrocortisone administration, subjects showed a strongly increased preference for the small, soon reward over the larger, delayed reward. In contrast, this effect was not found when testing occurred 195 min after hydrocortisone administration. Together, these results suggest that the physiological effects of acute, but not delayed, stress may increase temporal discounting.

1. Introduction

Stress is a prominent feature of everyday life, and people frequently make important economic decisions under its influence. Recent research has begun to ask whether stress causally affects economic choice; existing evidence suggests that acute stress may affect productivity (Angelucci and Córdova, 2014), risk preferences (Kandasamy et al., 2014; Porcelli and Delgado, 2009; Delaney et al., 2014; Bendahan et al., 2016), and social preferences (von Dawans et al., 2012; Vinkers et al., 2013). Here, we focus on the effect of the stress hormone cortisol on temporal discounting, i.e. the decrease in the subjective value of a reward when it is delayed.

The motivation for our study is two-fold. First, existing studies have produced inconclusive results concerning the effect of stress on discounting: Koppel et al. (2017) find increases in discounting after inducing physical stress in the form of heat pain; Delaney et al. (2014) find increases in temporal discounting after exposure to the Cold Pressor Task, a physical stressor consisting of holding one's hand in cold water. In contrast, we have previously found no effects of the Cold Pressor Task and a social stressor, the Trier Social Stress Test, on temporal discounting (Haushofer et al., 2013, 2015). One possibility for these discrepant findings is that the different induction methods lead to a host of physiological changes, not only in cortisol, but also in adrenaline and noradrenaline levels and heart rate. In this paper, we isolate one of these mechanisms by pharmacologically increasing levels of the stress hormone cortisol in a laboratory study. This manipulation allows us to ask whether increased levels of the stress hormone cortisol causally affect temporal discounting.

Second, recent evidence suggests that cortisol acts differentially on the organism over short and long time horizons through rapid and slow mechanisms, respectively. In particular, shortly after stress, corticosteroid actions interact with the neurotransmitter noradrenaline

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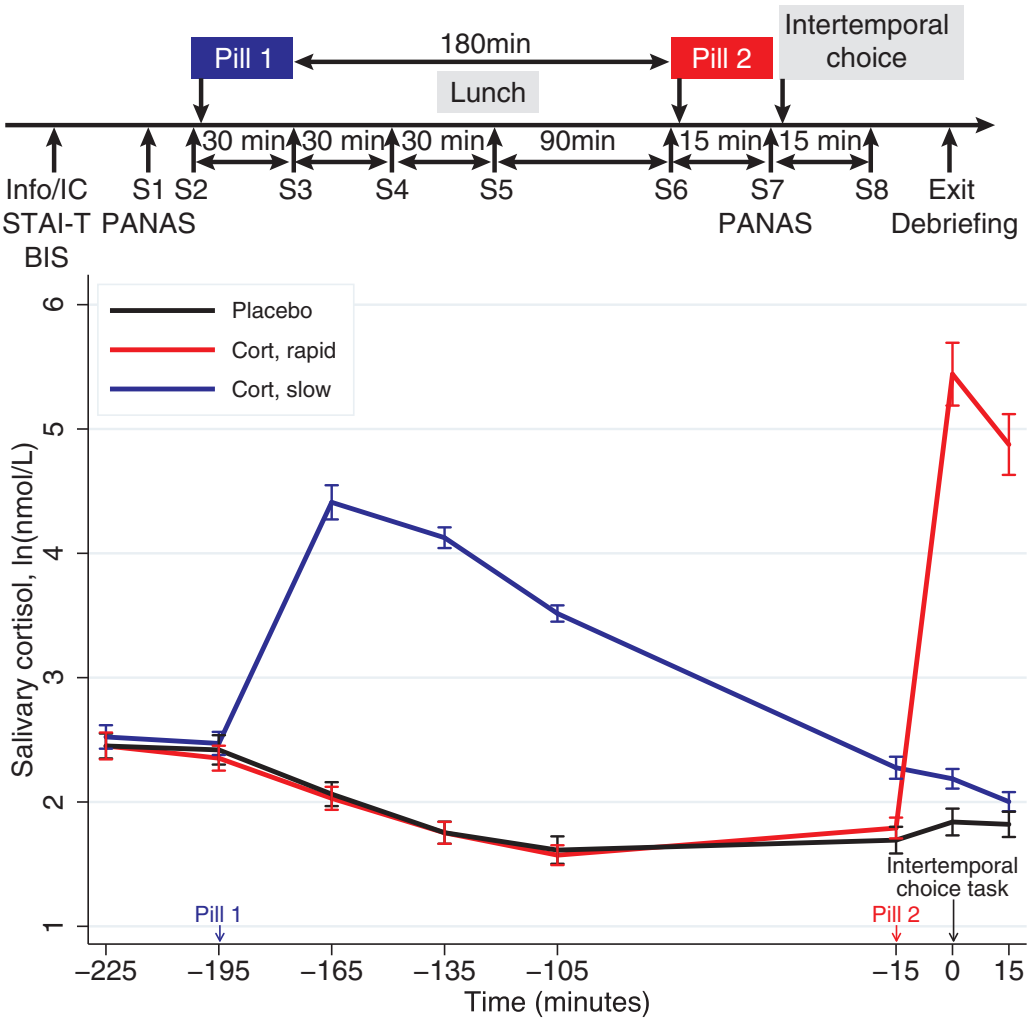

Fig. 1. Panel A: Timeline of the experiment. Upon arrival, participants read the information brochure (Info), filled out the informed consent (IC) and several baseline surveys. As a manipulation check of the hydrocortisone manipulation, saliva samples were taken throughout the experiment (S1–S8). Time between the samples was fixed and is indicated with arrows. The first and second pills (Pill 1 and Pill 2) were administered directly after S2 and S6, respectively. Positive and negative affect (PANAS) was measured at the same time as S1 and S7. The intertemporal choice task took place immediately after S7. Panel B: Salivary cortisol levels in the three treatment groups over time. Error bars represent one standard error of the mean (SEM). Participants received a pill 195 min (pill 1) and 15 min (pill 2) prior to the intertemporal choice task ($t = 0$). The pill either contained 10 mg hydrocortisone or placebo (albochin). In the "rapid cort" group, the first pill was placebo and the second hydrocortisone; the "slow cort" group, the first pill was hydrocortisone and the second placebo; in the "placebo" group, both pills were placebo. Saliva samples were taken at 225, 195, 165, 135, 105, 15 and 0 min before intertemporal choice, and 15 min after. Significant Bonferroni-corrected differences with placebo are depicted by *** = $p < 0.005$. Hydrocortisone administration in both groups significantly elevated salivary cortisol as compared to placebo, but did not differ immediately before each pill intake.

to synergistically promote rapid increases in neuronal activity (Karst et al., 2010, 2005). These rapid corticosteroid effects have been described for emotion- and arousal-related brain areas, such as the amygdala (van Marle et al., 2010; Hermans et al., 2011), and are thought to promote emotional and reflex-like behavior (Schwabe et al., 2010; Henckens et al., 2012) and attention (Vedhara et al., 2000), at the expense of goal-directed behavior (Schwabe et al., 2010) and higher cognitive functioning (Elzinga and Roelofs, 2005). This mechanism may help the organism to focus on the most salient, habitual aspects of an event (Roozendaal et al., 2006), at the cost of the more complex, cognitive aspects.

By contrast, delayed effects of cortisol on the brain are thought to restore homeostasis following episodes of stress (Diamond et al., 2007; Joëls et al., 2006). Through changes in gene transcription that require about 1 h to develop and last for several hours (Datson et al., 2008), stress-induced corticosteroid actions shut down the effects of noradrenaline (Pu et al., 2007, 2009; Joëls and de Kloet, 1989) and change neuronal activity in frontal brain regions such that, among other effects, the stress-induced release of hormones from the pituitary is terminated (Hill et al., 2011; Yuen et al., 2009). Behaviorally, these slower genomic effects promote consolidation (Barsegyan et al., 2010), contextual memory (Oitzl et al., 2001) and enhance working memory (Henckens et al., 2011), promote sustained attentional processing (Henckens et al., 2012), and strengthen connectivity between the PFC and amygdala (Henckens et al., 2010a). Thus, slower genomic corticosteroid effects may facilitate processing and remembering a stress episode in a cognitively controlled manner and allow the organism to learn from it for the future. Relatedly, it has recently been shown that stress has a time-dependent effect on both social (Vinkers et al., 2013; Margittai et al., 2015) and risk preferences (Bendahan et al., 2016). Thus, the early behavioral responses to cortisol are thought to shift the focus to the present, while the slower actions of cortisol are thought to prepare the organism for the future. We therefore hypothesized that cortisol would increase discounting shortly after administration, and decrease it at longer intervals after administration.

To test our hypotheses, we combine measures of intertemporal choice with pharmacological manipulation of the stress hormone cortisol. Specifically, we administered either placebo or hydrocortisone to healthy human participants, and then asked them to complete a temporal discounting task. Participants were divided into three groups: the "rapid cort" group received 10 mg of hydrocortisone 15 min before completing the discounting task; the "slow cort" group received 10 mg of hydrocortisone 195 min before the task. These times were based on previous evidence on the non-genomic and genomic effects of cortisol (Henckens et al., 2010b; Joëls and de Kloet, 1992; Morsink et al., 2006; Joëls et al., 2003). Both groups received a placebo pill at the respective other timepoint. The placebo group received placebo pills at both timepoints. Administering hydrocortisone at two different timepoints with respect to the discounting task allows us to trace out the time-course of the effect.

2. Experimental design

2.1. Participants

Seventy-nine male participants gave informed consent. Sample size was determined by a power analysis (power > 0.80; $\alpha = 0.05$) for detecting medium (Cohen's *d* ranging from 0.50 to 0.80) effects. *Ex post* minimum detectable effect sizes (MDE) are shown in Supplementary Materials B. The local ethical committee of the University of Amsterdam approved the study. Inclusion criteria as assessed by self-report were: no past or present psychiatric or neurological condition, and age between 18 and 35 years. Participants were asked to refrain from taking any drugs three days prior to participation, and to get a night of proper sleep, refrain from heavy exercise, alcohol and caffeine intake 12 h prior to participation, and not to eat, drink, smoke, or brush teeth 2 h before participation. We excluded one participant due to violation of the requirements, leaving us with 78 participants for analysis. Participants received a show-up fee of €30, which could alternatively be exchanged for course credit; in addition, a single trial of the intertemporal choice task was randomly chosen for payment (maximum €20, minimum €5). Due to a restriction imposed by the human subjects committee, all participants were paid the entire amount on the day following the experiment. We took two approaches to preserve the integrity of the intertemporal choice task while avoiding deception. First, no information was given about the timing of the payment before the experiment. Second, we include a robustness check where we control for whether the respondents believed that they would receive the chosen amount at the corresponding delay.

2.2. General procedure

In a between-subjects, placebo-controlled, double blind study design, participants were randomly assigned to either the rapid cort (hydrocortisone 15 min prior to testing) or slow cort (hydrocortisone 195 min prior to testing) or placebo group (see experimental outline in Fig. 1). Testing took place in between 12 pm and 8 pm, when endogenous cortisol levels are stable and relatively low (Pruessner et al., 1997).

Upon arrival at the lab, participants read an information brochure, were interviewed to assess eligibility for participation, and provided informed consent. Baseline self-reported mood state was assessed with the Positive Affect and Negative Affect Schedule (PANAS; Watson et al., 1988); state and trait anxiety were assessed with the State Trait Anxiety Inventory (STAIS/T; Spielberger, 2010); and a first baseline saliva sample was collected.

Directly following a second baseline saliva sample, participants received their first pill (cortisol or placebo). A 3 h waiting period followed, during which participants either read or studied in the same room, were provided lunch, and four more saliva samples were obtained at regular intervals (see Fig. 1). The second pill (cortisol or placebo) was given 3 h after the first. A second resting period of 15 min followed to allow cortisol plasma levels to reach their peak following administration for the rapid cort group (Czock et al., 2005). Participants then gave another saliva sample and again filled out the mood questionnaires (PANAS and STAIS), followed by the intertemporal choice task and the Barratt Impulsiveness Scale (BIS; Patton et al., 1995). A final saliva sample was taken, and a post-experimental questionnaire assessed (1) whether participants believed that their monetary decisions would indeed be rewarded with the promised amount at the promised time, (2) whether participants knew which substance they had received at what time.

2.3. Drug administration and assessment

Hydrocortisone and placebo (albochin) were administered through identically appearing pills. A single dose of 10 mg of hydrocortisone was employed to elevate endogenous cortisol to a level equivalent to moderate acute stress (Abercrombie et al., 2003). Salivary free cortisol concentrations were measured using saliva samples collected at 8 timepoints using salivettes (Sarstedt, Germany; Fig. 1). The salivettes were stored at −25 °C and analyzed using a chemiluminescence immunoassay (CLIA) with sensitivity of 0.16 ng/ml (IBL, Hamburg, Germany) by Technische Universität, Dresden, Germany.

The motivation for the timing of the rapid and slow corticosteroid conditions was similar to that of a previous study by Henckens et al. (2011), briefly recapitulated here. First, existing work has shown an elevation in human salivary cortisol levels in the first half-hour after administration (Henckens et al., 2010b), and non-genomic actions develop within minutes after corticosteroids reach the brain (Karst et al., 2005). In contrast, the genomic effects of corticosteroids begin 3 h after administration at the earliest (Morsink et al., 2006; Joëls et al., 2003). Thus, the delay of 15 min was chosen to capture the rapid effects of hydrocortisone before gene-mediated effects could arise, whereas the delay of 195 min was chosen to capture gene-mediated effects after the hormone levels themselves had returned (close) to baseline.

2.4. Intertemporal choice task

Participants performed 42 trials (6 blocks of 7) of an intertemporal choice task in which they made decisions between a sooner smaller reward and a later larger reward. The delay combinations for the sooner date, *t*, and the later date, *T*, were (0, 3), (0, 6), (0, 9), (0, 12), (6, 9), and (6, 12), where zero refers to “tomorrow”, and each other number refers to that number of months plus one day. The large reward was constant at €20, while the sooner smaller reward started at €10 and was then adjusted with a titration method (bisection algorithm) according to the choices the participant made (Mazur, 1988; Falk et al., 2016): for patient choices, the small-soon amount was increased, for impatient choices, it was decreased. Possible serial correlation and order effects in participants' responses were averaged out by randomizing the order of blocks, i.e. the order in which the various indifference points were determined. In addition, the side of the screen (left or right) on which the “late” and “soon” options were presented on each trial was randomized across trials. This procedure was repeated six times for each delay combination to identify an indifference point, yielding a precision of €0.0781. Every seventh trial was a repeat of the first one for that delay combination to measure consistency. The amount of the sooner reward at the end of this titration procedure was taken as the *indifference point* for the particular delay combination, i.e. the amount where participants were indifferent between receiving the sooner and later reward offered. Supplementary Table S4 shows that participants did not differentially engage in strategic behavior by condition, i.e. attempting to “game” the titration method by choosing the impatient option in the first iteration.

We consciously chose not to obtain baseline measures of discounting. The reason for this choice is that completion of the baseline tasks potentially contaminates the endline task because (a) participants might have preferences for consistency, leading to an underestimation of the treatment effect; (b) there might be learning effects; (c) incentivization is not straightforward with baseline and endline measures (if one trial from each is incentivized, endowment effects may contaminate the endline measurement; if only one trial in total is incentivized, participants may pay less attention to each individual trial). However, note that given randomization into treatment groups, endline comparison of any measure across treatment groups provides an unbiased estimate of the treatment effect.

2.5. Statistical analysis

Details on the statistical analyses are reported in the Supplementary Materials and Methods. Briefly, we assessed the effect of hydrocortisone administration on intertemporal choice by regressing each indifference

point on indicator variables for the rapid and slow cort treatments. Because each participant contributed 6 indifference points, we used seemingly unrelated regression (SUR) to jointly estimate a system of 6 equations. Supplementary Table S5 shows that the results were robust to estimating the equations individually using ordinary least squares (OLS) regression. As a robustness check, we also estimated the model with the inclusion of control variables, i.e. baseline positive/negative affect, state anxiety, anxiety sensitivity, and debt, which showed differences across groups at baseline; these results are reported in Supplementary Table S8.

Intertemporal choice can be decomposed into *impatience* and *present bias* (often referred to as *hyperbolicity*), where the former refers to an exponential decrease in subjective value with delay, and the latter to disproportionate value being attached to immediate outcomes (Laibson, 1997). Present bias is of particular interest to both scientists and policymakers because it implies time inconsistency, i.e. procrastination on negative outcomes (e.g. tasks) and impulsivity on rewards.

We therefore next estimated a non-structural model for present bias. We calculated present bias as the difference in indifference points for a given delay when the sooner date was "tomorrow" relative to when it was "6 months from now": more discounting over the same time horizon without a front-end delay than with a front-end delay is evidence of present bias. We performed the same analyses described above using this non-parametric measure of present bias.

Finally, we used the quasi-hyperbolic model of time preferences proposed by Laibson (1997), in which the utility at time t of a payment x at time T is modeled as follows:

$$U_t(x(t, T)) = \begin{cases} \delta^{T-t}u(x) & \text{if } t > 0 \\ \beta\delta^{T-t}u(x) & \text{if } t = 0 \end{cases}$$

This model is the most widely used discounting model in the economics literature; it is useful because it allows us to separately estimate parameters for impatience (δ) and present bias (β).[1] If $\beta = 1$, the individual is an exponential discounter; with $\beta < 1$, the individual is present biased, i.e. time-inconsistent. Again we estimated this model with and without the inclusion of control variables, using non-linear least squares.

2.6. Baseline comparison

Supplementary Table S1 reports baseline balance on a number of variables between the three treatment groups. Each column is the result of a regression of the baseline variable on indicator variables for the rapid and slow cort treatments. Placebo is the omitted category. Columns (1)–(4) present variables that are expected to biologically affect cortisol: age, body mass index, baseline cortisol levels, and whether or not the participant broke the study requirements of not eating or drinking 2 h before the beginning of the study. Columns (5)–(7) present economic variables that may affect intertemporal choices through budget constraints and preferences for consumption smoothing (see e.g. Carvalho et al., 2016): a dummy variable for whether the recipients expected their income to change in the immediate future, disposable income, and one for whether the recipient had debt. Finally, columns (8)–(12) include psychological variables that might affect intertemporal choices (see e.g. Lerner et al., 2013): positive and negative affect (measured by the PANAS), anxiety sensitivity (measured by the Anxiety Sensitivity Index), and state and trait anxiety (measured by the State-Trait Anxiety Inventory). We found a significant difference between the rapid cort and placebo groups in negative affect, and a marginally significant difference in state anxiety; and between the rapid and slow cort groups in debt and anxiety sensitivity, and marginally significant differences in positive/negative affect and state anxiety. There were no significant differences between the slow cort and placebo groups. Because the main comparison of interest is that of the hydrocortisone to the placebo groups, we control for negative affect and state anxiety in the main analyses, and include the remaining variables as additional controls in the robustness analyses described above. These results are reported in Supplementary Table S8.

[1] Other discounting functions could have been used as well (e.g. Takahashi, 2009).

3. Results

3.1. Manipulation check

Supplementary Table S2 and Fig. 1 display the salivary cortisol levels for the three experimental groups during the experiment. As expected, the average cortisol level for the participants in the rapid and slow cort groups was significantly elevated relative to that of the placebo group after they received the active pill. The relatively larger increase in cortisol levels for the rapid cort group between salivettes (6) and (7) compared to that of the slow cort group between (2) and (3) was due to the difference in timing of the first cortisol measure after the administration of the pill (15 min for the rapid cort group, 40 min for the slow cort group). Importantly, at the time of the intertemporal choice task (immediately after the seventh cortisol measure at minute 215), the average cortisol level for the rapid cort group was higher than that of the placebo group. The same was true for the slow cort group, but the absolute value of the cortisol elevation of this group relative to the placebo group, 2.27 nmol/l, was low in both absolute and relative terms. Furthermore, this difference became statistically insignificant within the duration of the intertemporal choice task (between minutes 215 and 230).

3.2. Intertemporal choice performance

3.2.1. Non-parametric estimation of treatment effects on indifference points

Fig. 2 displays the intertemporal choice performance (mean indifference points) for the three experimental groups, and Table 1 reports the results from the non-parametric regression analysis without (top panel) and with control variables (lower panel). The constant term reflects the average indifference point in the placebo group for each delay condition (e.g., the average indifference point in the placebo group when choosing between outcomes tomorrow and 3 months from tomorrow was €16.00). The coefficients for the rapid cort group and the slow cort group represent the difference in indifference points between the placebo group and each treatment group.

The left panel of Fig. 2 shows that participants discounted future outcomes: for all three experimental groups, the average indifference points decreased as a function of time. Comparing the left and the right panels of Fig. 2, we see that participants also exhibited present bias, i.e. more discounting with than without a front-end delay.

Fig. 2 and the upper panel in Table 1 show that the indifference points for the rapid cort group were lower than those of the placebo group, indicating higher discounting. The point estimates for this difference ranged from €1.91 for $(t, T) = (0, 3)$ (a difference of 11.94%) to €2.89 for $(t, T) = (0, 6)$ (a difference of 20.11%), and the differences were statistically significant at the 5% level for three blocks, $(t, T) = \{(0, 6), (6, 9), (6, 12)\}$, marginally statistically significant ($0.05 < p < 0.10$) for two blocks, $(t, T) = \{(0, 9), (0, 12)\}$, and statistically insignificant for one block, $(t, T) = (0, 3)$. When averaging indifference points across blocks within each participant, the coefficient for the rapid cort group, $\beta = -2.27$ (*s. e.* $= 1.05$), was statistically significant at the 5% level. Thus, hydrocortisone administration increased discounting immediately after administration. Fig. 3 presents kernel density estimates of this effect, i.e. smoothed estimates of the distribution of indifference points across the three experimental groups. It can be seen that hydrocortisone shifted the entire distribution of intertemporal choices. There were no statistically significant differences between the indifference points of the slow cort group and the placebo group, indicating that the effect of hydrocortisone on discounting had

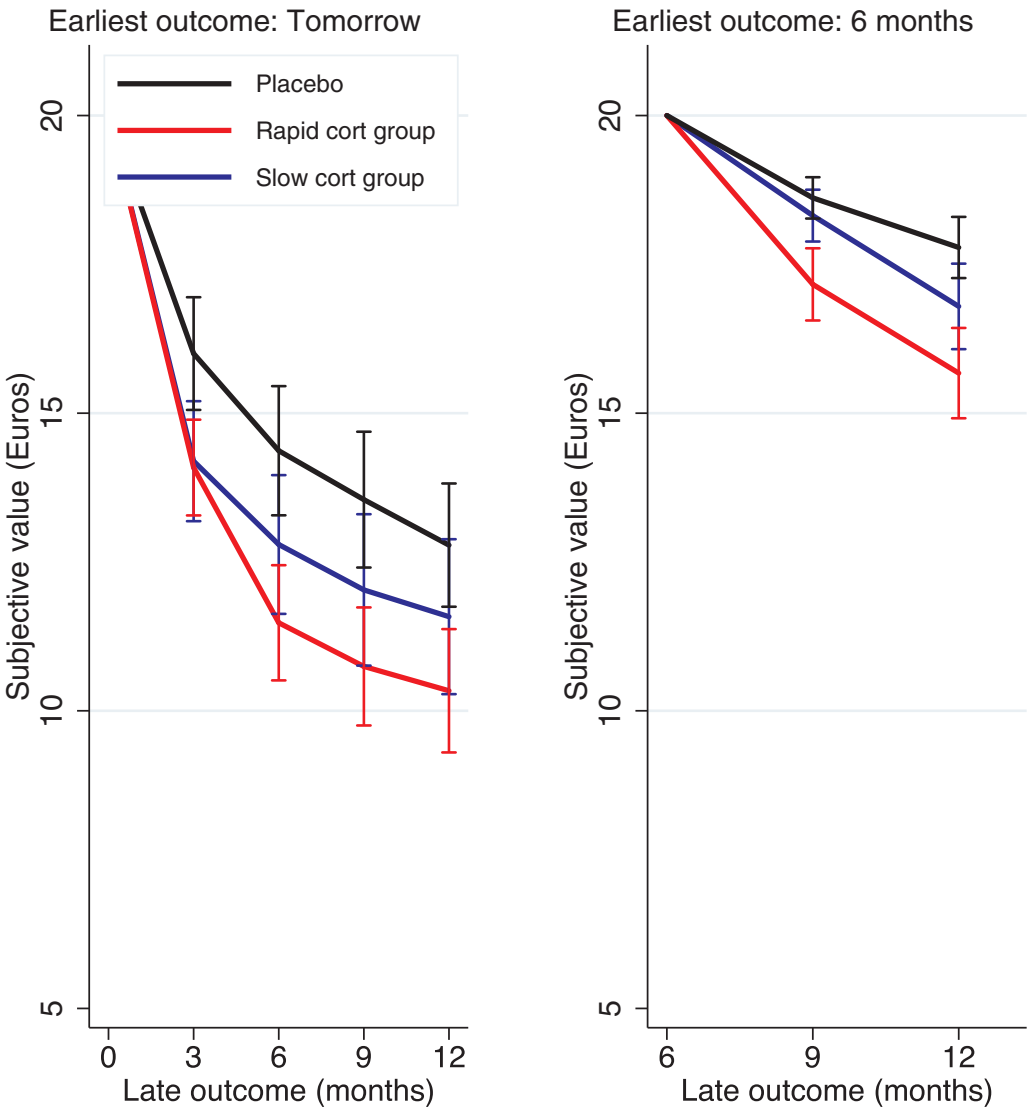

Fig. 2. Indifference points in intertemporal choice of placebo (black), rapid cort (red), and slow cort (blue) groups. Shown are mean indifference points ± 1 SEM. The subjective value of an indifference point can be interpreted as the amount at which a participant is indifferent between receiving that amount tomorrow vs. receiving €20 after the specified delay. Lower indifference points therefore indicate greater discounting. The left panel shows discounting between tomorrow and 3, 6, 9, and 12 months from tomorrow; the right panel shows discounting between 6 months and 9 and 12 months.

worn off 3 h after hydrocortisone administration, or that a putative delayed effect counteracted the acute effect.

The lower panel of Table 1 shows that the point estimates changed very little when controlling for negative affect and state anxiety. This was also true when controlling for all baseline variables for which there are baseline differences between any two groups; see Supplementary Table S8. Furthermore, the control variables did not explain variation in the indifference points, either individually (i.e. their coefficients were non-significant) or jointly (Wald tests of joint significance of both variables, presented in the lower part of the table, were non-significant). Supplementary Table S5 shows that the results did not change when we used ordinary least squares (OLS) instead of seemingly unrelated regression (SUR) to estimate all equations.

3.2.2. Distinguishing impatience and present bias (non-parametrically)

Results for the non-parametric measure of present bias are shown in Table 2. Higher values indicate more present bias. The constant, which reports the average present bias for the control group, was highly significantly different from zero for both delay lengths, indicating strong present bias for the control group. The point estimates on present bias were positive for both the rapid cort and the slow cort groups, but neither was statistically significant. Thus, participants in our sample displayed present bias, but hydrocortisone administration did not affect it.

3.2.3. Parametric estimation of impatience and present bias

Finally, we jointly analyzed treatment effects on impatience and present bias by estimating the quasi-hyperbolic model (Laibson, 1997). The results are presented in Table 3. Column (1) shows that the annual discount factor for the placebo group was $\delta = 0.77$, which translates into a annual discount rate of $r_{Placebo} = 0.31$ (reported in the lower panel of the table). This discount rate is high compared to outside option interest rates; however, it is not unreasonable compared to discount rates usually elicited in laboratory settings (Frederick et al., 2002). Table 3 column (1) shows that the estimated average present bias was $\beta_{Placebo} = 0.84$, which is reasonable compared to previous estimates (e.g. Andersen et al., 2008). The lower panel of the table shows that both the annual discount factor and present bias for the placebo group differed significantly from one.

Importantly, the annual discount factor was 15 percentage points lower for the rapid cort group than the placebo group. This difference translates into a difference in annual discount rates of 33 percentage points (31% versus 64%). Thus, the rapid cort group required an annual interest rate more than twice as high as the placebo group to be indifferent between receiving money tomorrow and one year from tomorrow. This difference stayed almost constant when we controlled for all variables for which there were baseline differences, and a similar difference was not found between the slow cort group and the placebo group. Furthermore, treatment did not affect present bias, consistent

Table 1
Indifference points (non-structural estimation).

	Indifference points for (*t*, *T*) without controls						
	(0,3)	(0,6)	(0,9)	(0,12)	(6,9)	(6,12)	Average
Rapid cort group	−1.91	−2.89*	−2.80	−2.45	−1.45*	−2.11*	−2.27*
	(1.23)	(1.44)	(1.49)	(1.45)	(0.69)	(0.90)	(1.05)
Slow cort group	−1.81	−1.58	−1.52	−1.20	−0.30	−0.99	−1.23
	(1.36)	(1.57)	(1.69)	(1.64)	(0.55)	(0.87)	(1.13)
Constant	16.00**	14.37**	13.55**	12.78**	18.62**	17.78**	15.52**
	(0.93)	(1.07)	(1.13)	(1.02)	(0.34)	(0.51)	(0.74)
N	78	78	78	78	78	78	78
Rapid vs. slow χ^2	0.01	0.77	0.65	0.58	2.46	1.18	0.83
Rapid vs. slow *p*	0.93	0.38	0.42	0.45	0.12	0.28	0.28
Joint sig. treatments χ^2	2.79	4.06	3.53	2.86	4.45	5.60	4.68
Joint sig. treatments *p*	0.25	0.13	0.17	0.24	0.11	0.06	0.10
	Indifference points for (*t*, *T*) with controls						
	(0,3)	(0,6)	(0,9)	(0,12)	(6,9)	(6,12)	Average
Rapid cort group	−2.27	−3.06*	−3.27*	−3.10*	−1.47	−2.10*	−2.54*
	(1.28)	(1.55)	(1.62)	(1.57)	(0.78)	(0.98)	(1.15)
Slow cort group	−2.03	−1.81	−1.84	−1.63	−0.35	−0.97	−1.44
	(1.31)	(1.56)	(1.68)	(1.60)	(0.58)	(0.90)	(1.12)
Negative affect	−0.65	−0.54	−0.90	−1.22	−0.10	0.04	−0.56
	(0.60)	(0.73)	(0.75)	(0.76)	(0.35)	(0.43)	(0.52)
State anxiety	0.30	0.58	0.47	0.61	0.16	−0.03	0.35
	(0.76)	(0.82)	(0.84)	(0.80)	(0.21)	(0.40)	(0.56)
Constant	16.19**	14.50**	13.80**	13.13**	18.64**	17.77**	15.67**
	(0.96)	(1.12)	(1.19)	(1.07)	(0.37)	(0.54)	(0.78)
N	78	78	78	78	78	78	78
Rapid vs. slow χ^2	0.04	0.67	0.79	0.79	2.20	1.10	0.91
Rapid vs. slow *p*	0.85	0.41	0.37	0.37	0.14	0.29	0.34
Joint sig. treatments χ^2	3.64	3.91	4.09	3.88	3.60	4.70	4.98
Joint sig. treatments *p*	0.16	0.14	0.13	0.14	0.17	0.10	0.08
Joint sig. controls χ^2	1.23	0.68	1.44	2.58	0.56	0.01	1.16
Joint sig. controls *p*	0.54	0.71	0.49	0.27	0.76	1.00	0.56

Notes: Regression analysis of differences in indifference points between the three treatment groups. *t* indicates the *early* date, with 0 being "tomorrow" and 6 being six months from "tomorrow;" *T* indicates the delay of the *later* date in months from "tomorrow". Indifference points indicate the point at which an individual is indifferent between the observed amount at the earlier date and €20 at the later date. Negative affect is standardized and measured by PANAS; State anxiety is standardized and measured by the State-Trait Anxiety Index. The lower panel of the table reports the result of Wald tests of equality between the rapid cort and slow cort groups, tests of joint significance for the rapid cort and slow cort groups, and tests of joint significance of the control variables.

* $p < 0.05$.

** $p < 0.01$.

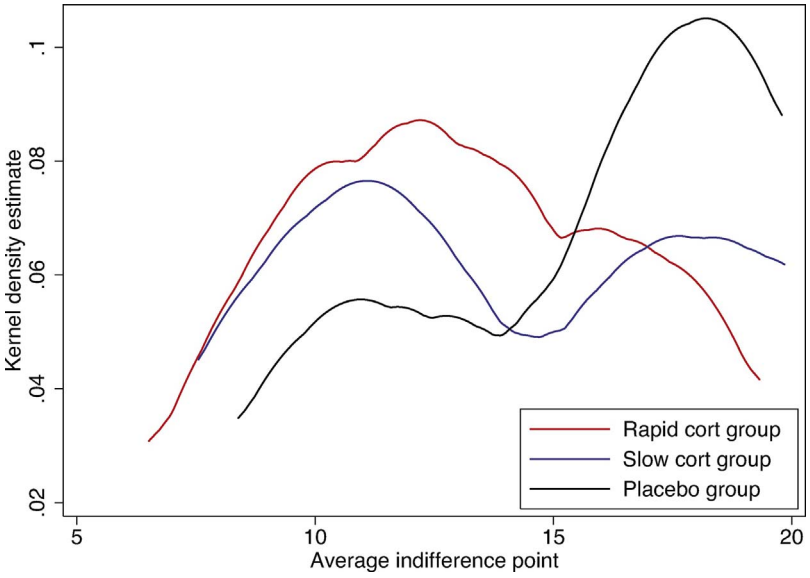

Fig. 3. Kernel density estimation of indifference points in intertemporal choice of placebo (black), short delay (red), and long delay (blue) groups, using an Epanechnikov kernel with optimal half-width (i.e. the half-width that would minimize the mean squared error if the data were Gaussian).

Table 2
Present bias (non-structural estimation).

	Present bias without controls		Present bias with controls	
	3 months	6 months	3 months	6 months
Rapid cort group	0.46	0.78	0.80	0.96
	(1.15)	(1.31)	(1.23)	(1.38)
Slow cort group	1.51	0.59	1.68	0.84
	(1.34)	(1.46)	(1.30)	(1.45)
Negative affect			0.55	0.58
			(0.64)	(0.67)
State anxiety			−0.15	−0.61
			(0.76)	(0.86)
Constant	2.62**	3.41**	2.45*	3.28**
	(0.92)	(1.03)	(0.94)	(1.07)
N	78	78	78	78
Rapid vs. slow *F*	0.78	0.02	0.57	0.01
Rapid vs. slow *p*	0.38	0.88	0.45	0.93
Joint sig. treatments *F*	0.67	0.18	0.85	0.27
Joint sig. treatments *p*	0.51	0.84	0.43	0.77
Joint sig. controls *F*			0.42	0.40
Joint sig. controls *p*			0.66	0.67

Notes: OLS regression estimates of differences in present bias between the three treatment groups. Present bias is calculated as the difference in indifference points between two blocks in which the delay, $T - t$, is identical, but the sooner time point, t, is different. "3 months" is the difference in indifference points between block $(t, T) = (0, 3)$ and block $(t, T) = (6, 9)$; "6 months" is the difference in indifference points between block $(t, T) = (0, 6)$ and block $(t, T) = (6, 12)$. Negative affect is standardized and measured by PANAS; State anxiety is standardized and measured by the State-Trait Anxiety Index. The lower panel of the table reports the result of Wald tests of equality between the rapid cort and slow cort groups, tests of joint significance for the rapid cort and slow cort groups, and tests of joint significance of the control variables.

* $p < 0.05$.

** $p < 0.01$.

with the results from the non-parametric analysis presented above. Thus, hydrocortisone administration increased discounting, but not present bias, immediately after administration, but not 3 h later.

3.3. Robustness

3.3.1. Payment belief

As mentioned above, all participants were paid the entire amount chosen in the intertemporal choice the day after the experiment due to a restriction imposed by the human subjects committee. Participants were not informed about this fact before the study, and it is therefore unlikely to have affected results. Given that the later amount offered was always strictly higher than the early amount, payoff-maximizing participants who believed that they would be receiving the entire amount "tomorrow" should exhibit no discounting in the intertemporal choice task. There were only 16 (21%) "fully patient" (indifference point ≥ 19) participants in our sample (3 in the rapid cort group, 6 in the slow cort group, and 7 in the placebo group). Under the assumption of payoff-maximization, this number represents an upper bound on the proportion of participants who believed that the entire amount would be paid out the day after the experiment. The behavior of most respondents was therefore not consistent with a belief that the intertemporal choice task was not perfectly incentivized.

3.3.2. Experimenter demand effects

A basic concern in experimental stress manipulations such as the Cold Pressor Task or Trier Social Stress Test is that participants are not blind to their experimental condition. This fact may result in "experimenter demand effects", i.e. participants making inferences about the effect the experimenter wishes to observe, and acting accordingly. One advantage of manipulating cortisol levels pharmacologically in a double-blind fashion as in this study is that participants are less likely to be aware of their treatment assignment, and therefore less likely to show demand effects. To assess whether participants were in fact aware of their treatment condition, we asked them on the day after the intertemporal choice task to guess which pill they received at both timepoints ("Hydrocortisone," "Placebo," or "Don't know"). Participants knew that at most one pill would be hydrocortisone.

One participant guessed "Don't know" for pill 1 and "Hydrocortisone" for pill 2, which indicates a lack of understanding. To be conservative, we treat this participant as if he guessed to be in the rapid cort treatment. One participant guessed "Placebo" for first pill and "Don't know" for the second pill, and five participants guessed "Don't know" for the first pill and "Placebo" for the second pill. For a conservative measure of how may participants correctly guessed their treatment, these six participants are interpreted as having correctly guessed their treatment to the extent that this is consistent with their responses (e.g. a participant in the slow cort group who guessed "Don't

Table 3
Joint estimation of impatience and present bias (structural estimation).

	Relative indifference points		
	(1)	(2)	(3)
Annual discount factor: placebo	0.77**	0.78**	0.76**
	(0.04)	(0.04)	(0.04)
Δ Annual discount factor: rapid cort group	−0.15*	−0.17*	−0.15*
	(0.07)	(0.07)	(0.08)
Δ Annual discount factor: slow cort group	−0.04	−0.06	−0.08
	(0.07)	(0.07)	(0.07)
Present bias: placebo	0.84**	0.84**	0.83**
	(0.05)	(0.05)	(0.05)
Δ Present bias: rapid cort group	−0.05	−0.06	−0.05
	(0.07)	(0.07)	(0.07)
Δ Present bias: slow cort group	−0.06	−0.07	−0.07
	(0.07)	(0.07)	(0.07)
Negative affect		−0.03	−0.04
		(0.03)	(0.03)
State anxiety		0.02	0.03
		(0.03)	(0.03)
Positive affect			0.03
			(0.02)
Anxiety sensitivity			0.01
			(0.02)
Debt			0.03
			(0.04)
Discount factor: Rapid vs. Slow, F	1.98	1.95	0.63
Discount factor: Rapid vs. Slow, p	(0.16)	(0.17)	(0.43)
Present bias: Rapid vs. Slow, F	0.03	0.02	0.08
Present bias: Rapid vs. Slow, p	(0.87)	(0.88)	(0.78)
Joint sig. (treatments), *F*	1.50	1.49	1.27
Joint sig. (treatments), *p*	(0.21)	(0.21)	(0.29)
Annual discount factor (placebo) = 1, *F*	42.49	30.31	29.56
Annual discount factor (placebo) = 1, *p*	(0.00)	(0.00)	(0.00)
Present bias (placebo) = 1, *F*	10.03	9.81	9.91
Present bias (placebo) = 1, *p*	(0.00)	(0.00)	(0.00)
Annual discount rate: Placebo	0.31	0.29	0.31
Annual discount rate: Rapid cort	0.63	0.65	0.64
Annual discount rate: Slow cort	0.38	0.39	0.47

Notes: Non-linear regression estimates of differences in discounting parameters, annual discount factor (δ) and present bias (β), between the three treatment groups. Δ indicates the difference in a parameter between the placebo group and a treatment group. Positive and negative affect are measured by PANAS, state anxiety is measured by the State-Trait Anxiety Index, anxiety sensitivity is measured by the Anxiety Sensitivity Index, and debt is a dummy for whether the participants had debt. The lower panel of the table reports the result of Wald tests of equality between the rapid cort and slow cort groups separately for impatience and present bias, tests of joint significance of the rapid cort and slow cort groups, and tests of the annual discount factor and the present bias coefficient for the placebo group against unity (corresponding to no discounting and no present bias). The lower panel of the table also reports the annual discount rate for each treatment group, calculated as $(1 - \delta)/\delta$.

* $p < 0.05$.

** $p < 0.01$.

know" for the first pill and "Placebo" for the second pill is interpreted as having correctly guessed being in the slow cort group).

Overall, 33.3% of the participants ($n = 26$) correctly guessed their treatment. Given that there were three treatments, this proportion corresponds to chance performance. Thus, participants were unable to guess which pill they received at which time point. Furthermore, almost half of the participants, 44.9% ($n = 35$), guessed "Don't know" for both pills, further confirming that it was not evident for the participants in which treatment group they were. Finally, when restricting the regressions presented in Table 1 to participants who did *not* correctly guess their treatment, the results changed very little (Supplementary Table S7) and were still significant.

3.3.3. Consistency

Another potential challenge is that participants in the rapid cort group may simply have made more errors (Franco-Watkins et al., 2006). We therefore asked whether treatment assignment affected the consistency of choices, i.e. the match between the first and last question in a block of 7 questions. 80.8% of participants ($n = 63$) were consistent in all of the blocks, and the remaining 19.2% ($n = 15$) were consistent in all but one block. Of these 15 participants, 6 were in the rapid cort group, 7 in the slow cort group, and 2 in the placebo group. These differences are too small to have driven our results, which hold across blocks.

3.3.4. Curvature of the utility function

In Section 3 we estimated the parameters of the quasi-hyperbolic model, i.e. discount rate and present bias, under the assumption of a linear utility function, i.e. $u(x) = x$. Because the curvature of the utility function affects the estimated discount rate, a potential effect of hydrocortisone on the curvature of the utility function could manifest itself as a change in present bias or impatience. To assess the robustness of our results to different curvatures of the utility function, we conducted a simulation exercise in which we assumed linear utility for the placebo group, and a power utility function for the rapid and slow cort groups, i.e. $u(x) = x^{\alpha}$. We repeated the main analysis for $\alpha \in \{1.0;0.95;0.90;0.85;0.80\}$. Results are presented in Supplementary Table S10. The difference in discount factors between the rapid cort and the placebo group was still significant on a 5% significance level for $\alpha = 0.95$, was marginally significant for $\alpha \in \{0.90;0.85\}$, and insignificant for $\alpha = 0.80$. To assess whether these values of α cover a reasonable range, we can ask what they imply for risk aversion. To get a sense of how risk-averse a decision-maker with $\alpha = 0.80$ is, note that he would be indifferent between a certain payment of $10,000 and the toss of a fair coin for $100,000 or nothing. Thus, our results are robust to significant changes in utility function curvature. In addition, Kandasamy et al. (2014) found no significant increase in the curvature of the utility function for participants who, as in our study, had been given an acute dose of hydrocortisone. It is therefore unlikely that our results can be explained by changes in utility function curvature.

3.4. Mechanisms

We obtained several measures of psychological state both at baseline and immediately after the participants had completed the intertemporal choice task, and can therefore ask whether the effect of hydrocortisone on intertemporal choice was mediated by any of these variables. Table S11 reports regression results for the effect of treatment on positive affect, negative affect, state anxiety, and impulsiveness, all measured with questionnaires. For positive affect, negative affect, and state anxiety, we subtract the baseline measure from the endline measure; impulsiveness was only measured at endline.

In the rapid cort group relative to placebo, we found a marginally significant decrease in positive affect ($p < 0.10$), and significant increases in negative affect and state anxiety. We detected no effects of the rapid cort treatment on impulsiveness, and no effects of the slow cort treatment on any outcome. Thus, of the psychological variables measured, positive affect, negative affect, and state anxiety present the most likely potential mechanisms for the effects of hydrocortisone administration on discounting. However, note that the other variables might also show effects with more statistical power.

We next asked whether these variables were predictive of discounting behavior. Table S12 shows the results for OLS regressions of the average indifference points for each participant on the affective variables. Interestingly, the affective variables were uncorrelated with discounting behavior. As expected given the findings in Lerner et al. (2013), the point estimate of the change in positive affect was positive, indicating slightly lower discounting for participants who experienced higher increases in positive affect, while the point estimates of the change in negative affect and state anxiety were negative, indicating slightly higher discounting for participants who experienced higher increases in negative affect. However, none of the affective variables significantly predicted behavior in the discounting task and the predictive power of the model including all four psychological variables was low (the R^2 is 7%, cf. Table S12, column 5). One possible explanation for these results is that the study was not adequately powered to detect these effects. Future research should further test the psychological mechanisms of the effects of hydrocortisone on discounting behavior, e.g. through a change in time perception (Takahashi, 2005; Han and Takahashi, 2012).

4. Discussion

In this study we demonstrate an effect of hydrocortisone administration on intertemporal choice. Specifically, we show that hydrocortisone increases participants' impatience, as measured by their willingness to give up a larger later reward in order to gain a smaller sooner reward immediately after hydrocortisone administration. In contrast, hydrocortisone does not affect present bias. In addition, hydrocortisone administration does not affect impatience or present bias when participants are tested several hours later, suggesting either that the effect of acute stress decreases over 3 h, or that a delayed effect of hydrocortisone counteracts the rapid effect.

This study contributes to the emerging literature on the effect of stress on economic choice in general, and intertemporal choice in particular. Previous correlational studies support the plausibility of the hypothesis that stress may affect intertemporal choice (Takahashi, 2004). However, experimental findings are conflicting. As described above, physical stress, experimentally induced by thermal stimulation or by the Cold Pressor Task, has been shown to increase discounting in some studies (Koppel et al., 2017; Delaney et al., 2014), but not others (Haushofer et al., 2015), while social stress induced by the Trier Social Stress Test appears not to affect discounting (Haushofer et al., 2013, 2015). Our finding that increased levels of the stress hormone cortisol lead to increased discounting suggests that these discrepant existing findings might be reconciled by differential effects of the stress induction methods on cortisol levels; this is a topic for future study. Relatedly, negative affect has been shown to increase discounting (Lerner et al., 2013; McLeish and Oxoby, 2007), while positive affect decreases discounting (Pyone and Isen, 2011; Ifcher and Zarghamee, 2011); to the extent that negative affect correlates with stress and cortisol levels, these findings mirror our results.

The rapid effect of hydrocortisone on impatience is in line with current views that shortly after stress, individuals turn to simple behavioral strategies. For instance, humans exposed to a psychosocial stressor switch from complex, goal-directed learning strategies to simpler, reflex-like strategies (Schwabe et al., 2007, 2010). This shift is accompanied by the activation of a salience network (Hermans et al., 2011), and mediated by the joint actions of cortisol and another prominent neurotransmitter involved in the stress response, norepinephrine (Schwabe et al., 2010). This increase in habitual responding is broadly consistent with our finding of increased impatient

responding under the influence of hydrocortisone in light of the fact that impatient responding in intertemporal choice tasks is commonly understood to be partly due to impulsive responses. However, we obtain this effect without administering compounds that are known to stimulate the noradrenergic system, suggesting that some behaviors may be affected by cortisol alone, at least in the short run.

More broadly, this study extends the literature that estimates the effect of stress on economic behaviors. Existing evidence suggests that stress increases risk aversion in the gains domain (Delaney et al., 2014; Porcelli and Delgado, 2009), and that this effect is mediated by cortisol levels (Kandasamy et al., 2014). Acute stress has also been shown to increase pro-social behavior in economic exchange games (von Dawans et al., 2012). Together, these findings begin to map the landscape of the effect of stress on economic behavior, and the present study contributes by demonstrating that pharmacologically elevated cortisol levels increase temporal discounting.

One limitation of our study is that the soonest delay available was tomorrow, which complicates studying present bias. Future studies will need to explore different time scales, varying both the delay between hydrocortisone administration and the task, as well as the rewards delays within the intertemporal choice task, to fully understand the complexity of the effects of stress and stress hormones on intertemporal choice. Moreover, the intertemporal choice task in the present study was not fully incentivized, in the sense that participants were paid the amount of a randomly chosen trial from the intertemporal choice task, but this payment was made on the day following the experiment, raising the possibility that the behavioral effect may have been underestimated. Furthermore, the elicitation method (titration) was not fully incentive compatible. Future studies should test the robustness of our results in a fully incentivized and incentive compatible way.

Conflicts of interest

The authors declare no conflicts of interest.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.psyneuen.2017.10.002.

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