



## Dissociable roles of glucocorticoid and noradrenergic activation on social discounting



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### ABSTRACT

People often exhibit prosocial tendencies towards close kin and friends, but generosity decreases as a function of increasing social distance between donor and recipient, a phenomenon called social discounting. Evidence suggests that acute stress affects prosocial behaviour in general and social discounting in particular. We tested the causal role of the important stress neuromodulators cortisol (CORT) and noradrenaline (NA) in this effect by considering two competing hypotheses. On the one hand, it is possible that CORT and NA act in concert to increase generosity towards socially close others by reducing the aversiveness of the cost component in costly altruism and enhancing the emotional salience of vicarious reward. Alternatively, it is equally plausible that CORT and NA exert dissociable, opposing effects on prosocial behaviour based on prior findings implicating CORT in social affiliation, and NA in aggressive and antagonistic tendencies. We pharmacologically manipulated CORT and NA levels in a sample of men ( $N = 150$ ) and found that isolated hydrocortisone administration promoted prosocial tendencies towards close others, reflected in an altered social discount function, but this effect was offset by concurrent noradrenergic activation brought about by simultaneous yohimbine administration. These results provide inceptive evidence for causal, opposing roles of these two important stress neuromodulators on prosocial behaviour, and give rise to the possibility that, depending on the neuroendocrine response profile, stress neuromodulator action can foster both tend-and-befriend and fight-or-flight tendencies at the same time.

### 1. Introduction

Although almost all people engage in prosocial behaviour at times, generosity tends to decrease with increasing social distance between donor and recipient. After all, while many of us do not hesitate to donate money to our close family members in need, very few of us would be willing to give the same amount to disadvantaged strangers. This decline in generosity as a function of increasing social distance is called social discounting, a phenomenon which has triggered significant research interest in recent years (Jones and Rachlin, 2006; Kalenscher, 2017; Margittai et al., 2015; Strang et al., 2017; Strombach et al., 2015, 2014; Vekaria et al., 2017).

Due to the high prevalence of acute stress in daily life, research focusing on how it impacts social decision making has increased manifold in recent years (Porcelli and Delgado, 2017; Starcke and Brand, 2012). Acute stress is associated with the activation of the hypothalamic-pituitary-adrenal axis (HPA axis) system as well as autonomic arousal (Selye, 1950), and increases in two main

neuromodulators, cortisol (CORT) and noradrenaline (NA) respectively. These substances impact brain function in a symphonic, time-dependent fashion, with imminent elevations of NA, shortly followed by non-genomic CORT effects after stress onset, and subsequent genomic CORT response in the aftermath of stress (Hermans et al., 2014; Joëls and Baram, 2009).

In stark contrast to the canonical view that acute stress primarily leads to fight-or-flight, it has now been reliably shown that it can also foster prosocial behaviour in some situations, in both men and women (Buchanan and Preston, 2014; Margittai et al., 2015; Taylor et al., 2000; Tomova et al., 2017; Von Dawans et al., 2012).

In recent work (Margittai et al., 2015), we specifically focused on whether social closeness is a determining factor in acute stress effects on prosocial behaviour, and thus investigated how it altered social discounting. Results showed that exposure to psychosocial stress (Trier Social Stress Test for Groups, Von Dawans et al., 2011) increased generosity, but only towards individuals who were socially close to the decision maker. These findings were interpreted in the context of the

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tend-and-befriend hypothesis (Taylor, 2006), a coping mechanism that helps to counteract the negative effects of stress by investing into social networks providing help and comfort. As socially close others are more likely to offer protection in time of need, it is reasonable to focus affiliative efforts, and thus become more prosocial only towards them. Extending these findings Berger et al. (2016) demonstrated, that CORT responses to psychosocial stress were positively correlated with the tendency to affiliate amongst men, lending support to the idea that CORT plays a key role in social affiliative coping and thus in prosocial behaviour after stress. Furthermore, CORT has already been implicated as a positive predictor of empathy, a concept indisputably related to prosocial behaviour (Zilioli et al., 2015). The role of NA in prosocial behaviour, and its putative interaction with CORT, is less clear. CORT and NA acting in concert reduce loss aversion (Margittai et al., 2018), promote attention to salient stimuli (Hermans et al., 2011), and sharpen vigilance contrasts, and NA-related arousal caused by observing another person in distress has been found to be related to subsequent costly helping (Hein et al., 2011). This may suggest that generosity towards socially close others after stress might be boosted by the conjoint action of CORT and NA, by reducing the aversiveness of the costs in costly altruism, and at the same time enhancing the emotional salience of vicarious reward signals and feelings of warm glow. By contrast, NA has been widely associated with arousal and aggression both in animal and human studies (Nelson and Trainor, 2007), and it is known to reduce social play and affiliation in animals (Achterberg et al., 2016). Thus, it is equally plausible that CORT by itself promotes prosocial behaviour, particularly towards socially close others, while the concomitance of NA inhibits these prosocial tendencies.

Here, we set out to decide between these two competing hypotheses by investigating the causal effect of CORT and NA manipulation on social discounting. We pharmacologically manipulated CORT and NA levels by oral, exogenous administration of hydrocortisone or yohimbine (an alpha-2 adrenergic receptor antagonist) respectively. These substances were given separately or concomitantly in a placebo-controlled double-blind experimental design. We measured how elevations in CORT and NA level impact on social discounting using the same task that has been reported by (Margittai et al., 2015).

## 2. Materials and methods

### 2.1. Participants

One hundred and fifty male participants took part in the experiment. We opted to employ male participants only because there is evidence of gender differences in HPA-axis reactivity as well as effects of oral contraceptives and menstrual cycle phase on HPA-axis reactivity in female participants (Kirschbaum et al., 1999). Sample size was determined using G\*Power (Faul et al., 2007). Assuming a medium effect size (also see Margittai et al., 2015), the sample size necessary to achieve a power of 0.8 was  $n = 128$ . We eventually opted to collect data from 150 participants, thus exceeding the minimum sample size requirement, to have a contingency for potential exclusions or other problems. Hence, we are confident that our study was sufficiently powered to detect the required effects.

Before participation individuals completed a screening interview and those who reported regular use of medication, chronic physical or mental illness, heavy smoking, drinking or drug use or being students of Psychology or Economics were not invited to participate. 7 participants disclosed after the experiment that they either had illnesses or were taking medication, and they were consequently excluded from further analyses. All participants had fluent knowledge of German, gave their written, informed consent and received financial compensation for participation. The study was approved by the ethics committee of the University Hospital Düsseldorf and conformed to the regulations of the Declaration of Helsinki. Participants were instructed not to engage in sexual activities, take medication or alcohol for 24 h prior to

participation, not to smoke, or drink anything containing caffeine for 4 h prior to participation, and to refrain from physical exercise, eating and drinking anything other than water for 2 h before participation. These criteria were similar to what had been employed in other studies (e.g. Vinkers et al., 2013).

### 2.2. Trait measures

Prior to being invited to the laboratory, all participants completed a number of trait questionnaires online, designed to exclude potential confounds between the experimental groups:

We measured trait anxiety (State-Trait Anxiety Inventory – STAI, (Spielberger et al., 1983), impulsivity (Barratt Impulsiveness Scale – BIS-15, (Meule et al., 2011), reward and punishment sensitivity (BIS/BAS scale, Carver and White, 1994), social desirability (Social Desirability Scale – SDS-17, Ströber, 2001), empathy (Saarbrücker Persönlichkeitsfragebogen – SPF, Paulus, 2007), chronotype (reduced version of the Morningness-Eveningness Questionnaire – rMEQ, Randler, 2013) and general willingness to take risks. Additionally we recorded age, BMI, baseline salivary cortisol, baseline salivary alpha-amylase, baseline subjective feelings of stress (VAS) and mood (PANAS; Watson et al., 1988).

### 2.3. Pharmacological manipulation, physiological and subjective stress measures

Participants were randomly assigned to one of four experimental conditions: (A) placebo (PLAC,  $N = 36$ ), (B) placebo + yohimbine (YOH, 20 mg, Cheplapharm,  $N = 38$ ), (C) placebo + hydrocortisone (CORT, 20 mg, Jenapharm,  $N = 38$ ), (D) yohimbine + hydrocortisone (YOH+CORT, 20 mg each,  $N = 38$ ). The number of tablets taken was identical in the four conditions, thus participants were unable to guess which condition they were in on the basis of the number of pills. The dosage was chosen to be in line with previous studies (Margittai et al., 2018, 2016; Schwabe et al., 2012, 2010). To assess increases in cortisol levels and noradrenergic activation, saliva samples (using Salivette devices from Sarstedt, Germany) were collected at baseline and +30, +60 and +75 min after pill ingestion and subsequently frozen at  $-20\text{ }^{\circ}\text{C}$  until transport and analysis using the same method as reported by (Rohleder et al., 2004). 25 of the 1500 samples were compromised and thus could not be analysed. These values were excluded from analyses. All other samples were analysed for concentrations of salivary cortisol (CORT) and salivary alpha amylase (sAA), an indirect marker of noradrenergic activity. For each participant, two samples were taken approximately 10 min and 20 min before pill intake and their values averaged to determine individual baseline. In case one of the values was missing, we used the remaining value as the baseline. Subjective feelings of stress (using visual analogue scales – VAS) were taken at the same time as the saliva samples. Changes in positive and negative mood were assessed once at baseline and once 60 min after drug intake (shortly before the experimental tasks), using PANAS (Watson et al., 1988) scales. Change scores were calculated by subtracting baseline from the later measure.

### 2.4. Elicitation of social environment and experimental task

Our aim was to investigate how the decline in generosity across social distance is affected by CORT and NA. Thus, we asked participants prior to pill intake to describe their social environment using a similar method reported by Margittai et al. (2015) and Strombach et al. (2014, 2015). Individuals were asked to give the names of representatives for social distances (SD) 1, 2, 3, 5, 10 and 20, with SD 1 representing the person they feel closest to, with decreasing closeness as a function of increasing social distance. Although we also included distances 50 and 100 in the experiment, participants were not asked to provide a name for these, as they represent remote individuals or strangers whose

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