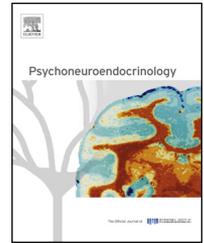




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The biological effects of acute psychosocial stress on delay discounting

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Received 21 November 2012; received in revised form 25 March 2013; accepted 29 April 2013

KEYWORDS

Delay discounting;
Inter-temporal choice;
C-reactive protein;
Cortisol;
HPA axis;
TSST;
Impulsivity

Summary Organisms prefer to receive rewards sooner rather than later because they excessively discount the subjective value of future rewards, a phenomenon called delay discounting. Recent studies have reported an association between cortisol—which is secreted by the hypothalamic–pituitary–adrenal (HPA) axis—and delay discounting. However, no study has examined whether acutely induced psychosocial stress modulates delay discounting. Thus, the present study examined the effect of acute psychosocial stress and its hormonal and inflammatory correlates on the rate of delay discounting. To accomplish this purpose, we assessed the participants' discounting rates using the questionnaire version with inter-temporal choice before and after an acute psychosocial stress task (the Trier Social Stress Test; TSST). The results demonstrated that TSST increased rates of delay discounting in only cortisol responders (not in non-responders), indicating the possible influence of the pathway from the HPA axis to the dopaminergic systems under acute stress. Furthermore, the findings of correlation analysis indicated a U-shaped relationship between baseline level of C-reactive protein and delay discounting rate, suggesting a complex relationship between inflammatory markers and delay discounting rate.

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1. Introduction

In the field of neuroeconomics, decision making among monetary rewards available after different delays is called intertemporal choice. Typically, organisms prefer to receive rewards sooner rather than later due to excessive discounting of the subjective value of future rewards, which is called delay discounting (Ainslie, 1975; Wittmann and Paulus, 2008). Delay discounting is related to impulsivity, which is a core attribute in several psychiatric illnesses. Previous studies reported relationships between delay discounting and attention deficit hyperactivity disorder (ADHD) in children (Winstanley et al., 2006), smoking (Mitchell, 1999), addiction (Hamilton and Potenza, 2012), depression (Takahashi et al., 2008a,b,c), and suicidal behaviour (Takahashi, 2011).

Recent studies have provided evidence indicating that neurochemical and hormonal levels are related to delay discounting. For example, Takahashi (2004) first found that low cortisol levels at rest were associated with large delay discounting rates and suggested involvement of the interaction between the glucocorticoid and dopaminergic pathways in the orbitofrontal, striatal, and amygdaloid circuits. Individual differences in cortisol levels predicted delay discounting 6 months later, indicating a stable relationship of this hormone with impulsivity (Takahashi et al., 2010). In addition, single nucleotide polymorphisms in FK506 binding protein 5 (FKBP5), which is a co-chaperone of the glucocorticoid receptor and modulates the hypothalamic–pituitary–adrenal (HPA) axis, are associated with impulsivity (Kawamura et al., 2013). The authors found that the minor allele (T) of rs1360780, which causes overexpression of FKBP5, was negatively associated with delay discounting and concluded that this negative association might be due to glucocorticoid receptor resistance and increased levels of plasma cortisol. Another steroid hormone, testosterone, has an inverted-U relationship with the delay discounting rates of gains in males (Takahashi et al., 2006). The authors suggested complex interactions between testosterone and dopaminergic activities.

Salivary alpha-amylase, which reflects sympathetic nervous activity, is also related to delay discounting. Takahashi et al. (2007) demonstrated that individuals with lower salivary alpha-amylase were more impulsive in intertemporal choice. In the experimental situation with higher ecological validity, the degree of discounting for delayed primary reward (i.e., water) was positively correlated with salivary alpha-amylase level (Takahashi et al., 2008a,b,c). More recently, Diller et al. (2011) examined the relationship between heart-rate (HR) reactivity to acute stress and delay discounting; they found that female participants with higher reactivity showed larger delay discounting, but this trend did not hold in males. Their results indicated that the reactivity of the autonomic nervous system might be related to impulsivity and the possible existence of gender differences in this relationship.

Recent studies have begun to examine how neurochemical and hormonal levels modulate delay discounting (for a review, Takahashi, 2009). These studies have suggested the pivotal roles of the dopaminergic systems in delay discounting. The activation of dopaminergic systems is related to impulsive intertemporal choice between immediate and delayed monetary reward (McClure et al., 2004) and the

subjective value of a delayed reward (Kable and Glimcher, 2007). Another possible mechanism is via serotonergic pathways. Mobini et al. (2000) reported that reduced serotonergic activity increased discounting rates in rodents. However, human studies have failed to show a significant increment in delay discounting rates (Crean et al., 2002; Schweighofer et al., 2006). Thus, the possible role of the serotonergic system is still controversial. More recent studies have indicated a positive association between regional white matter volume of the hippocampus and the delay discounting rate (Yu, 2012). The author suggested an important role of the hippocampus in delay discounting.

These neural pathways are modulated under acute psychosocial stress. Previous studies demonstrated that acute laboratory stressors increase cortisol secretion through activation of the HPA axis (Kirschbaum et al., 1993). Pruessner et al. (2004) found that the salivary cortisol response to acute psychosocial stress was significantly correlated with dopamine release in the ventral striatum, suggesting a facilitating effect of cortisol on dopaminergic neuronal activity. In addition, previous studies have reported that transient activation of HPA axis might play an important role in stress-induced alteration of the serotonergic pathways and the hippocampus (Chaouloff, 2000; Takahashi et al., 2002). These previous findings indicate the possibility that acute psychosocial stress might transiently change delay discounting, especially among people who show high cortisol reactivity. Recently, Lempert et al. (2012) reported that higher delay discounting was associated with a larger cortisol response to acute anticipatory stress. However, Lempert et al. (2012) employed a between-participants design and did not assess temporal change in delay discounting from the pre- to post-stress periods.

It is also known that not only acute psychosocial stress but also peripherally released inflammatory cytokines, the latter of which enhance or inhibit systemic inflammation and are secreted mainly by immune cells, can influence the HPA axis and serotonin and dopamine metabolism in the amygdala, hippocampus, and nucleus accumbens (Raison et al., 2006). Howren et al. (2009) reported that resting levels of peripheral C-reactive protein (CRP) are positively associated with depressive symptoms. CRP is a nonspecific acute-phase protein and is largely regulated by circulating levels of interleukin-6 and interleukin-1, which have roles in cytokine-induced changes in behaviour and neurochemical activity. This association holds even for the mild depressive symptoms of non-clinical individuals (Suarez et al., 2004). In addition, depressive patients show larger delay discounting rates than healthy controls, possibly due to an impaired serotonergic system (Takahashi et al., 2008a,b,c). Those studies lead us to hypothesize that there is a close relationship between CRP and delay discounting. However, no study has examined whether and how CRP level is related to delay discounting.

The purpose of the present study is to examine (1) the effects of acute psychosocial stress on delay discounting, using a within-participants design to assess individual delay discounting before and after exposure to an acute laboratory stressor and (2) the relationship between the CRP and the delay discounting rate. To accomplish this purpose, we engaged a widely used acute psychosocial stress paradigm and assessed individual discounting rates before and after

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