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Endogenous Cortisol Predicts Decreased Loss Aversion in Young Men

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Human and nonhuman animals respond asymmetrically to predicted punishments and rewards (Dayan & Seymour, 2008; Kahneman, 2011). In human decision making, for example, people pay more to avoid losses than to gain equivalent rewards. Because such *loss aversion* counterproductively diminishes an individual's expected payoffs, it has become one of the most studied choice biases. It is unclear whether biological markers of punishment or stress exposure—most notably the glucocorticoid stress hormone cortisol of the hypothalamic-pituitary-adrenal (HPA) axis—predict this particular form of behavioral punishment sensitivity. Acute glucocorticoid administration desensitizes subjects to threat and punishment, whereas chronic administration sensitizes them, increasing anxiety (Aerni et al., 2004; de Quervain & Margraf, 2008; Schelling et al., 2006; Soravia et al., 2006). This mirrors the mainstream view that acute stress responses are adaptive, whereas chronic exposure is detrimental (Chrousos, 2009).

There is evidence that HPA-axis traits specifically undermine decision making. HPA disturbances predict addictive behavior (Koob & Kreek, 2007; Marinelli & Piazza, 2002; Putman, Antypa, Crysovergi, & van der Does, 2010; Sinha, 2008), and the relation between long-term HPA activity and pathological gambling (Wohl, Matheson, Young, & Anisman, 2008) may reflect altered punishment sensitivity. In nonclinical populations, the threat of financial loss (i.e., imminent poverty) chronically elevates cortisol (Haushofer, de Laat, & Chemin, 2012). Yet it is unknown whether chronically elevated cortisol, in turn, alters exposure to new losses by altering decision making. Such a feedback cycle might be adaptive (negative feedback) or maladaptive (positive feedback), depending on whether it limits or exacerbates financial loss. In the present study, we sidestepped the issue of causation and simply assessed whether an individual's maladaptive loss aversion increased with chronic exposure to endogenous cortisol, which we assayed using hair samples.

Method

Fifty-seven healthy male undergraduates (18–30 years old) took part in the study. An additional 4 participants were excluded because 3 had insufficient hair and 1 outlier's hair cortisol was greater than 150 picograms per milligram. Sample size was sufficient for power of .99 to detect a correlation coefficient of .5 with a Type I error rate of .05 (no stopping rule). We used only male participants to eliminate potential nuisance variation attributable to gender differences in risk taking or HPA-axis function (e.g., Byrnes, Miller, & Schafer, 1999; Uhart, Chong, Oswald, Lin, & Wand, 2006).

Subjects made 20 binary choices from an existing set of 140 choices (Sokol-Hessner et al., 2009) designed to measure loss aversion (λ) and risk aversion (ρ). On each

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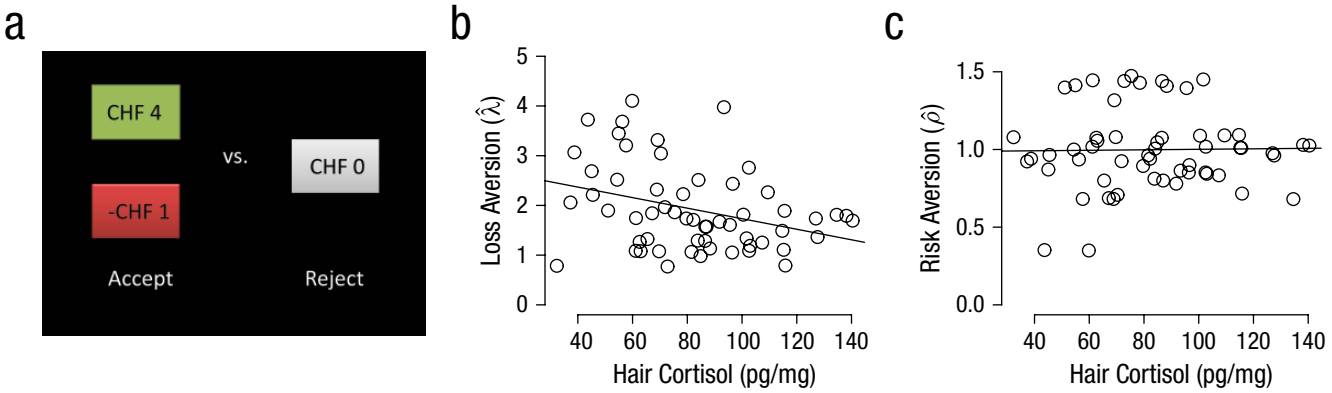

Fig. 1. Typical decision screen and results from the experiment. On each trial (a), subjects had to choose between a guaranteed payoff amount and a lottery in which they had an equal chance of winning or losing specific amounts. The scatter plots (with best-fitting regression lines) show (b) loss aversion ($\hat{\lambda}$) and (c) risk aversion ($\hat{\rho}$) as a function of hair-cortisol level.

trial, subjects had to choose between a guaranteed payoff y and a lottery in which they had an equal chance of winning x or losing z (Fig. 1a). Following Wang, Filiba, and Camerer (2010), we dynamically selected the most informative choice from the set for each trial, on the basis of subjects' previous choices. This alternative to staircase and bisection methods (Engelmann, Capra, Noussair, & Berns, 2009; Engelmann, Damaraju, Padmala, & Pessoa, 2009) optimally uses subjects' early choices to exclude redundant later choices. Subjects were given 20 Swiss francs (CHF) to start. One trial was randomly selected for actual payment. Payoffs ranged from −8.75 to 36 CHF (average = 6.29 CHF).

We used the utility function $u(w+) = w^{\rho}$ to determine positive payoffs ($w+$) and the utility function $u(w-) = -\lambda(w^{\rho})$ to determine negative payoffs ($w-$). Loss aversion was quantified by λ: $\lambda = 1$ was loss neutral, $\lambda > 1$ was loss averse, and $\lambda < 1$ was loss seeking. Following Sokol-Hessner et al. (2009) and Wang et al. (2010), we assumed that subjects would choose probabilistically. The following softmax function mapped preferences to the probability of accepting the lottery (μ describes how deterministic choices are):

$$p(\text{lottery}\,|\,\rho,\lambda,\mu) = \frac{1}{1+e^{-\mu\left(\frac{1}{2}u(x)+\frac{1}{2}u(z)-u(y)\right)}}$$

We correlated subjects' (expected marginal posterior) loss aversion, $\hat{\lambda} = E(\lambda|\,\text{lottery}_{1,\ldots,20})$, and risk aversion, $\hat{\rho} = E(\rho|\,\text{lottery}_{1,\ldots,20})$, with a measure of their total exposure to cortisol over the 2 months prior to the study (see Sauvé, Koren, Walsh, Tokmakejian, & Van Uum, 2007; Stalder & Kirschbaum, 2012; Van Uum et al., 2008; for more details on dynamic estimation, see the Supplemental Material available online).

Results

The mean loss- and risk-aversion parameters $\hat{\lambda}$ and $\hat{\rho}$ were 1.86, 95% confidence interval (CI) = [1.65, 2.09], and 1.00, 95% CI = [0.94, 1.08], respectively (which were not significantly different from 2 and 1, respectively; one-sample t test, $n = 56$); these results were similar to those of previous studies (Engelmann & Hein, 2013; Hsu, Lin, & McNamara, 2008; Sokol-Hessner et al., 2009). We observed a significant negative Pearson's correlation between loss aversion ($\hat{\lambda}$) and cortisol, $r(55) = -.33$, 95% CI = [−.54, −.076], $p = .012$ (Fig. 1b). No correlation was observed with risk aversion ($\hat{\rho}$), $r(55) = .016$, 95% CI = [−.25, .27], $p = .9$ (Fig. 1c). The loss aversion-cortisol relationship remained evident in a multiple linear regression controlling for smoking and shift work, two factors known to influence cortisol: $\beta = -10.2$, 95% CI = [−18.6, −2.0], $p = .016$.

Discussion

The hormonal response to stressors and punishment is governed by the evolutionarily prespecified HPA cascade, which is widely conserved in many animal species. Here, we observed that individuals with lower chronic cortisol displayed stronger loss aversion, a disadvantageous form of punishment sensitivity that diminishes individuals' long-term payoffs (Shiv, Loewenstein, & Bechara, 2005). Conversely, individuals with higher endogenous cortisol weighted losses and gains more equally (i.e., they were less loss averse). These results generate new questions. First, does long-term cortisol cause changes to loss aversion? Second, is decreased loss-gain asymmetry only due to lower predicted punishment from a unit monetary loss, or are there also higher predicted rewards from a unit monetary gain (punishment-reward sensitivity)? We briefly address existing knowledge relevant to these questions.

Cortisol crosses the blood-brain barrier, where it has neuro-modulatory actions (de Kloet, Joëls, & Holsboer, 2005; de Kloet, Oitzl, & Joëls, 1999; Fernandes, McKittrick, File, & McEwen, 1997; King & Liberzon, 2009) and influences behavior (Buchanan, Brechtel, Sollers, & Lovallo, 2001; Øverli, Kotzian, & Winberg, 2002). Neuro-modulatory actions include phasic and long-term changes to glucocorticoid receptors in limbic and prefrontal regions. Behavioral actions include altered punishment- and reward-related behavior (Sapolsky, Romero, & Munck, 2000), effects that depend on the time course of exposure (i.e., acute vs. chronic). Putman et al. (2010) demonstrated that acute, exogenous glucocorticoids decrease punishment sensitivity on a gambling task in humans: 40 mg of oral hydrocortisone increased risk seeking when subjects were faced with probable losses. Acute beta-adrenergic antagonists, which increase human gambling in the face of large probable losses (Rogers, Lancaster, Wakeley, & Bhagwagar, 2004) also acutely increase cortisol levels (Kizildere, Gluck, Zietz, Scholmerich, & Straub, 2003).

There are interactions between glucocorticoids and the central 5-hydroxytryptamine (5-HT) system (Gorzalka & Hanson, 1998), itself implicated in punishment processing. For example, long-term endogenous exposure to glucocorticoids predicts increased 5-HT_{2A} receptor binding in the parietal cortex (Fernandes et al., 1997). Although these alterations may reflect an adaptive stress response, other studies demonstrate that increased 5-HT_{2A} receptor activity is anxiogenic and amplifies behavioral response to stressors (Weisstaub et al., 2006). Our results encourage the speculation that, within the healthy population, long-term exposure to glucocorticoids may indeed be adaptive, reducing oversensitivity to potential losses. Further work should examine whether this hormone-behavior relationship reflects some prespecified coordination between psychological and biological responses to punishment.

Author Contributions

J. R. Chumbley designed the experiment, analyzed the data, and wrote the manuscript. I. Krajbich and J. B. Engelmann interpreted the data. E. Russell, S. Van Uum, and G. Koren analyzed the hair. E. Fehr supervised the project.

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Declaration of Conflicting Interests

The authors declared that they had no conflicts of interest with respect to their authorship or the publication of this article.

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Supplemental Material

Additional supporting information can be found at http://pss.sagepub.com/content/by/supplemental-data

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