



# Testosterone and cortisol jointly modulate risk-taking



Pranjal H. Mehta<sup>a,\*</sup>, Keith M. Welker<sup>b</sup>, Samuele Zilioli<sup>c</sup>, Justin M. Carré<sup>d</sup>

<sup>a</sup> University of Oregon, 1227 University of Oregon, Eugene, OR 97043, USA

<sup>b</sup> University of Colorado Boulder, Department of Psychology, 325 UCB, Boulder, CO 80309, USA

<sup>c</sup> Wayne State University, 42 W Warren Avenue, Detroit, MI 48202, USA

<sup>d</sup> Nipissing University, 100 College Drive, North Bay, ON P1B8L7, Canada

Received 26 November 2014; received in revised form 10 February 2015; accepted 27 February 2015

## KEYWORDS

Testosterone;  
Cortisol;  
Risk-taking;  
Impulsivity;  
Traits

**Summary** Recent theories propose that testosterone should be positively related to risk-taking, but empirical support is mixed. Building on the *dual-hormone hypothesis*, the present research tested whether testosterone's role in risk-taking depends on cortisol. Study 1 ( $N = 115$ ) tested this hypothesis in a mixed-sex sample with self and informant reports of risk-taking. Study 2 ( $N = 165$ ) tested this hypothesis in a male-only sample with the Balloon Analog Risk Task, a behavioral measure of risk-taking. Across both studies, there was a positive association between basal testosterone and risk-taking among individuals low in basal cortisol but not individuals high in basal cortisol. This pattern emerged in both males and females and across multiple measures of risk-taking (self reports, informant reports, behavior). These studies provide novel empirical support for the claim that testosterone and cortisol jointly regulate risk-taking. Discussion focuses on putative mechanisms as well as implications for real-world risk-taking behaviors.

© 2015 Elsevier Ltd. All rights reserved.

Risk-taking behaviors—behaviors that can harm the self or others (Steinberg, 2008)—include sexual risk-taking (e.g., unprotected sex, Caruthers et al., 2014), dangerous driving (e.g., Simons-Morton et al., 2011), financial risk-taking (e.g., Noussair et al., 2014), and substance abuse (Castellanos-Ryan et al., 2013). Risk-taking propensity may have evolved because of its adaptive benefits in the context of reproductive competition (Daly and Wilson, 1997; Ellis

et al., 2012), but hyper-risky behaviors in modern society can create numerous individual costs and societal burdens, such as the spread of infections, accidents resulting in injury or death, and instability in financial markets.

Recently several scholars have proposed that higher testosterone concentrations are related to increased risk-taking (e.g., Apicella et al., 2008; Steinberg, 2008), but findings are inconsistent. Although some studies have indeed shown positive associations between naturally occurring testosterone or exogenous administered testosterone and risk-taking (Van Honk et al., 2004; White et al., 2006; Apicella et al., 2008; Coates and Herbert, 2008; Vermeersch

\* Corresponding author. Tel.: +1 541 346 0475.

E-mail address: [mehta@uoregon.edu](mailto:mehta@uoregon.edu) (P.H. Mehta).

et al., 2008; Sapienza et al., 2009; Campbell et al., 2010; Goudriaan et al., 2010; Ronay and von Hippel, 2010; Stanton et al., 2011a; Määttä et al., 2013; Peper et al., 2013; van der Loos et al., 2013; Apicella et al., 2014; Evans and Hampson, 2014), other studies have shown null or even negative associations (for null effects, see Rosenblitt et al., 2001; Zethraeus et al., 2009; Boksem et al., 2013; Ortner et al., 2013; van der Loos et al., 2013; Derntl et al., 2014; for negative associations, see van Anders et al., 2012; see also Stanton et al., 2011b for a curvilinear association).

One candidate explanation for these inconsistencies is that testosterone's role in risk-taking may depend on cortisol, the hormonal end product of the hypothalamic-pituitary-adrenal (HPA) axis. High cortisol is associated with psychological stress and behavioral inhibition (Blair et al., 2004; Dickerson and Kemeny, 2004; Roelofs et al., 2009; Tops and Boksem, 2011; Pfattheicher and Keller, 2014), whereas low cortisol is associated with psychological relaxation and approach-oriented behaviors (Terburg et al., 2009; Ventura et al., 2012). According to the *dual-hormone hypothesis*, testosterone and cortisol should jointly regulate status such that testosterone should be positively related to status-relevant behaviors such as dominance only when cortisol concentrations are low but not when cortisol concentrations are high (Dabbs et al., 1991; Mehta and Josephs, 2010; Popma et al., 2007). Recent studies that measured basal hormone profiles have provided initial empirical support for the dual-hormone hypothesis on measures of aggression, dominance, and social status (Dabbs et al., 1991; Popma et al., 2007; Mehta and Josephs, 2010; Edwards and Casto, 2013; Pfattheicher et al., 2013; van Den Bos et al., 2013; Tackett et al., 2014). Status-relevant behaviors such as aggression are positively related to risk-taking or include risk-taking as a component (Tackett et al., 2014), neural systems that underlie aggression and risk-taking overlap to some extent (Mehta and Beer, 2010; Peper et al., 2013), and evolutionary theories suggest that risk-taking may have evolved as a behavioral strategy for attainment of social status (Daly and Wilson, 1997; Ellis et al., 2012). Thus, it seems plausible that the dual-hormone hypothesis may extend beyond measures of aggression and dominance to measures of risk-taking as well. To address this open question, we measured testosterone, cortisol, and risk-taking in two studies. In both studies we tested for independent associations between basal hormone concentrations and risk-taking (zero-order correlations) as well as hormonal interactions (testosterone  $\times$  cortisol interaction consistent with the dual-hormone hypothesis). According to traditional neuroendocrine theories, testosterone should be positively associated with risk-taking regardless of cortisol concentrations. According to the dual-hormone hypothesis, testosterone and cortisol should interact such that testosterone should be positively related to risk-taking *only* among individuals low in cortisol but not among individuals high in cortisol.

## 1. Study 1

Study 1 tested whether the interaction between basal testosterone and basal cortisol predicted trait risk-taking, which was assessed through a self-report scale (Zuckerman,

1991) as well as judgments by informants—well-acquainted individuals such as friends, significant others, and family members (Funder and Colvin, 1988; Vazire, 2006, 2010; Vazire and Carlson, 2011). Although self-reports are reasonably accurate predictors of behavior, they are susceptible to cognitive and motivational biases (e.g., the motivation to present oneself in a desirable manner). Informant ratings are advantageous because they predict behavior above and beyond self-reports (Vazire, 2010; Vazire and Carlson, 2011). A combination of self and informant reports provide a more complete picture of a person's behavioral tendencies than either perspective alone. Moreover, a dual-hormone interaction on self-reported risk-taking is more likely to be driven by a common third variable compared to a dual-hormone interaction on informant-reported risk-taking. Hence, the use of informant reports likely provides a more stringent test of the dual-hormone hypothesis. We conducted analyses to test whether testosterone and cortisol jointly predicted self- and informant-reported trait risk-taking.

### 1.1. Methods

#### 1.1.1. Participants

Participants ( $N=115$ ) between the ages of 18 and 30 years were recruited to control for age-related changes in steroid hormones and risk-taking (46.1% male, mean age: 20.57;  $SD=2.82$ ). Participants were a mix of students and community participants in the greater Austin area who completed the study in exchange for payment. Compensation varied between \$10 and \$25 depending on decisions made in tasks unrelated to the current research questions. The sample was diverse (48.2% Caucasian, 7.1% African-American, 27.7% Asian, 14.3% Latino, 2.7% who reported mixed ethnicity). By the standards of Cohen (1988, 1992), this sample size has adequate power (power  $>.80$ ) to detect effects of a magnitude of Pearson's  $r=.26$  and above. All procedures received ethics approval from the UT-Austin Institutional Review Board.

#### 1.1.2. Materials and procedure

**1.1.2.1. Self-reported trait risk-taking.** Participants completed online self-report measures prior to reporting to the lab. Self-reported trait risk-taking was assessed using Zuckerman's impulsive sensation-seeking scale (1991). This scale consists of nineteen true or false items concerning the tendency to take risks for the purpose of excitement and having unique experiences ( $\alpha=.83$ ). Scores on this scale have been shown to predict risk-taking behaviors (e.g., Hoyle et al., 2000; Zuckerman and Kuhlman, 2000; Steinberg, 2008; Pharo et al., 2011; Lauriola et al., 2014).

**1.1.2.2. Saliva samples and informant contact information.** After completing the online measures, participants reported to the lab between 1030 h and 1730 h. Participants provided informed consent, filled out questionnaires relevant to hormone measurement, and then provided a 2 mL saliva sample (Schultheiss and Stanton, 2009). The sample was immediately transported to a freezer. Participants were also asked to nominate at least one person to provide information on their personality (Vazire, 2006). Participants provided email addresses for one to three informants and were told that these informants would be contacted to fill out a short

Download English Version:

<https://daneshyari.com/en/article/6818814>

Download Persian Version:

<https://daneshyari.com/article/6818814>

[Daneshyari.com](https://daneshyari.com)