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Author: Pranjal Mehta Keith M. Welker Samuele Zilioli
Justin M. Carré

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Highlights

- Testosterone interacts with cortisol to predict risk-taking
- These effects were observed in two independent studies (total n = 280)
- Risk-taking measures included self reports, informant reports, and behavior.
- Similar effects emerged in males and females

Testosterone and cortisol jointly modulate risk-taking

Pranjal Mehta[a], Keith M. Welker[b], Samuele Zilioli[c], Justin M. Carré[d]

[a] University of Oregon, 1227 University of Oregon, Eugene, OR 97043, USA

[b] University of Colorado Boulder, Department of Psychology, 325 UCB, Boulder, CO 80309, USA

[c]Wayne State University, 42 W Warren Avenue, Detroit, MI 48202, USA

[d]Nipissing University, 100 College Drive, North Bay, ON P1B8L7, Canada

Correspondence concerning the article should be addressed to:

Pranjal H. Mehta, Department of Psychology, 1227 University of Oregon, Eugene, OR, 97403

Email: mehta@uoregon.edu. Phone: 541.346.0475

Abstract

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.

Keywords: *testosterone, cortisol, risk-taking, impulsivity, traits*

Testosterone and cortisol jointly modulate risk taking

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 & 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 & Herbert, 2008; Vermeersch et al., 2008 ; Sapienza et al., 2009; Campbell et al., 2010; Goudriaan et al., 2010; Ronay & von Hippel, 2010; Stanton et al., 2011a; Määttänen et al., 2013; Peper et al., 2013; van der Loos et al., 2013; Apicella et al., 2014; Evans & 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 & Kemeny, 2004; Roelofs et al., 2009; Tops & Boksem, 2011; Pfattheicher & 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 & 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 & Josephs, 2010; Edwards & 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 & 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 & 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 x 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 & Colvin, 1988; Vazire, 2006, 2010; Vazire & 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 & 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 (α = .83). Scores on this scale have been shown to predict risk-taking behaviors (e.g., Hoyle et al., 2000; Zuckerman & 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 & 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 survey as part of the study. Participants then completed tasks that were unrelated to research questions in the current paper and were paid for their participation.

1.1.2.3 Informant-Reported Risk-Taking. We followed published guidelines for collecting informant report data (Vazire, 2006). Informants were contacted via email using standardized text. The email indicated that [Name of participant] (referred subsequently to as "X") recently participated in a psychology study and nominated them to provide information about X's personality. Informants were then given a unique id number and were directed to a website that included a personality questionnaire about X. Informants filled out an online consent form prior to filling out the questionnaire. Informants were told that their responses would be used for psychological research only and would not be shared. No compensation was given to informants. If informants did not complete the questionnaire after the first email, a second reminder email was sent. Comparable to previous research, at least one informant provided ratings for 81% of the sample (Vazire, 2006). Among those participants with at least one informant rating, the average number of informants was 1.84 (SD = 0.73; approximately 35% had ratings from one informant, 45% had ratings from two informants, and 20% had ratings from three informants).

The informant report questionnaire included two face-valid items that assessed risk-taking. Informants were asked to indicate to what extent they saw X as someone who "enjoys taking risks" and "tends to play it safe" using a 7-point Likert type scale (1 = Disagree Strongly, 7 = Agree Strongly). We reverse coded the second item. Ratings on these items were highly correlated (r = .54, $p < .001$, Cronbach's α = .70) and thus were averaged to form one index of informant-reported risk-taking. For

those participants for whom multiple informants completed the questionnaire, we averaged across informant ratings.

1.1.2.4 Hormone assays. Saliva samples were stored in polystyerene tubes and frozen at -20°C. Once data collection was complete, the samples were shipped frozen overnight to Yerkes Endocrine Core Laboratory (Emory University, Atlanta, GA) and were assayed for testosterone and cortisol using radioimmunoassay kits purchased from Diagnostic Systems Laboratories, Inc. Intra-assay variability for testosterone averaged 8%, and inter-assay variability averaged 11%. Intra-assay variability for cortisol averaged 6%, and inter-assay variability averaged 10%.

1.1.3 Statistical Analyses

Testosterone was standardized separately within men and women (Mehta & Josephs, 2010), and cortisol was log base 10 transformed due to skewness (Skewness = 2.59, *SE* = .23). Testosterone standardized within sex was not substantially skewed (Skewness = .26, SE = .23). High scores on the testosterone distribution indicate high levels relative to other individuals of the same sex. This data analysis strategy in which males and females are combined in one analysis is in line with prior research on the dual-hormone hypothesis and has important benefits (Mehta & Josephs, 2010; Tackett et al., 2014). First, statistical power is increased in a combined analysis. Second, patterns of hormone–behavior relationships can be examined for statistically significant sex differences. Moderated regression analyses were conducted using mean-centered predictors to calculate an interaction term. Significant interactions were decomposed using the procedures of Aiken and West (1991).

1.2 Results

1.2.1 Preliminary Analyses

Tables 1 and 2 display zero-order correlations and descriptive statistics for the entire sample (Table 1), as well as for males and females separately (Table 2). As noted in the tables, there is a moderate correlation between self-reports and informant reports of trait risk-taking. The size of the correlation is consistent with self-informant agreement for other traits (Vazire, 2006). As expected, an

independent samples t-test (equal variances not assumed) revealed that men's testosterone levels (M = 141.90, SD = 39.53) were higher than women's (M = 27.27, SD = 11.32), $t(59.42) = 20.40$, $p < .001$, d = 5.30, 95% CI: 103.39, 125.88)[1]. However, men and women did not differ in basal cortisol ($t(110) = .93$, p = .355, d = .18), self-reported risk taking ($t(113) = -.36$, p = .723, d = -.07), or informant-reported risk-taking ($t(92) = -.49$, p = .624, d = -.10). This non-significant sex difference in risk-taking measures converges with recent meta-analyses, which found only small aggregate effect sizes for sex differences in risk-taking (Lauriola et al., 2014; Nelson, 2014). These meta-analyses included several individual studies that also found non-significant sex differences in risk-taking in line with the present results. Consistent with previous research (e.g., Popma et al., 2007; Mehta et al., 2008; Mehta & Josephs, 2010), testosterone and cortisol (log10 transformed) were moderately correlated (r = .38, $p < .001$). Although time of day was associated with testosterone (standardized within sex, r = -.24, p = .009) and cortisol (r = -.40, $p < .001$), age was not associated with testosterone (r = .03, p = .795) and marginally associated with cortisol (r = .17, p = .066).

1.2.2 Testosterone, Cortisol, and Trait Risk-Taking

Moderated regression analyses were used to examine whether testosterone and cortisol interacted to predict risk taking. These analyses were conducted in two separate regression models for self-reported and informant-reported risk taking. The results of these analyses are displayed in Table 2.

1.2.2.1 Self-reported trait risk-taking. Consistent with the dual-hormone hypothesis, there was a statistically significant Testosterone X Cortisol interaction for self-reported trait risk-taking (β = -.22, CI_{95}: [-6.06, -.52], $r_{partial}$ = -.22 p = .020). Simple slopes analyses (Aiken & West, 1991) revealed that testosterone was positively associated with self-reported risk-taking when cortisol was low (b = 1.78, se = .64, $t(106) = 2.79$, p = .006). However, when cortisol was high (+1 SD), the association between testosterone and self-reported risk-taking was non-significant (b = -.10, se = .54, $t(106) = -.18$, p = .859).

[1]The variance in testosterone levels is expected to be larger in men relative to women. The non-integer degrees of freedom in this test are due to correcting the degrees of freedom when equal variances cannot be assumed in an independent samples t-test.

Controlling for time of day (β = .06, p = .581) and age (β = -.01, p = .930) did not alter the significance of the Testosterone X Cortisol interaction (β =-.22, CI_{95}: [-.6.07, -.41], $r_{partial}$ = -.22, p = .025).

1.2.2.2 Informant-reported trait risk-taking. In further support of the dual-hormone hypothesis, there was a statistically significant Testosterone X Cortisol interaction for informant-reported risk taking (β = -.31, CI_{95}: [-1.96, -.40], $r_{partial}$ = -.28, p = .003). Similar to the pattern for self-reports, there was a positive association between testosterone and informant-reported risk-taking when cortisol was low (-1 SD) (b = .53, se = .19, t(88) = 2.77, p = .007), but not when cortisol was high (+1 SD) (b = -.17, se = .15, t(88) = -1.12, p = .268). This interaction remained significant (β =-.35, CI_{95}: [-.2.09, -.55], $r_{partial}$ = -.35, p = .001) when controlling for age (β = .26, p = .012) and time of day (β = .07, p = .557).[2]

1.2.3 Testing Moderation by Sex

Some prior research on the dual-hormone hypothesis has shown similar testosterone x cortisol interactions on behavior for males and females (e.g., Mehta & Josephs, 2010; Tackett et al., 2014), whereas other researchers have shown testosterone x cortisol interactions in males but not females (e.g., Welker et al., 2014). Hence, we also examined if sex moderated the interactive effects of testosterone and cortisol in two regression models featuring all main effects and cross products for self-reported and informant-reported risk taking. There were no three-way Sex X Testosterone X Cortisol interactions predicting self-reported (β = -.01, p = .891, 95% CI: -6.15, 5.35) or informant-reported (β = -.06, p = .607, 95% CI: -2.19, 1.29) risk-taking[3]. These results indicate that the testosterone x cortisol interaction effects shown in Figure 1 did not statistically differ between males and females[4].

[2]In Study 1, time of day X cortisol X testosterone interactions were non-significant for self-reported risk-taking (p = .117) and informant reported risk-taking (p = .572).

[3]Even though there were non-significant Sex X Testosterone X Cortisol interactions on self- and informant-reported risk-taking, we conducted follow-up analyses in which we examined Testosterone X Cortisol interactions separately in men and women to confirm that the interaction effects were similar across the sexes. We did not expect statistically significant effects in all of these analyses due to the dramatic reduction in statistical power. For self-reported risk-taking, the interaction terms show similar patterns in men (β = -.29, p = .056) and women (β = -.29, p = .033). For informant-reported risk-taking, the interaction terms also showed similar patterns in men (β = -.30, p = .114) and women (β = -.35, p = .018). These analyses converge with our main analyses, which showed statistically significant Testosterone X Cortisol interactions and non-significant Sex X Testosterone X Cortisol interactions.

[4]Even though analyses revealed statistically significant Testosterone X Cortisol interactions and non-significant Sex X Testosterone X Cortisol interactions, there was a statistically significant Cortisol X Sex interaction for self-reported risk-taking (β = -.28, p = .007) with a pattern that conceptually replicates prior research (Lighthall et al.,

2. Study 2

Study 1 demonstrated that endogenous cortisol and testosterone interact to predict risk-taking in line with the dual-hormone hypothesis. The results conceptually replicated with self and informant reports of trait risk-taking. An important next step is to test whether this dual-hormone interaction extends to a behavioral measure of risk-taking in an independent sample. Study 2 was designed to test the synergistic effects of endogenous testosterone and cortisol on a validated laboratory task of risk-taking behavior: the Balloon Analog Risk Task (BART) (Lejeuz et al., 2002). We used a well-powered sample of men who provided saliva samples and completed the BART. Based on the predictions of the dual-hormone hypothesis, we expected that testosterone and cortisol would interact to predict risk-taking behavior such that testosterone would be positively related to risk-taking only among low-cortisol individuals but not among high-cortisol individuals.

2.1 Methods

2.1.1 Participants

Participants were 165 male undergraduate students (M_{age} = 20.64, SD = 3.00) from Wayne State University who participated for partial course credit. The sample was diverse (38.2% Caucasian, 19.4% Black, 18.1% Asian, 4.8% Latino, .6% Native American, and 18.8% Other). This study was part of a larger research protocol examining the role of hormones and individual differences predicting risk taking (e.g., Welker et al., in press). The results presented here do not overlap with previously published research. Two participants were missing cortisol data, two were missing both cortisol and testosterone data, and one participant failed to understand the directions on the risk taking measure. Thus, the final sample size consisted of 160 participants, which had adequate power (.8) to detect effects greater than or equal to r = .22.

2.1.2 Materials and Procedure

2009; Van den Bos et al., 2009). Specifically, simple slopes analyses revealed a positive association between cortisol and risk-taking in men (b = 4.80, se = 2.22, t(103) = 2.16, p = .033) and a negative trend in women (b = -3.81, se =2.09, t(103) = -1.82, p = .071). The cortisol X sex interaction was non-significant for informant-reported risk-taking (β = -.14, p = .207).

Participants arrived in the laboratory, were briefed on the study procedures, and completed the informed consent. To reduce the effects of diurnal variation in testosterone and cortisol, participation sessions were held between 1100 h and 1700 h, with the exception of one participant session that was held until 6 PM. Participants first completed questionnaires for approximately 25-30 minutes before providing their baseline saliva sample via unstimulated passive drool through a straw.

2.1.2.1 Risk-Taking Behavior. After providing saliva samples, participants completed a digital version of the Balloon Analog Risk Task (BART; Lejeuz et al., 2002), a widely-used behavioral measure of risk-taking propensity wherein participants had to acquire "money points" in a temporary reserve by clicking an on-screen button to pump up 30 virtual balloons in sequence. After each pump participants earned $.05, and for each $.10 of points earned, a raffle ticket was entered in a drawing for a $150 gift card. All balloons were set to randomly explode after any number of pumps between one and 30 were administered. Participants were also given the option to save the points from the temporary reserve before the balloon exploded and start pumping up the next balloon in the task. Thus, each decision to continue pumping the balloon was an act of risk-taking behavior. Similar to the other researchers (Lejeuz et al., 2002), the average number of pumps that were given to balloons that did not explode was used as the measure of risk taking. The BART has been found to have test-retest reliability (White et al., 2008) and high external validity, predicting other measures of risk-taking behaviors such as alcohol use, substance use, and risky sexual behavior (Lejeuz et al., 2002). The BART has convergent validity with other psychological measures predicting risk-taking, such as impulsiveness, behavioral constraint, sensation seeking, and psychopathy (Lejeuz et al., 2002; 2003; Hunt et al., 2005). For example, Lejeuz et al. (2002) report moderate positive correlations between BART scores and self-report trait measures implicated in risk-taking such as impulsiveness ($r = .28$) and sensation seeking ($r = .35$). Additionally, the BART has been used to study risk-taking from a wide variety of approaches including studying the neural mechanisms of risk-taking (Hao et al., 2008), inner-city drug use (Hopko et al., 2006), and risk-avoidance in anxiety (Maner et al., 2007).

2.1.2.2 Salivary Hormone Samples and Assays. Saliva samples were stored in polystyerene tubes and frozen at -20°C until they were assayed using commercially available kits from DRG international. Intra-assay variability for testosterone averaged 6%, and inter-assay variability averaged 9%. Intra-assay variability for cortisol averaged 6%, and inter-assay variability averaged 6%. Basal testosterone outliers were identified (N = 2, appearing in the upper distribution tail) and Winsorized to 3 SDs.[5] Because cortisol values were strongly positively skewed (Skewness = 6.28, SE = .19), cortisol values were transformed using a log10 transformation. Testosterone concentrations did not show such a substantial skew (Skewness = .97, SE = .19).

2.2 Results

2.2.1 Preliminary Analyses

Table 4 presents the descriptive statistics and correlations for the measures included in Study 2. Consistent with Study 1, testosterone and cortisol (log10 transformed) were moderately correlated (r = .33, p < .001). Time of day was not significantly associated with testosterone (r = -.07, p = .389) or transformed cortisol concentrations (r = -.04, p = .635). Age was unrelated to testosterone levels (r = -.08, p = .299), but had a marginally significant, negative association with transformed cortisol (r = -.16, p = .051).

2.2.2 Testosterone, Cortisol, and Risk-Taking Behavior

Moderated regression analyses were used to assess whether testosterone and cortisol interacted to predict risk-taking behavior. Testosterone and transformed cortisol concentrations were mean-centered and multiplied to create an interaction term. The results of this analysis are presented in Table 4. Conceptually replicating Study 1, there was a statistically significant Testosterone X Cortisol interaction on risk-taking behavior (β =-.22, CI_{95}: [-.08, -.01], $r_{partial}$ = -.21, p = .005). Decomposing this interaction (Hayes, 2013) revealed a marginally significant positive association between testosterone and risk-taking behavior when cortisol was low (b = .02, se = .01, t(156) = 1.97, p = .051; -1 *SD*), but a non-significant negative association between testosterone and risk-taking behavior when cortisol was high (b = -.01, se =

[5] Study 1 did not have any testosterone outliers that exceeded 3 SDs.

.01, $t(156) = -1.42$, $p = .156$; +1 *SD*). These simple slopes are presented in Figure 2. Adding time of day ($\beta = .01$, $p = .880$) and age ($\beta = -.06$, $p = .429$) as covariates to this model did not alter the significance of the Testosterone X Cortisol interaction ($\beta = -.21$, CI_{95}: [-.08, -.01], $r_{partial} = -.21$, $p = .011$).[6]

2.2.3 Internal Meta-Analysis

Next we conducted an internal meta-analysis across both studies to provide a better estimate of the magnitude and pattern of the dual-hormone interaction on risk-taking. This meta-analytic approach boosts statistical power and allows for more precise estimation (Cumming, 2013). We standardized testosterone, cortisol, and risk-taking within each study and aggregated the two datasets (e.g., Zilioli et al., 2014a). Then, we tested the testosterone X cortisol interaction effect across both studies. This analysis revealed a marginally significant main effect of testosterone of small effect size ($\beta = .11$, $t(266) = 1.75$, $p = .081$, $r_{partial} = .11$, 95% CI: [-.01, .23]), no main effect of cortisol ($\beta = .01$, $t(266) = .12$, $p = .902$, $r_{partial} = .01$, 95% CI: [-.11, .12]), and a statistically significant T x C interaction of moderate effect size ($\beta = -.22$, $t(266) = -4.26$, $p < .001$, $r_{partial} = .-.25$, 95% CI: [-.32, -.12]). Simple slopes analyses indicated a positive association between testosterone and risk-taking when cortisol was low ($b = .32$, $se = .09$, $t(266) = 3.41$, $p < .001$), but a non-significant negative association between testosterone and risk-taking when cortisol was high ($b = -.11$, $se = .07$, $t(266) = -1.58$, $p = .114$).

3. Discussion

Previous studies have demonstrated that testosterone interacts with cortisol to predict behaviors related to dominance, aggression, and social status (Dabbs et al., 1991; Popma et al., 2007; Mehta & Josephs, 2010; Geniole et al., 2011; Denson et al., 2013; Edwards & Casto, 2013; Pfattheicher et al., 2013; Van Den Bos et al., 2013; Tackett et al., 2014; Welker et al., 2014; Zilioli et al., 2014[CB1]b). The present research provides new evidence that testosterone and cortisol interact to predict risk-taking. Across two studies (total N = 280), we found a positive association between basal testosterone and risk-taking among low basal cortisol — but not high basal cortisol—individuals. This effect was evident in both males and females and replicated across measures of self-reported trait risk-taking, informant-

[6] In study 2, there was no significant time of day X cortisol X testosterone interaction (p = .318).

reported trait risk-taking, and risk-taking behavior. The fact that the results emerged on three different markers of risk-taking in two independent samples provides greater confidence in the robustness of the effects. These findings advance knowledge on the neuroendocrinology of risk-taking in suggesting that testosterone and cortisol work in concert to regulate risk-taking according to the predictions of the dual-hormone hypothesis (Mehta & Josephs, 2010) (for a related theory that predicts hormone ratio effects instead of statistical interaction effects, see Terburg, et al., 2009; Montoya et al., 2012).

A viable mechanism for the current results resides in the functional crosstalk between the HPG and HPA axis, with cortisol potentially buffering the pathway between testosterone and risky behavior. Glucocorticoids inhibit HPG axis function at multiple levels, decrease androgen receptor levels, and suppress the effects of testosterone on target tissues (Smith et al., 1985; Johnson et al., 1992; Burnstein et al., 1995; Chen et al., 1997; Tilbrook et al., 2000; Viau, 2002; Lin et al., 2014), which all may lead to an inhibitory effect of elevated cortisol on testosterone's behavioral effects. Specifically, when HPA axis activity is reduced as reflected by low cortisol levels, the reproductive axis may operate efficiently leading to a robust positive effect of testosterone on risk-taking. In contrast, when HPA axis activity is heightened (i.e. high cortisol), the reproductive axis may be blocked or inhibited resulting in a null association between testosterone and risk-taking.

A related mechanism for dual-hormone effects on risk-taking is through increased activity in neural systems implicated in reward sensitivity. In both animal and human studies, testosterone boosts reward-seeking behaviors, psychological states associated with reward (e.g., enjoyment, Mehta et al., 2014), and anticipation of reward via interactions with dopamine in the ventral striatum (e.g., nucleus accumbens) (Packard, et al., 1997; 1998; van Honk et al., 2004; Hermans et al., 2010; Op de Macks et al., 2011). In contrast, exogenous cortisol down-regulates activity in a neural reward network including the ventral striatum (Montoya et al., 2014; but see also Lewis et al., 2014), and activity in this region predicts increased risk-taking behaviors (Galvan et al., 2007; Rao et al., 2008; Somerville et al., 2010). Thus, the joint effect of testosterone and cortisol on risk-taking may be driven by the reinforcing effects of dopamine in these motivational and reward regions of the mesolimbic pathway.

A third putative mechanism for dual-hormone interactions on risk-taking may be through inhibition of pre-frontal regions implicated in self-regulation and impulse control (e.g., orbitofrontal cortex, OFC). Reduced OFC activity is related to risky decisions (Eshel et al., 2007), and testosterone's association with increased risk-taking and impulsive aggression is explained by reduced OFC engagement (Mehta & Beer, 2010) as well as reduced OFC volume (in males, Peper et al., 2013). In contrast, heightened cortisol during risk-taking is associated with increased OFC activity (Freeman & Beer, 2010). Further, testosterone reduces functional connectivity between the OFC and subcortical regions such as the amygdala (van Wingen et al., 2010; Spielberg et al., 2014), and cortisol is related to increased functional connectivity between the medial prefrontal cortex and amygdala (Veer et al., 2012). These findings suggest that dual-hormone interactions on risk-taking may be explained by structural and functional differences in the OFC as well as prefrontal-subcortical connectivity.

The current findings may also reflect hormonal interactions between approach and avoidance motivational systems (Carver & White, 1994). Testosterone has been associated with approach-oriented (e.g., dominance motivation, Mazur & Booth, 1998) and appetitive motivation (e.g., Packard et al., 1997; van Honk et al., 2004; Hermans et al., 2010; Op de Macks et al., 2011), whereas cortisol is associated with social evaluative stress and behavioral inhibition (Dickerson & Kemeny, 2004; Roelofs et al., 2009). Thus, a profile of high testosterone (approach and appetitive motivation) and low cortisol (low behavioral inhibition) may lead to heightened risk-taking, whereas the greater behavioral inhibition tendencies associated with high cortisol may counteract the influence of high testosterone on risk-taking behavior (for similar arguments, see Popma et al., 2007; Terburg, et al., 2009; Mehta & Josephs, 2010; Carré & Mehta, 2011; Maner et al., 2012; Montoya et al., 2012).

The present results have implications for risky behaviors in a number of different domains. The trait and behavioral risk-taking measures examined in the present studies are reliable predictors of real-world risk-taking behaviors, including sexual risk-taking, substance use, and dangerous driving (Hoyle et al., 2000; Zuckerman & Kuhlman, 2000; Lejeuz et al., 2002; 2003; Hunt et al., 2005; Steinberg, 2008; Pharo et al., 2011; Lauriola et al., 2014;). Thus, an important extension of the current research will be to

investigate whether testosterone and cortisol interact to predict these real-world risk-taking behaviors as well (see also Tackett et al., 2014). Another future direction will be to test the dual-hormone hypothesis on financial risk preferences. Indeed, overly risky financial decisions can lead to devastating monetary losses for the self and others, and stable and fluctuating components of the endocrine system are theorized to play a role in the macro-level behavior of financial markets (Coates & Herbert, 2008; Apicella et al., 2014; Kandasamy et al., 2014).

The current research may also inform developmental theories of risk-taking. Risk-taking is heightened during adolescence (Figner et al., 2009), and one of the leading causes of death among this age group can be attributed to poor and risky choices (e.g., motor-vehicle crashes, other unintentional injuries; Eaton et al., 2008). Developmental researchers have theorized that pubertal increases in sex hormones such as testosterone may increase risky behavior (Sommerville et al., 2010; Peper et al., 2013), but the present study brings up the possibility that pubertal testosterone may interact with cortisol to modulate risky decision-making in adolescents (see related evidence for dual-hormone interactions predicting externalizing psychopathology in adolescents, Tackett et al., 2014). Further, changes in risky decisions have been observed in older adults compared to younger adults (Mata et al., 2011). Thus, we suspect that the dual-hormone hypothesis may potentially explain changes in risk-taking throughout the lifespan including older adulthood.

The present findings also have implications for biosocial theories of status attainment. Status in face-to-face groups can be defined as an individual's prominence, respect, and influence in the eyes of others (Anderson & Kilduff, 2009). Males in particular take risks in order to out-compete rival males for the attention of attractive females, and these risk-taking behaviors may increase access to mating opportunities and enhance one's status (Daly & Wilson, 1997; Ronay & von Hippel, 2010; Ellis et al., 2012). In support of a relationship between risk-taking and status pursuit, research indicates that the presence of peers increases risk-taking compared to the absence of peers among adolescents through enhanced reward processing (ventral striatum activity; Chein et al., 2011). These results suggest that heightened risk-taking may be a behavioral strategy aimed at enhancing one's reputation in the eyes of

others especially during adolescence (Ellis et al., 2012). However, it remains unclear to what extent risk-taking in the present research was influenced by status concerns. Future research should build upon the present findings by considering contextual factors such as the presence versus absence of peers in order to understand how dual-hormone interactions are related to risk-taking in the pursuit of social status.

There are several limitations of the present research that should be addressed in future studies. First, we assessed late morning or afternoon hormone concentrations and examined their associations with risk-taking. Testosterone and cortisol measured around the same time of day are moderately stable across several weeks (Liening et al., 2010), suggesting they are reasonable measures of basal testosterone and cortisol. Test-retest reliability of these two hormones along with evidence for test-retest reliability of the risk-taking measures we employed (Zuckerman & Kuhlman, 2000; White et al., 2008) suggest that the current results are likely driven by associations between stable hormone profiles and stable risk-taking propensity. Nevertheless, there are diurnal rhythms in testosterone and cortisol, and these hormones can fluctuate in decision-making contexts (e.g., Coates & Herbert, 2008; Apicella et al., 2014). Thus, it will be important for future studies to collect additional saliva samples in order to investigate stable and fluctuating components of the HPG and HPA axes. Second, we cannot be certain that testosterone and cortisol have causal influences on risk-taking because we measured endogenous hormone concentrations. Future studies that pharmacologically manipulate testosterone and cortisol are needed to confirm causality. Third, although we tested the dual-hormone hypotheses on widely used trait and behavioral measures of risk-taking, it is important to extend the results of the present study to other behavioral measures of risk-taking, such as the Iowa Gambling Task (Bechara et al., 1994), Columbia Card Task (Figner et al., 2009), economic decision-making measures (e.g., Gneezy & Potters, 1997; Apicella et al., 2014), and real-world behaviors such as substance use and sexual risk-taking.

The two studies revealed consistent positive slopes between testosterone and risk-taking only among low-cortisol individuals in line with the dual-hormone hypothesis, but there was a trend toward a negative slope between testosterone and risk-taking behavior among high-cortisol individuals especially in Study 2 (Figure 2). This negative slope was non-significant in Study 2 and the internal meta-analysis

and therefore may be due to random statistical variation or may have a very small effect size (for similar negative slopes, see Mehta & Josephs, 2010; Edwards & Casto, 2013; Tackett et al., 2014). Follow-up research will be required to unpack the pattern and mechanisms for testosterone's role in risk-taking among high-cortisol individuals.

Finally, future research is needed to corroborate whether dual-hormone associations with risk-taking are similar across the sexes. Our first study included both males and females and showed a consistent pattern of the dual-hormone interaction across males and females, but Study 2 included a very large sample of only males making it unknown whether these results will extend to females. We encourage future researchers to collect large samples of males and females and test for dual-hormone interactions in both sexes. These future studies should also explore other aspects of these neuroendocrine systems that may account for variability in risk-taking, such as sex differences or individual differences in neural sensitivity to steroid hormones (Ketterson et al., 2009; Rosvall et al., 2012).

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Conflicts of Interest

The authors declare no conflicts of interest.

Author Contributions

PHM and KMW contributed equally to this work. PHM, KMW, and JMC designed the studies and analyzed the data. PHM and KMW wrote the first draft of the paper. JMC and SZ assisted with data analysis interpretation and helped write the paper.

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Table 1. Correlations and Descriptive Statistics for Study 1 (Entire Sample).

	1	2	3	4	5	6	7	8	M	SD	N
1. T (pg/mL)	—								80.56	63.91	114
2. C (ng/mL)	.19*	—							.21	.17	112
3. ZT	.38***	.24*	—						.01	1.00	112
4. C_{log10}	.20*	.90***	.29**	—					-.77	.28	112
5. Self-report RT	.01	.04	.16	.07	—				9.46	4.43	115
6. Inf.Report RT	-.07	-.20†	.03	-.11	.43***	—			4.20	1.22	94
7. Age	.18†	.20*	.03	.17†	-.04	.16	—		20.53	2.82	115
8. Time	-.14	-.34***	-.24**	-.40***	-.01	.08	-.20*	—	14:44	2:36	115

†p < .07, *p < .05, **p < .01, *** p < .001

Note: T = Testosterone, C = Cortisol, ZT = Testosterone standardized within sexes, C_{log10} = Log transformed cortisol, Self-report RT = Self-reported Trait Risk-Taking, Inf. Report RT = Informant-Reported Trait Risk-Taking, Time = time of saliva sample.

Table 2. Correlations and Descriptive Statistics for Study 1 (Males and Females Separated).

Men	1	2	3	4	5	6	7	M	SD	N
1. T (pg/mL)	—							141.90	39.53	53
2. C (ng/mL)	.31*	—						.23	.19	51
3. C_{log10}	.29*	.89***	—					-.74	.28	51
4. Self-report RT	.10	.19	.28*	—				9.30	4.28	53
5. Inf.Report RT	-.14	-.21	-.12	.30*	—			4.13	1.09	43
6. Age	-.03	.28*	.24†	-.15	.27†	—		21.15	3.29	53
7. Time	-.34*	-.28*	-.32*	.01	.20	-.17	—	14:38	2:40	53

Women	1	2	3	4	5	6	7	M	SD	N
1. T (pg/mL)	—							27.27	11.32	61
2. C (ng/mL)	.17	—						.20	.15	61
3. C_{log10}	.29*	.92***	—					-.79	.283	61
4. Self-report RT	.20	-.11	-.09	—				9.60	4.58	62
5. Inf.Report RT	.14	-.19	-.09	.52***	—			4.26	1.33	51
6. Age	.11	.03	.06	.11	.08	—		20.00	2.23	62
7. Time	-.16	-.40**	-.47***	-.04	-.02	-.24†	—	14:50	2:33	62

†p < .07, *p < .05, **p < .01, *** p < .001

Note: T = Testosterone, C = Cortisol, C_{log10} = Log transformed cortisol, Self-report RT = Self-reported Trait Risk-Taking, Inf. Report RT = Informant-Reported Trait Risk-Taking, Time = time of saliva sample.

Table 3.

Multiple Regression Model of Testosterone x Cortisol Interaction Predicting Self-Reported and Informant Reported Risk-Taking (n = 110 for self reports, n = 92 for informant reports)

	B	β	t(106)	p	$r_{partial}$	95% CI
Self-Reported Risk Taking						
Testosterone	.84	.19	1.93	.056	.18	(-.02, 1.70)
Cortisol[1]	.15	.01	.10	.922	.01	(-2.88, 3.18)
Testosterone x Cortisol[2]	-3.29	-.22	-2.36	.020	-.22	(-6.06, -.52)
	B	β	t(88)	p	$r_{partial}$	95% CI
Informant Reported Risk-Taking						
Testosterone	.18	.15	1.38	.170	.15	(-.08, .43)
Cortisol[1]	-.66	-.16	-1.52	.133	-.16	(-1.52, .21)
Testosterone x Cortisol[2]	-1.18	-.31	-3.02	.003	-.31	(-1.96, -.40)

Notes.
B indicates unstandardized regression coefficients. β indicates standardized regression coefficients.
[1] Log-transformed because of skew in the distribution
[2] Interaction term computed from mean centered predictors.

Table 4. Correlations and Descriptive Statistics for Study 2.

	1	2	3	4	5	6	M	SD	N
1. T (pg/mL)	—						102.48	40.65	163
2. C (ng/mL)	.19*	—					2.97	3.55	161
3. C_{log10}	.33**	.77**	—				0.33	0.34	161
4. Risk Taking	.01	-.04	.07	—			9.81	3.21	164
5. Age	-.08	-.12	-.16†	-.09	—		20.64	3.00	163
6. Time of Day	-.07	.04	-.04	.01	.00	—	13.17	1.46	165

†p < .052, *p < .02, **p < .001

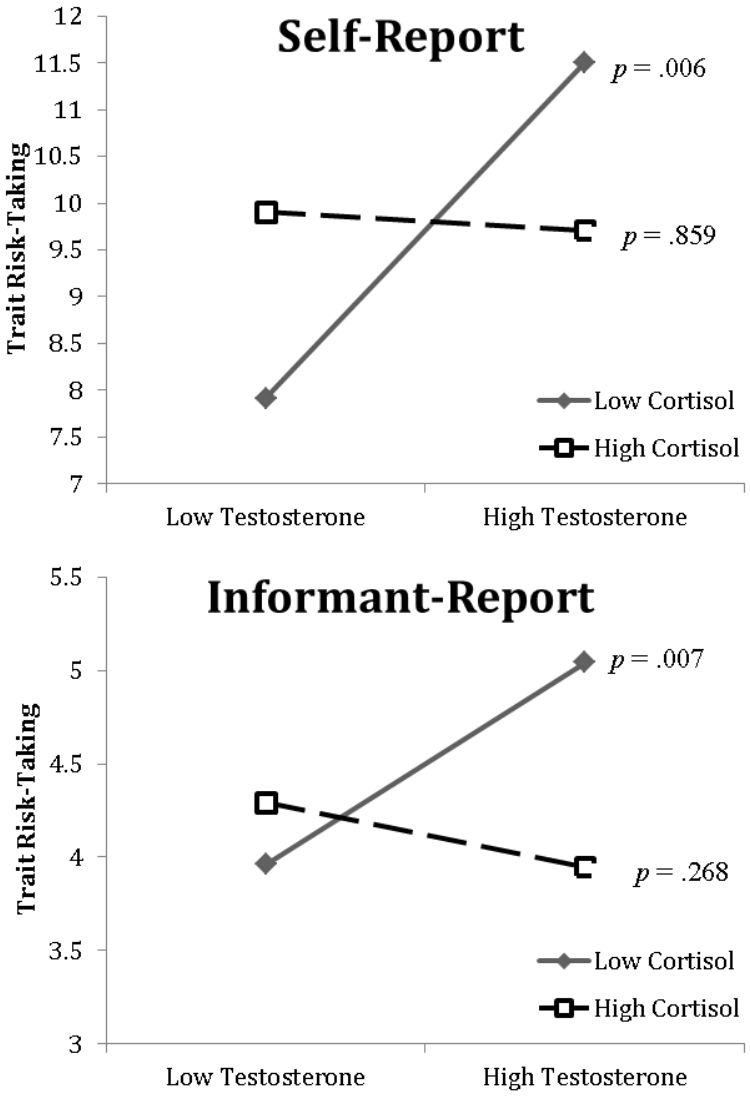
Table 5.

Multiple Regression Model of Testosterone x Cortisol Interaction Predicting Risk-Taking Behavior (n = 160 men)

	B	β	t(156)	p	$r_{partial}$	95% CI
Risk Taking Behavior						
Testosterone	.00	.06	.65	.516	.05	(-.01, .02)
Cortisol[1]	.48	.05	.62	.537	.05	(-1.06, 2.03)
Testosterone x Cortisol[2]	-.05	-.22	-2.67	.008	-.21	(-.08, -.01)

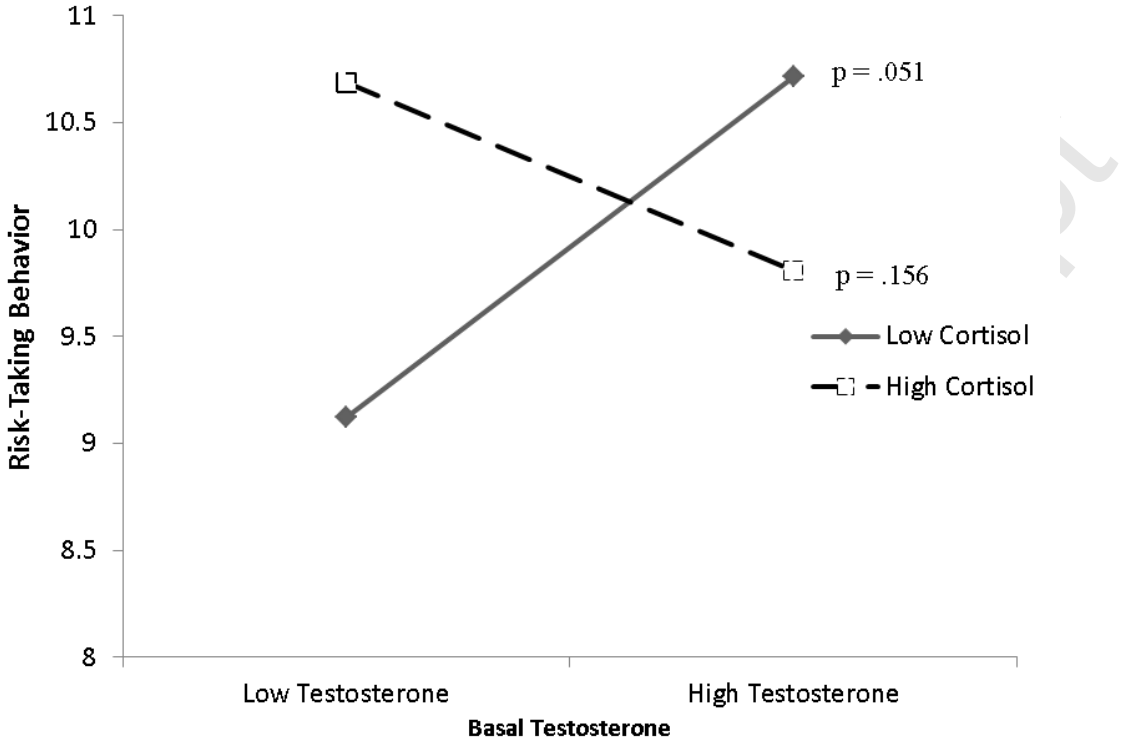
Notes.
B indicates unstandardized regression coefficients. β indicates standardized regression coefficients.
[1] Log-transformed because of skew in the distribution
[2] Interaction term computed from mean centered predictors.

Figure 1. Self-Reported and Informant-Reported Risk Taking as a Function of Testosterone and Cortisol (Study 1).



Note: Plotted points represent conditional low and high values (±1 SDs) of Testosterone (standardized within each sex) and Cortisol (log-transformed)

Figure 2. Risk-Taking Behavior as a Function of Testosterone and Cortisol (Study 2).



Note: Plotted points represent conditional low and high values (±1 SDs) of Testosterone and Cortisol (log-transformed)