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Endogenous testosterone and cortisol jointly influence reactive aggression in women

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Summary The dual-hormone hypothesis posits that the effect of testosterone on social behavior is moderated by cortisol. The present study tested this hypothesis with a competitive reactive aggression paradigm in 53 healthy undergraduate women. Salivary cortisol and testosterone were assessed at baseline. Participants were personally insulted and subsequently given the opportunity to retaliate by administering blasts of white noise to the provocateur. Participants were randomly assigned to win or lose the aggressive competition. Basal testosterone positively predicted reactive aggression and state dominance, but only among participants with high concentrations of basal cortisol. The corresponding, reverse pattern was found for state submissiveness. Winners also had higher concentrations of testosterone than losers following the aggressive competition. We discuss the role of heightened reactivity to social provocation as a possible explanation for these effects.

1. Introduction

Reactive aggression is known as hostile, impulsive, or affective aggression and can encompass verbal and physical assaults, road rage, domestic and workplace violence, and homicide. This type of aggression is in contrast to instrumental or proactive aggression, which is aggression enacted to obtain a secondary goal (e.g., a violent assault to obtain money). Decades of research have documented social, genetic, personality, neurobiological, and environmental determinants of reactive aggression. Of relevance for the present research is recent work implicating the hormones testosterone and cortisol as risk factors for aggression and violence (Terburg et al., 2009; Carré and Mehta, 2011; Carré et al., 2011; Eisenegger et al., 2011). We examined the interactive influence of endogenous concentrations of testosterone and cortisol on reactive aggression among undergraduate women during a competitive aggression paradigm.

1.1. The dual-hormone hypothesis

Testosterone is a steroid hormone secreted by the hypothalamic–pituitary–gonadal (HPG) axis and has been implicated in aggression and dominance (Eisenegger et al., 2011). Cortisol is a glucocorticoid hormone released by the hypothalamic–pituitary–adrenal (HPA) axis, often in response to stress. Cortisol has been implicated in submissive behavior,

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feelings of submissiveness, inhibition, and low levels of aggression (van Goozen et al., 1998; Goldsmith and Lemery, 2000; McBurnett et al., 2000; Pajer et al., 2001; van de Wiel et al., 2004; Oosterlaan et al., 2005; Denson et al., 2009). Endogenous testosterone and cortisol concentrations measured at the same time of day are relatively stable (Liening et al., 2010). These relatively stable hormone profiles explain part of the individual differences in aggressiveness, dominance, and submission (Sellers et al., 2007; Newman and Josephs, 2009). However, testosterone and cortisol concentrations are also responsive to chronic and immediate social experiences.

Although the link between testosterone and aggression is robust in animals (Brain and Haug, 1992), evidence for this relationship in humans can be inconsistent (Book et al., 2001; Archer et al., 2005; Book and Quinsey, 2005; Eisenegger et al., 2011). As men generally have higher concentrations of testosterone than women (McDermott et al., 2007), the majority of the testosterone-aggression literature has focused on men (Virkkunen, 1985; Dabbs and Morris, 1990; Gray et al., 1991; Berman et al., 1993; Popma et al., 2007; Mehta and Josephs, 2010). However, a smaller group of studies has explored testosterone's role in female aggression. Consistent with the research in men, some studies show a positive relationship between testosterone and aggression in women (Dabbs et al., 1988; Dabbs and Hargrove, 1997; Oliveira et al., 2009), whereas others reported null effects (Carré et al., 2011).

Recently *the dual-hormone hypothesis* was proposed to account for the inconsistent findings of testosterone on social behavior (Popma et al., 2007; Mehta and Josephs, 2010). According to the dual-hormone hypothesis, the effect of testosterone on aggression in humans is moderated by cortisol such that testosterone is positively associated with aggression only when cortisol concentrations are low. The dual-hormone hypothesis concerns the interactive effects of cortisol and testosterone because cortisol can inhibit activation of the HPG axis and testosterone can inhibit activation of the HPA axis (Viau, 2002; Terburg et al., 2009). Nonetheless, it has been noted that both axes "still largely function independently" (Yildirim and Derksen, in press, p. 12), suggesting that cortisol may influence testosterone via behavior rather than through the physiological inhibition of testosterone synthesis or activation. Specifically, the behavioral inhibition associated with higher concentrations of cortisol may counteract the approach orientation associated with testosterone (e.g., Windle, 1994).

In support of the dual-hormone hypothesis is research demonstrating that low levels of cortisol and high levels of testosterone characterize aggressive clinical populations such as violent offenders, psychopathic individuals, and adolescents with conduct disorder (Dabbs et al., 1991; Popma et al., 2007; Glenn et al., 2011). Two previous studies on testosterone—cortisol interactions and aggression were conducted in males using reports of aggression (Dabbs et al., 1991; Popma et al., 2007). Dabbs et al. (1991) reported that endogenous testosterone positively correlated with severity of violence among male adolescent offenders only when cortisol was low. Popma et al. (2007) reported the same dual-hormone interaction on self-reported impulsive aggression in delinquent adolescent males.

Aggressive behavior often emerges as a retaliatory response to social provocation (Bettencourt et al., 2006). However, only one laboratory study to our knowledge has examined the testosterone—cortisol interaction as a predictor of provoked aggression (Geniole et al., 2011). Undergraduate men were provoked by being either socially excluded during a ball-tossing game with two other fictitious participants or included in the game. They then completed an aggression task whereby they could aggress by taking money away from the other fictitious participants. During the aggression task, the fictitious participant who had socially excluded (or included) the actual participant also periodically took their money. Thus, by "adding insult to injury", the provocation level was likely higher in the social exclusion condition than the social inclusion condition. Moreover, aggressive behavior was positively correlated with enjoyment of the aggression task for participants in the social exclusion condition but not the social inclusion condition. This suggests that socially excluded participants enjoyed aggressively retaliating against the provocateur, likely because the strength of provocation was stronger in the social exclusion than inclusion condition. Although their results were not statistically significant, inspection of the pattern of data suggests that when socially included, testosterone positively predicted aggression at low values of cortisol (1 *SD* below the sample mean), but not at high values (1 *SD* above the sample mean). When socially excluded, this pattern was reversed: testosterone positively correlated with aggression at *high* values of cortisol, albeit not significantly so.

The Geniole et al. (2011) study did not have adequate statistical power to identify significant testosterone—cortisol interactions, but the patterns raise the intriguing notion that the dual-hormone effects observed in prior research may be reversed in the presence of highly potent forms of social provocation such as social exclusion or a personal insult. Indeed, social exclusion increases aggressive behavior and threatens fundamental human needs (Williams, 2001; Leary et al., 2006). Moreover, personal insults and similar social provocations have been described as "perhaps the most important single cause of human aggression" (Anderson and Bushman, 2002, p. 37). Thus, we investigated the notion that testosterone may be positively related to reactive aggression after social provocation in individuals with *high* cortisol, but not in individuals with low cortisol. This hypothesis was further informed by findings that cortisol administration increases reactive aggression in women (Böhnke et al., 2010) and testosterone administration lowers empathy in women when fetal testosterone levels are taken into account (van Honk et al., 2011). Inspired by these previous studies, we examined the interaction of endogenous testosterone and cortisol as predictors of reactive aggression in women who were personally insulted by another female participant.

1.2. Social competition

The present study examined aggressive behavior within the context of a competitive aggression paradigm. Outside of the aggression context, in response to competition, testosterone concentrations sometimes increase for winners and decrease for losers (Booth et al., 1989; Gonzalez-Bono et al., 1999;

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Giammanco et al., 2005; Edwards et al., 2006). Mazur's (1985) biosocial theory of status explains these findings. The model describes a feedback loop between an individual's testosterone level and his or her assertiveness in attempting to achieve or maintain interpersonal status. Following human and animal competition, winning is often associated with high testosterone concentrations (rising or maintained at an elevated level). Work with rodents has shown that this increase in testosterone reinforces further competitiveness as the winner is increasingly willing to compete for higher status (Trainor et al., 2004). Conversely, a loss of status sometimes lowers testosterone concentrations in humans, which may inhibit the individual from engaging in further potentially damaging competition (Mehta and Josephs, 2006).

Scholars have theorized that this biosocial model of status is specific to men because of several null results in women (Mazur and Booth, 1998). Two recent studies challenge this view. Female badminton and soccer players who won their matches rose in testosterone and female losers dropped in testosterone, suggesting that the biosocial model of status may indeed extend to women (Oliveira et al., 2009; Jimenez et al., in press). Although the findings from these studies are promising, both involved physical activity and were conducted in uncontrolled field settings. A recent laboratory study experimentally manipulated wins and losses in a competitive setting without physical activity by having women compete for money on a paper-and-pencil perception and attention task (Costa and Salvador, in press). Winners showed increases in testosterone. No effects were observed for losers. The present research extended the biosocial model of status to *aggressive competition* in women.

Regarding cortisol, loss of status may increase cortisol (Dickerson and Kemeny, 2004), but not always in response to competition (Mazur et al., 1997). One study showed cortisol increases among winners of a women's rugby match (Bateup et al., 2002). However, most of the prior hormone research has been conducted in males (for exceptions, see Carré et al., 2009 and Mehta et al., 2008). In the present research, we manipulated whether women won or lost an aggressive competition and examined changes in testosterone and cortisol. This is the first study to our knowledge to do so within a competitive aggression context.

1.3. The present research

In the current study, undergraduate women were personally insulted by a fictitious female participant and given the opportunity to retaliate by delivering blasts of white noise to the provocateur under the guise of a competitive reaction time task. Participants either won or lost the aggressive competition. We expected baseline testosterone and cortisol to jointly influence aggressive behavior in response to social provocation. According to the dual-hormone interaction pattern shown in previous work (Dabbs et al., 1991; Popma et al., 2007; Mehta and Josephs, 2010), testosterone should be positively related to aggression when cortisol is *low*, but not when cortisol is high. However, we speculated that this interaction pattern would reverse in the present study because of the presence of a social provocation, a possibility suggested by recent work (Geniole et al., 2011). If this reversal hypothesis is correct, then testosterone should be positively related to reactive aggression after social provocation in individuals with *high* cortisol, not individuals with low cortisol. We also expected that testosterone would increase among participants randomly assigned to win the competition, whereas testosterone would decrease among participants assigned to lose the competition. Given the mixed findings in the literature, we did not make predictions about cortisol changes in response to competition.

2. Method

2.1. Participants and design

This study was carried out in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants and the UNSW Human Research Ethics Committee approved the protocol. A total of 60 female undergraduate students from the University of New South Wales participated in the study as part of their course requirements or in exchange for AUD $20. Exclusion criteria included: use of oral contraceptives; pregnancy/breastfeeding; immune, cardiovascular, metabolic, or kidney disorders; smoking, steroid use; cold or infection. Participants were also asked not to exercise 12 h prior to the study or consume caffeine 2 h prior. Data from 4 participants were not analyzed because they had baseline concentrations of cortisol or testosterone more than ± 3 *SD*s from the sample mean. An additional 3 participants were excluded from analyses because they were suspicious of the study's aims. This left a total of 53 women (M_{age} = 20.74 years, *SD* = 6.08). Participants were randomly assigned to the win (n = 30) or lose conditions (n = 23).

2.2. Materials and procedure

2.2.1. Baseline mood and hormone assessment

All data collection occurred during a 2 h session between 1130 h and 1930 h from March to June 2011. Following eligibility screening, participants were told that they were participating in a study investigating hormones and social interaction. They then completed a 28-item baseline mood adjective checklist (Nowlis, 1965). Seven items assessing angry affect were selected for analysis on an a priori basis (i.e., *angry, hostile, scornful, grouchy, annoyed, offended, irritable*; α = .90). Participants rated the extent to which they were currently experiencing each emotion descriptor on 7-point scales (1 = not at all; 7 = very much). Participants also reported how long they had been awake, which was used as a covariate in subsequent analyses. In order to bring arousal levels to baseline, participants then viewed a 30-min excerpt of a Discovery Channel *Planet Earth* nature video. They then provided two saliva samples for testosterone and cortisol. Salivettes were used to obtain samples for cortisol and a separate passive drool sample was taken for testosterone.

2.2.2. Provocation procedure

Participants were given 10 min to prepare a 2-min speech based on talking points provided by the experimenter (e.g., life goals), which they would later present via a bogus web conference to a participant ostensibly in the laboratory down the hall. In reality, the web conference

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was prerecorded. To make this deception more realistic, the experimenter began the web conference with a series of simple instructions for a female actor, timed to ensure that the instructions given by the experimenter and the responses from the actor were coordinated. The experimenter then instructed the participant and the actor not to interrupt each other during the speeches, which helped ensure that the participant did not discover the deception. The actor always spoke first for 2 min, followed by the actual participant's 2 min speech. Participants were then told that they were to evaluate their partner's speech and vice versa via a single online chat message. All participants were then provoked through insulting feedback ostensibly written by the fictitious participant: ''Your speech was kinda boring to be honest...your life goals seemed vague and unrealistic. I got the impression from your speech that you probably haven't thought this through as much as most students by this stage. Overall a pretty disappointing speech coming from a uni student... It felt like a waste of my time listening to you.'' This provocation procedure reliably increases anger, aggression, and blood pressure (Denson et al., 2010, 2011; Memedovic et al., 2010).

2.2.3. Aggressive behavior and win/lose manipulation

Participants were told that they would be engaging in a competitive reaction time task, in which they could deliver blasts of white noise to the same participant who insulted them. They completed a modernized 25-trial version of the Taylor (1967) Aggression Paradigm (TAP; Bushman, 1995; Denson et al., 2010, 2011). Participants were told that in order to win the reaction time task, they had to be the fastest to click the mouse when the color of a small box on the screen changed from yellow to red. Participants selected numerical values on separate scales from 0 to 10 for both the *intensity* and the *duration* of the white noise blast to be delivered to the fictitious participant. The 0 value served as a non-aggressive option (0 db intensity, 0 s duration), whereas the remaining scale points had decibel and time values next to them (intensity ranged from 1 = 60 db to 10 = 105 db; duration ranged from 1 = 0.5 s to 10 = 2.0 s). Although the procedure involves 25 trials, the first trial provides the best measure of reactive aggression as participants had been insulted by the fictitious participant but had not yet received a noise blast from the bogus participant (Bushman and Baumeister, 1998; Konijn et al., 2007). The remaining 24 trials consisted of 3 blocks of 8 trials each, during which the noise blasts from the bogus participant steadily increased in loudness and duration (i.e., low, medium, and high). After the first trial, participants tend to retaliate by selecting matching levels of loudness and duration (Bushman, 1995). Because intensity and duration were strongly correlated, $r = .84$, $p < .001$, aggression was operationalized as the composite of intensity and duration scores (0–10) selected by participants on the first trial.

The trials were programmed such that participants in the *lose* condition lost 19 out of 25 trials, whereas those in the *win* condition won 19 out of 25 trials. Upon completion of the reaction time task, the screen read ''Congratulations you WON 19 out of 25 trials'' or ''Sorry you LOST 19 out of 25 trials''. The participant then had to notify the experimenter that they had finished the game. The experimenter noticed the participants' scores and reinforced the win or loss, respectively, by remarking ''I've never seen a score that high'' or ''I've never seen a score that low''.

2.2.4. Post-test measures and hormone assessment

Following the aggression paradigm, participants completed measures of state dominance (e.g., ''I feel that I am better than the other student'', $\alpha = .85$); state submissiveness (''The other student is more skilled than me'', $\alpha = .85$); and a second mood adjective checklist (angry affect items' $\alpha = .88$). The dominance and submission items were rated on 7-point scales (1 = strongly disagree; 7 = strongly agree; see Appendix A). The items ''I feel like a winner'' and ''I feel like a loser'' were also utilized to check the effectiveness of the win/lose manipulation. Participants provided saliva samples 10 min (time 2) and 30 min (time 3) following completion of the aggression paradigm, respectively.

2.3. Hormone assays

Samples were stored in a −20 °C freezer until study completion. They were then assayed by a professional reference laboratory at the University of Dresden, Germany, for cortisol and free testosterone. Sampling tubes were centrifuged for 5 min, and hormone concentrations were measured by commercially available chemiluminescence-immuno-assays with high sensitivity (IBL International, Hamburg, Germany). Intra and interassay coefficients of variations were below 10%.

2.4. Statistical analyses

As a manipulation check, between-group ANOVAs tested the efficacy of the win/lose manipulation. Repeated measures ANOVAs tested change in affect due to the provocation. Testosterone and cortisol concentrations were natural log-transformed to normalize the distributions. Hierarchical regression analyses were conducted for each dependent variable (aggression, dominance, submissiveness) in which the mean-centered testosterone and cortisol values were entered at the first block along with two covariates (i.e., time awake and days since last menstrual cycle). At the second block, the testosterone × cortisol interaction term was entered into the model. In the presence of a significant interaction, post hoc probing was conducted to ascertain the nature of the interaction (Aiken and West, 1991). Low and high values of the hormone concentrations were operationalized as ± 1 *SD* from the sample mean. Our low and values for testosterone were 11.05 and 38.52 pg/ml, respectively. Low and high values for cortisol were 4.03 and 10.95 nmol/l, respectively. Thus, our low and high values were within the normal range for freely cycling women for the assays used in the present research (IBL International, Hamburg, Germany). To determine whether testosterone and cortisol concentrations were influenced by the win/lose manipulation, between-group (win versus lose) ANCOVAs were conducted at times 2 and 3 while controlling for time awake, menstrual cycle phase, and baseline cortisol and testosterone. Three participants did not report the number of days since last menstrual cycle and one did not report how long they had been awake. Thus, these four participants were excluded from the hormone analyses (although including them did not alter the significance of the interactions).

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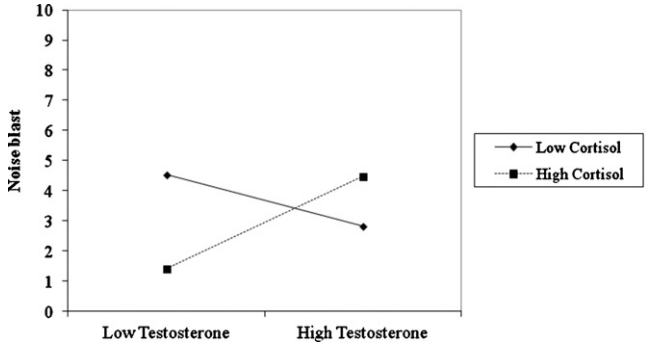

Figure 1 Interaction between basal testosterone and cortisol on reactive aggression in response to an insult. Testosterone was positively related to aggressive retaliation only at high levels of cortisol.

3. Results

3.1. Manipulation checks

Participants reported an increase in angry affect from baseline suggesting an effective social provocation procedure (M_{pre} = 1.72, SD = 0.96; M_{post} = 2.74, SD = 1.38), $F(1,48) = 29.09$, $p < .001$, $\eta^2 = .38$.[1] Participants in the win condition endorsed the item "I feel like a winner" (M = 4.80, SD = 1.42) to a greater extent than those in the lose condition (M = 2.83, SD = 1.50), $F(1,51) = 23.93$, $p < .001$, $\eta^2 = .32$. Conversely, participants in the lose condition (M = 3.61, SD = 1.85) endorsed the time "I feel like a loser" to a greater extent than those in the win condition (M = 2.17, SD = 1.26), $F(1,51) = 11.35$, $p = .001$, $\eta^2 = .18$.

Testosterone and cortisol did not interact to predict anger, nor were there any significant zero-order correlations between hormone concentrations and any of the dependent measures.

3.2. Aggressive behavior

Regression analyses revealed a basal testosterone × cortisol interaction, $b = 3.84$, $t(43) = 2.93$, $p = .005$, $R^2_{adjusted} = .28$ (Fig. 1). Simple slope analyses revealed that there was a significant positive relationship between testosterone and aggression at high levels of cortisol, $b = 2.68$, $t(43) = 2.87$, $p = .006$, but not at mean levels of cortisol, $b = 0.60$, $t(43) = 0.98$, $p = .34$, or at low levels of cortisol, $b = -1.48$, $t(43) = -1.57$, $p = .12$.

3.3. State dominance and submission

Regression analyses also revealed a testosterone × cortisol interaction for self-reported feelings of dominance following the win/lose manipulation, $b = 1.94$, $t(43) = 2.86$, $p = .006$, $R^2 = .17$ (Fig. 2, top panel). Simple slope analyses revealed that there was a significant positive relationship between testosterone and dominance at high levels of cortisol, $b = 1.05$, $t(43) = 2.16$, $p = .036$, and no relationship at mean levels of cortisol, $b = -0.01$, $t(43) = -0.02$, $p = .99$, or at low levels of cortisol, $b = -1.06$, $t(43) = -2.17$, $p = .36$. For state submission, analyses also revealed a marginally significant testosterone × cortisol interaction, $b = -1.32$, $t(43) = -1.83$, $p = .07$, $R^2 = .09$ (Fig. 2, bottom panel). Simple slopes analyses revealed that there was a positive relationship between testosterone and submissiveness at low levels of cortisol, $b = 1.01$, $t(43) = 1.94$, $p = .06$, but not at mean levels of cortisol, $b = 0.29$, $t(43) = 0.85$, $p = .40$, or at high levels of cortisol, $b = -0.43$, $t(43) = -0.83$, $p = .41$. Experimental condition did not interact with these variables suggesting that these relationships remained reliable regardless of whether participants won or lost the competitive aggression paradigm, although there were main effects of condition on both state dominance (M_{win} = 4.58, SD = 1.16; M_{lose} = 3.47, SD = 0.96), $F(1,51) = 13.56$, $p = .001$, $\eta^2 = .21$), and submission (M_{win} = 2.79, SD = 1.11; M_{lose} = 3.79, SD = 1.14), $F(1,51) = 10.26$, $p = .002$, $\eta^2 = .17$).[2,3]

3.4. Hormone responses to the win—lose manipulation

The ANCOVAs revealed a significant main effect of condition on testosterone at time 2, $F(1,40) = 5.11$, $p = .029$, $\eta^2 = .11$, such that participants in the win condition had higher

[1] Due to computer error during the questionnaire portion of the study, anger data from four participants were missing.

[2] We also deconstructed the testosterone × cortisol interactions in the opposite manner to examine the effect of cortisol on the dependent measures at low (−1 SD), mean, and high (+1 SD) values of testosterone. These analyses revealed an effect of cortisol at low levels of testosterone, $b = -2.88$, $t(43) = -2.96$, $p = .005$, but not at mean levels, $b = -.068$, $t(43) = -1.11$, $p = .28$, or high levels of testosterone, $b = 1.52$, $t(43) = 1.57$, $p = .12$. For state dominance, there was a marginally significant effect of cortisol on state dominance at low levels of testosterone, $b = -1.08$, $t(43) = -1.88$, $p = .07$. There was a significant effect of cortisol at high levels of testosterone, $b = 1.14$, $t(43) = 2.41$, $p = .02$, but not at mean levels of testosterone, $b = 0.03$, $t(43) = 0.08$, $p = .94$. There. There were no effects of cortisol on state submissiveness at any level of testosterone, $ps > .12$.

[3] We conducted additional analyses controlling for body mass index. The interactions remained significant (aggression: $\beta = .35$, $p = .023$; state dominance, $\beta = .43$, $p = .009$; state submission, $\beta = -.37$, $p = .026$).

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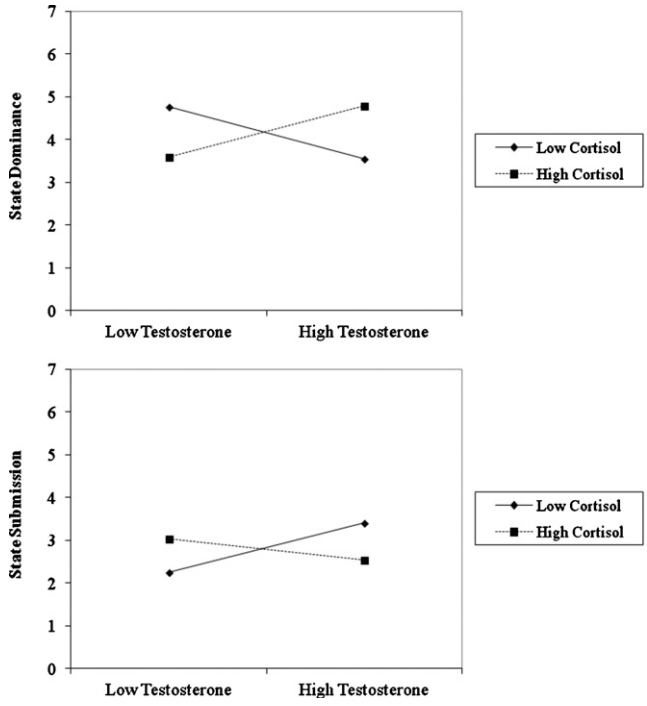

Figure 2 Interaction between basal testosterone and cortisol on feelings of dominance and submission following a competitive aggression paradigm that participants were randomly assigned to win or lose.

concentrations of testosterone ($M_{untransformed}$ = 32.42, *SD* = 20.08) than participants in the lose condition ($M_{untransformed}$ = 21.32, *SD* = 10.73) (see Fig. 3). The win—lose manipulation did not influence testosterone at time 3 or cortisol at any time point. There were no baseline testosterone × cortisol interactions or testosterone × cortisol × condition interactions on hormones at times 2 and 3.

4. Discussion

This study documented the first reversal of the dual-hormone hypothesis. In response to social provocation, basal testosterone positively predicted increased reactive aggression among women with high concentrations of basal cortisol, but not among women with average or low concentrations of

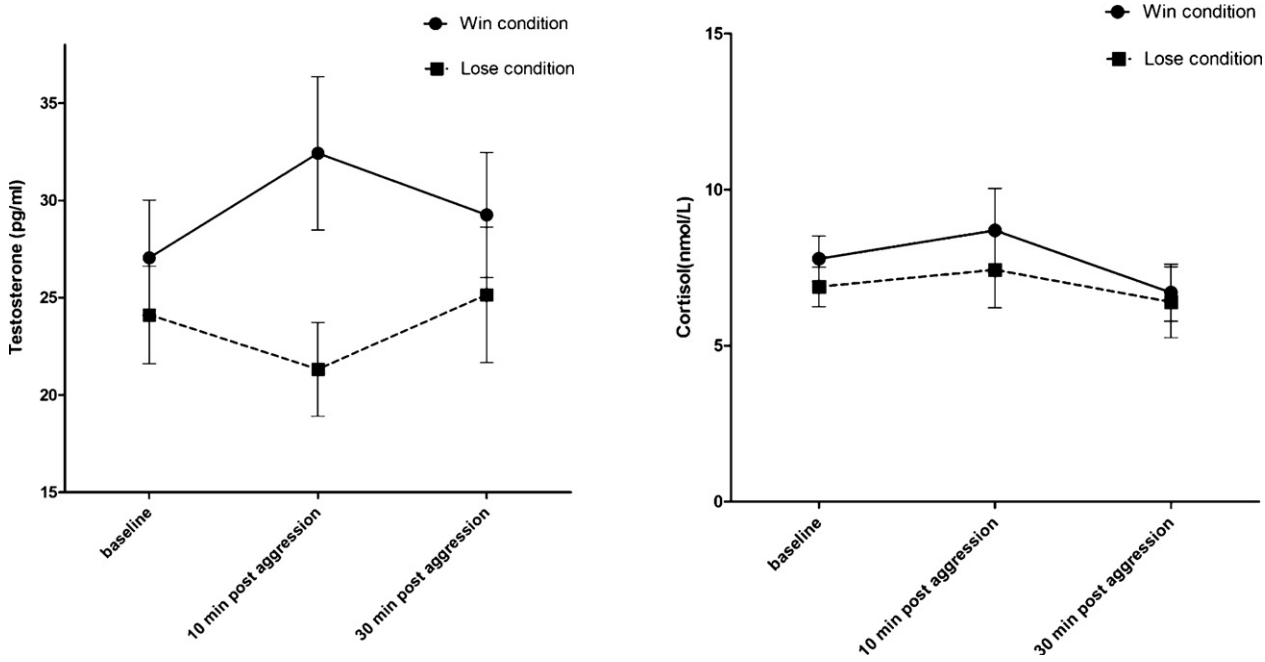

Figure 3 Testosterone (left panel) and cortisol (right panel) concentrations and standard errors over the duration of the experiment as a function of whether participants were randomly assigned to win or lose an aggressive competition. Hormone values are untransformed.

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cortisol. This is in contrast to past work with male violent offenders and adolescent delinquents showing that testosterone positively correlated with reported aggressiveness when cortisol was low (Dabbs et al., 1991; Popma et al., 2007). These differences between our findings and the two studies with men may be due to sample differences in gender, age, or student versus offender status. However, it is noteworthy that our findings in undergraduate women are strikingly consistent with a study of undergraduate men (Geniole et al., 2011). The common thread between our study and the Geniole et al. study is the presence of a potent social provocation (e.g., being social excluded in the case of Geniole et al., and being personally insulted in the present study). Thus, these data suggest that when provoked, testosterone may influence aggression at high rather than low levels of cortisol. However, we caution that this conclusion should remain tentative until research with a no-provocation condition is conducted.

One possible reason for these patterns of data is that high levels of cortisol may indicate hypersensitivity to socially threatening situations (Dickerson and Kemeny, 2004; Denson et al., 2009). For instance, one placebo-controlled experiment in which cortisol was administered to healthy young men and women undergraduates found an aggression-augmenting effect of cortisol in women but not men (Böhnke et al., 2010). Moreover, in an fMRI study, amygdala activity to viewing emotional images was greater among participants high in endogenous cortisol than those low in cortisol (van Stegeren et al., 2007). When combined with high levels of testosterone, this increased emotional reactivity among individuals high in cortisol may increase the likelihood of aggressive retaliation. Nonetheless, the interactive relationship between cortisol and testosterone on anger and aggression remains complex. For instance, one fMRI study, in which male undergraduates were induced to control their anger in response to a provocation, found the standard dual-hormone effect. Specifically, testosterone predicted increased neural reactivity in cortical and subcortical structures implicated in emotion, arousal, and cognitive control among participants with low levels of cortisol (Denson et al., in press). Additional neuroimaging research found increased cortical and subcortical neural activation among men high in testosterone and low in cortisol (Hermans et al., 2008). Thus, more research with diverse samples, manipulations of social contexts known to increase aggression, and actual measures of aggressive behavior are needed to clarify when and how cortisol and testosterone jointly influence aggression.

Self-reported feelings of submissiveness and dominance showed the same pattern as the aggression data, suggesting that when cortisol was high, testosterone was positively related to feelings of heightened dominance and lower submissiveness. In prior research, men showed the reverse pattern such that testosterone was positively correlated with dominance at low levels of cortisol (Mehta and Josephs, 2010). Although Geniole et al.'s (2011) study of men found similar results to our study of women, the present findings may reflect complex interactions between gender roles, cortisol, and testosterone. Dominant and aggressive behavior is expected from men, but not from women. Thus, in men, high concentrations of cortisol may reduce dominant behavior. By contrast, high concentrations of cortisol in women may be associated with heightened stress reactivity and negative emotionality, which could increase reactive aggression.

In addition to the dual-hormone reversal, our data provide evidence showing that women's testosterone levels were higher following winning an aggressive competition than losing. Cortisol concentrations did not change after the aggressive competition. The heightened testosterone after winning is consistent with Mazur's (1985) biosocial theory of status. Our data extend this theoretical position to women within a competitive, aggressive context.

The present study was limited in to women. However, women commit some forms of aggression such as intimate partner violence at rates nearly equal to men (Archer, 2000; Ehrensaft et al., 2004). By identifying hormonal risk for perpetrating reactive aggression, the present study may help inform evidence-based treatments for aggression and violence. Although much more research needs to be done, therapeutic interventions that take individual differences in hormone concentrations and social context into account may eventually contribute to the reduction of aggression. Such interventions could improve the lives of perpetrators and those around them.

Funding body agreements and policies

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Contributors

Thomas F. Denson: study conceptualization, statistical analyses, writing. Pranjal H. Mehta: study conceptualization, writing. Daniela Ho Tan: data collection, statistical analyses, writing.

Conflicts of interest

The authors declare no conflicts of interest.

Appendix A

State Dominance Scale I feel that I am better than the other student. I usually win at games like this. I am more skilled than the other student. I feel like a winner. If given the time reaction task again I would beat the other student.- State Submission Scale I feel that the other student was just better than me. I don't tend to win a lot. The other student is more skilled than me. I feel like a loser. I don't think I could beat the other student if given the time reaction task again.

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