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Endogenous testosterone and cortisol jointly influence reactive aggression in women

Thomas F. Denson [a,*], Pranjal H. Mehta [b], Daniela Ho Tan [a]

[a] School of Psychology, University of New South Wales, Sydney, New South Wales 2052, Australia
[b] Department of Psychology, University of Oregon, Eugene, OR 97403, USA

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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,

* Corresponding author. Tel.: +61 2 93851305; fax: +61 2 93853641.
E-mail address: t.denson@unsw.edu.au (T.F. Denson).

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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;