



Testosterone, cortisol, and psychopathic traits in men and women



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HIGHLIGHTS

- The relationships between testosterone cortisol and psychopathy were examined.
- Testosterone was positively related to psychopathy.
- Cortisol was positively related to psychopathy in men.
- Cortisol moderated the relationship between testosterone and psychopathy in men.

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ABSTRACT

Cortisol and testosterone are theorized to independently and jointly influence antisocial behaviors. The current research examined the independent and interactive effects of baseline testosterone and cortisol on individual differences in psychopathic traits in a relatively large non-clinical sample ($N = 237$). Participants completed the Self-Report Psychopathy – Short Form (SRP; Paulhus, Neumann, & Hare, in press) and provided saliva samples. Analyses indicated that testosterone and cortisol were positively correlated with psychopathic traits in men, but beyond these effects, cortisol moderated the relationship between testosterone and psychopathy in men. The relationship between testosterone and psychopathy within men was positive when cortisol levels were high, but negative when cortisol levels were low. These results have implications for work surrounding the dual hormone hypothesis and suggest that nonclinical variability in psychopathy can be predicted by baseline testosterone and cortisol.

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1. Introduction

An abundance of literature shows that psychopathic traits are linked to many undesirable characteristics and antisocial behaviors (see [18,22,23,31,62,66] for reviews). Individuals high in psychopathy show increased instrumental aggression [11], coupled with decreased startle responses to distressful stimuli [3,47,48], low anxiety [61], fearlessness (e.g. [32]), lower autonomic nervous system activity [5,17,29,30] and decreased amygdala reactivity to fearful faces [7].

1.1. Psychopathy and neuroendocrine function

Testosterone and cortisol are two hormones associated with traits conceptually linked to psychopathy. Testosterone, the end-product of

the hypothalamic–pituitary–gonadal (HPG) axis, is positively correlated with antisocial behaviors including problems in work settings, criminal activity, risky behaviors, and drug use [35]. Additionally, testosterone is positively correlated with reward-seeking [13], has a small, positive association with aggression (see [2] for a meta-analysis). Low empathy, another prominent feature of psychopathy, is also linked to higher levels of endogenous testosterone [54] and exogenous administration of testosterone reduces empathic behavior [25,59]. Despite support for a relationship between testosterone and traits conceptually linked to psychopathy, only two studies have directly assessed the association between testosterone and psychopathic traits. In a relatively small sample of criminal offenders ($n = 61$), Stalenheim, Eriksson, von Knorring, & Wide [57] reported a positive correlation between testosterone and the antisocial facet of psychopathy. In contrast, a larger non-clinical sample revealed no bivariate relationships between baseline testosterone concentrations and psychopathy [20].

Cortisol, the end-product of the hypothalamic–pituitary–adrenal (HPA) axis, is positively related to fear, sensitivity to punishment, and withdrawal behavior [56]. Notably, psychopathic traits are characterized by deficits in these very same processes [32] and with individual

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differences in cortisol secretion (see [56] for review). Specifically, psychopathic criminals have lower diurnal levels of cortisol compared to nonpsychopathic criminals [9] and within psychopathic samples, cortisol levels are negatively correlated with aggression [36]. Men scoring high in psychopathy also exhibit lower cortisol reactivity to a standard psychosocial stressor, relative to men scoring low on psychopathic traits [45,46].

This literature suggests that testosterone and cortisol may contribute to normal variability in psychopathic traits. However, testosterone and cortisol do not have exclusively independent relations with psychopathy and antisocial behaviors. The HPA and HPG axes are not closed systems, but are thought to have mutually inhibitory effects on each other [60] and may interact and co-regulate social behavior [8,58]. Specifically, when cortisol levels are low, high testosterone levels are associated with dominance [37], overt aggression [51], perceived status in women [15], and violent crime [12]. One exception to this pattern of findings is a recent report that testosterone levels were positively correlated with reactive aggression in women when cortisol levels are high [14].

Terburg et al. [58] hypothesized that psychopathy would be more pronounced in individuals with high testosterone and low cortisol levels. Glenn et al. [20] reported that although baseline testosterone and cortisol did not predict psychopathy, the ratio of baseline testosterone to cortisol reactivity (the testosterone/cortisol ratio) positively predicted individual differences in psychopathic traits when testosterone levels were high. Glenn et al. [20] concluded that the HPA and HPG axes work simultaneously to predispose individuals toward psychopathy. Although Glenn et al. [20] did not find a significant testosterone \times cortisol moderation effect, it is important to note that Glenn et al. did not examine whether the testosterone \times cortisol effect was specific to men or women. Although work surrounding the dual-hormone hypothesis is developing, testosterone \times cortisol interactions in women and men have been linked to dominance and aggression [12,14,38,51]. Testosterone and cortisol interactions related to aggression and antisocial behaviors have been found in men and women, but in different directions. Some researchers have found that testosterone positively predicts antisocial behavior in boys and adult men when cortisol levels are low, not high [12,51], while other researchers have found that testosterone predicts aggression in women when cortisol is high [14]. Therefore, it is important to examine whether gender is a moderator of the presence of dual hormone interactions. Considering this work, researchers investigating the neuroendocrine correlates of psychopathic traits (or antisocial behavior) should consider both testosterone and cortisol levels, since these systems appear to work collectively in modulating antisocial behavior [8].

1.2. Overview of current research

Few studies have systematically examined associations between testosterone and cortisol levels and specific dimensions of psychopathy. Although some studies have examined the relationship between testosterone and psychopathy, few of these studies have utilized larger sample sizes (but see [20]), which can enhance generalizability and power in statistical analyses. Therefore, it is necessary to test the relationship between testosterone, cortisol, and psychopathy in a larger sample of men and women. Furthermore, the construct of psychopathy appears to be dimensional in nature [22] and studies of large non-clinical samples not only assist in generalizability but also avoid the potential confounds (e.g., substance abuse) that may affect the results from incarcerated samples (e.g., [22]). The current research overcomes the limitations of previous research by examining the extent to which baseline testosterone, cortisol, and the interaction of testosterone and cortisol map onto variability in psychopathic traits in a large non-clinical sample.

Based on our review of the previous literature, we hypothesized that testosterone would be positively related to psychopathy in men. Consistent with dual hormone research on the joint effects of testosterone and cortisol (e.g., [38]), we examined whether testosterone and cortisol

would interact to affect psychopathy. Because previous research has found dual-hormone interactions occurring in different directions, we did not form any hypotheses about how testosterone and cortisol would jointly affect psychopathy. Due to the absence of research showing that baseline cortisol and psychopathy are related in nonclinical populations, we did not form any hypotheses about the relationship between basal cortisol and psychopathy.

2. Methods

2.1. Participants

The participants were 237 undergraduate students (51.9% women, *M* age = 21.73, *SD* = 4.66) who received course credit and ten dollars as compensation. This sample size, using the effect size metric of Pearson's *r* and a two tailed alpha of .05 [10] is capable of estimating large and medium effect sizes (*r* = .5 and .3, respectively) with excellent power (power > .99), and small effect sizes (*r* = .1) with low power (power = .34). The current study was part of a larger protocol examining relationships between hormones, personality traits, and social behavior (see [6,7]). None of the analyses in the current manuscript overlap with our previously published data.

2.2. Materials and procedure

To increase the purity of saliva samples, participants were instructed not to eat or brush their teeth at least 2 h prior to arrival in the lab. Participants completed the Self Report Psychopathy – Short Form (SRP-SF; [49]), a 29-item measure of psychopathy assessing the Interpersonal, Affective, Lifestyle, and Antisocial factors of psychopathy. The SRP-SF has a four-factor latent structure [6,7,34,40,41,65], and is strongly positively correlated with both the Hare Psychopathy Checklist – Revised and the Youth Psychopathic Traits Inventory [1,41,49], as well as with a psychopathy self-report based on the five-factor model of personality [33]. Although often within nonclinical ranges, the continuously distributed traits measured by the SRP-SF are associated with relevant external correlates, such as positively with criminal offenses and externalizing psychopathology [41], negatively with moral reasoning [55], negatively with amygdala activation to fearful faces [6,7], and positively associated with aggression and alcohol use [63]. On-going latent variable model-based research with the SRP has shown it to be invariant across sex, based on a mega world-sample (30 k+), and that such traits are associated with world regional data such as Gross Domestic Product (GDP), fertility, and infant mortality [42]. Recent work using the SRP-SF has found that general population scores on the SRP-SF have a mean of 26.23 (*SD* = 7.07; [55]). Additionally, psychopathic offenders have a mean SRP-SF score of approximately 77 [49], and those who meet the cutoff for psychopathy of the Psychopathy checklist – Revised (PCL-R) have a mean SRP-SF score of 98 [49]. Although only 16 of our participants had psychopathy scores that exceeded 77 and no scores exceeded 98, even relatively low levels of SRP-SF scores have meaningful associations with violence, alcohol use, and intelligence, among other variables [22].

A confirmatory factor analysis (CFA) using MPLUS was conducted on the SRP-SF items using the four-factor model via robust weighted least squares estimation method, with all items loading only on their respective factors. The 4-factor model showed acceptable fit ($\chi^2(344) = 728.25, p < .001, CFI = .91, RMSEA = .07$), and a delta-chi squared test revealed that this model had significantly better fit than the two-factor model of psychopathy ($\Delta\chi^2(5) = 78.47, p < .001$). The two factor model contained the Interpersonal-Affective factor and Lifestyle-Antisocial factor and did not demonstrate acceptable fit ($\chi^2(349) = 806.73, p < .001, CFI = .89, RMSEA = .08$). This four factor model of psychopathy is presented in Fig. 1, which displays standardized coefficients for all estimates. As can be seen in Fig. 1, the factors are strongly inter-correlated and thus provide evidence that the total SRP-SF score can be treated as a unidimensional scale [43]. The

Hare Self Report-Psychopathy-Short Form (SRP-SF) Scale Four-factor 28-item model (N = 236)

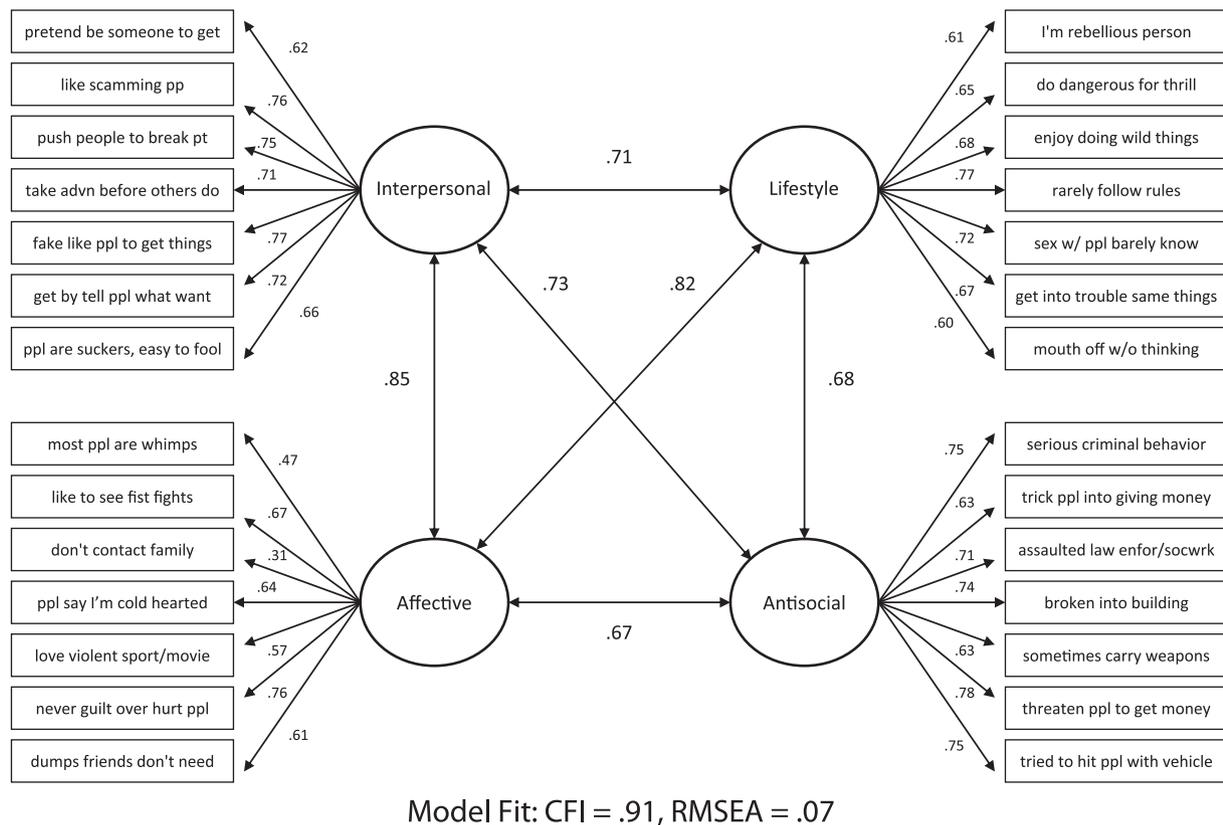


Fig. 1. Path model for confirmatory factor analysis of the Self Report Psychopathy – Short Form. Circles represent the four latent factors of psychopathy, while rectangles represent the scale item indicators. Double arrow lines between the latent factors indicate correlated error variances of the latent factors, while single arrow lines between the indicators and latent factors represent factor solution. All coefficients are standardized.

Cronbach's alphas for the four factors indicated acceptable reliabilities (alphas .62–.80).

2.2.1. Saliva samples

After completing self-report questionnaires (which included the SRP-SF), participants provided a baseline saliva sample via unstimulated passive drool into polystyrene culture tubes. All samples were stored at -20°C until assayed using commercially-available enzyme immunoassay kits (DRG International). All samples were assayed in duplicate and the average of the two samples was used in all analyses. All testing took place between 11 am and 5 pm to control for diurnal variation in testosterone and cortisol concentrations.

Mean intra-assay coefficients of variation for testosterone and cortisol were 11.70% and 10.01%, respectively. Four participants provided insufficient saliva for processing testosterone. Also, because we performed the cortisol assay after the testosterone assay, cortisol concentrations from 50 participants were unable to be determined because of insufficient volume.¹

¹ We also asked participants to indicate if they used oral contraceptives, the number of cigarettes they smoked per week, number of alcoholic beverages consumed weekly, average hours of sleep per night, and the amount of hours slept in the night prior to the study. These measures were not correlated with our measures of testosterone and cortisol, with two exceptions: Alcohol consumption had a small, positive association with Testosterone (standardized separately in men and women; $r = .18, p = .007$), and cigarette use had a small, negative association with cortisol ($r = -.15, p = .037$). Controlling for alcohol and cigarette use did not change the significance of any presented analyses, with the exception that the main effect of cortisol in our first regression model (Table 1) became marginally significant ($p = .059$). However, these two covariates were not useful covariates in any steps of the regression model ($ps \geq .717$).

3. Results

Cortisol concentrations were positively skewed and thus were log transformed for all analyses. Since men and women differed in testosterone levels, testosterone was standardized separately for men and women for models with interactions [28,37–39,44,68]. Three univariate outliers were found for testosterone concentrations and one univariate outlier was found for cortisol (>3 SDs). These values were winsorized to 3 SDs [64]. Preliminary analyses indicated that relationships between hormones and psychopathic traits were similar across each of the four psychopathy facets. Specifically, testosterone showed a small, positive effect sizes across all four facets in men (rs from .05 to .19, significant only for the interpersonal dimension, $p = .045$) and small, null effects in the four facets in women (rs from $-.04$ to .08). Cortisol had small to moderate positive effect sizes with the four facets in men (rs from .17 to .34; ps from $-.001$ to .090) and null effects in women (rs from $-.08$ to .13). Thus, all analyses were conducted on total psychopathy scores.

Although cortisol levels (log transformed) did not differ between men ($M = .50, SD = .17$) and women ($M = .51, SD = .15, t(185) = -.515, p = .607, d = .08$), men had higher testosterone levels ($M = 92.35, SD = 34.84$) compared to women ($M = 41.09, SD = 20.59, t(231) = 13.82, p < .001, d = 1.82$). Also, men scored higher in psychopathy ($M = 61.99, SD = 12.98$) compared to women ($M = 49.87, SD = 12.83, t(234) = 7.22, p < .001, d = .94$). Because testosterone and cortisol are affected by diurnal variation (e.g. [50]), we used time of day as a covariate in all analyses. In our study, time of day was negatively correlated with cortisol concentrations in women ($r(91) = -.26, p = .013$), but not men ($r(96) = -.10, p = .36$). Time of day was negatively correlated with testosterone concentrations

in men ($r(111) = -.24, p = .011$), but not in women ($r(122) = -.03, p = .72$).

We proceeded to test our hypotheses using a four-step set of moderated multiple regression models. Because these models contained interactions, we mean-centered the values of testosterone and cortisol prior to computing interactions. Interaction effects were interpreted using the conditional effects probing tool developed by Preacher, Curran, and Bauer [52]. In the first step, we regressed total psychopathy scores on testosterone concentrations (centered, standardized separately for men and women) and cortisol concentrations (centered, log transformed), with gender and time of day as covariates. In the second step, we examined if testosterone and cortisol jointly interacted to affect psychopathy by adding a testosterone \times cortisol interaction term. The third model tested whether sex moderated the relationship between testosterone and cortisol by adding a testosterone \times sex interaction term and a cortisol \times sex interaction term. Finally, the fourth model tested whether sex moderated the interactive effect of testosterone \times cortisol on psychopathy with a three-way sex \times cortisol \times testosterone interaction term. The results of this 4-model regression are presented in Table 1.

3.1. Effects of baseline testosterone and cortisol on psychopathy

Model 1 revealed that the relationship between testosterone (standardized separately for men and women) and psychopathy was marginally significant in a positive direction ($\beta = .12, p = .088$) and the relationship between cortisol and psychopathy was positive and significant ($\beta = .13, p = .049$). Because we had substantially less saliva to measure cortisol ($N = 187$) than testosterone ($N = 233$), this resulted in less statistical power to estimate the effects of testosterone on psychopathy than what is afforded by our sample in step one. To attenuate this loss of power, we examined a similar model to step 1 without cortisol included as a predictor, finding that testosterone was a significant

positive predictor of psychopathy ($\beta = .15, t(181) = 2.24, p = .026$) when controlling for gender and time of day.

3.2. Testosterone \times cortisol moderation

Consistent with Glenn et al. [20], model 2 did not reveal a significant testosterone \times cortisol interaction across all participants, $\beta = .06, p = .40$. We return to investigate whether testosterone \times cortisol interactions specifically occurred in men or women in model 4.

3.3. Gender moderation of the prediction of psychopathy by testosterone and cortisol

Model 3 did not reveal a significant sex \times testosterone interaction, $\beta = .05, p = .49$,² but did yield a significant sex \times cortisol interaction, $\beta = -.15, p = .031$. Probing this two-way interaction with simple slopes analysis revealed that cortisol was a positive predictor of psychopathy within men ($b = 21.54, se = 8.07, t(177) = 2.67, p = .008$), but not women ($b = -5.42, se = 9.77, t(177) = -.56, p = .58$).

3.4. Gender moderation of dual hormone effects on psychopathy

Model 4 revealed that the entered sex \times cortisol \times testosterone interaction was significant ($\beta = -.15, p = .038$), suggesting that a cortisol \times testosterone interaction was specific to either men or women. Simple slopes analysis procedures revealed that there was a significant conditional testosterone \times cortisol interaction for men ($b = 19.48, se = 8.42, t(176) = 2.31, p = .022$), but not for women ($b = -5.31, se = 8.31, t(176) = -.64, p = .53$). These conditional interactions are presented in Fig. 2. The conditional testosterone \times cortisol within men was significant because the direction of the relationship between testosterone and psychopathy was positive when cortisol levels were high (1 SD above the mean; $b = 2.99, se = .165, t(176) = 1.81, p = .072$), but negative when cortisol levels were low (1 SD below the mean; $b = -3.17, se = 2.43, t(176) = -1.31, p = .19$). The presence of the two-way testosterone \times cortisol conditional interaction indicates that these two slopes were significantly different from each other.³ Examining the four facets individually, the three-way testosterone \times cortisol \times sex interaction effect was significant in the lifestyle and antisocial facets ($ps \leq .043$), marginally significant in the interpersonal facet ($p = .081$), and nonsignificant in the affective facet ($p = .485$).

Table 1

Multiple regression analyses with testosterone, cortisol, and sex predicting total psychopathy scores.

Model and predictors	β	t	p
Model 1	$F(4,180) = 15.29, p < .001, R^2 = .25$		
Testosterone	0.12	1.72	0.088
Cortisol	0.13	1.98	0.049
Time	-0.08	1.25	0.213
Sex	-0.45	-6.93	<.001
Model 2	$F(5,179) = 12.35, p < .001, R^2 = .26$		
Testosterone	0.10	1.45	0.149
Cortisol	0.12	1.68	0.095
Time	-0.08	-1.26	0.208
Sex	-0.45	-6.90	<.001
T \times C	0.06	0.84	0.403
Model 3	$F(7,177) = 9.64, p < .001, R^2 = .28$		
Testosterone	0.10	1.39	0.166
Cortisol	0.09	1.25	0.214
Time	-0.10	-1.49	0.139
Sex	-0.44	-6.84	<.001
T \times C	0.08	1.16	0.248
Sex \times C	-0.15	-2.17	0.031
Sex \times T	0.05	0.70	0.49
Model 4	$F(8,176) = 9.14, p < .001, R^2 = .29$		
Testosterone	0.08	1.12	0.263
Cortisol	0.11	1.50	0.135
Time	-0.11	-1.67	0.097
Sex	-0.41	-6.24	<.001
T \times C	0.08	1.20	0.232
Sex \times C	-0.11	-1.53	0.129
Sex \times T	0.08	1.20	0.233
Sex \times C \times T	-0.15	-2.09	0.038

4. Discussion

The present study examined the joint effects of cortisol, testosterone, and sex on psychopathy in a relatively large, well-powered, nonclinical sample of both men and women. Additionally, this study examined these effects using the largest sample of women to date. Consistent with previous research [57], the present study found a weak, positive relationship between testosterone and psychopathy across men and women. Within men, cortisol was found to be positively related to psychopathy. Additionally, cortisol moderated the relationship between testosterone and psychopathy in men, such that testosterone was positively related to psychopathy when cortisol was high, but negatively related to psychopathy when

² At the request of an anonymous reviewer, we also evaluated the simple slopes of testosterone predicting psychopathy in men and women without cortisol or interactions relevant to cortisol in the model. Simple slopes analysis revealed a marginally significant trend in men ($b = 2.21, se = 1.33, t(227) = 1.66, p = .098$), but no effects of testosterone in psychopathy in women ($b = .60, se = 1.20, t(227) = .50, p = .616$). The testosterone \times sex interaction in this model was not significant ($p = .371$).

³ We also used the Johnson–Neyman technique for probing interactions to probe the Testosterone \times Cortisol Interaction within men. This analysis showed that the conditional slopes of testosterone predicting psychopathy were significantly different when centered log-transformed cortisol reached 1.12 SDs above the mean.

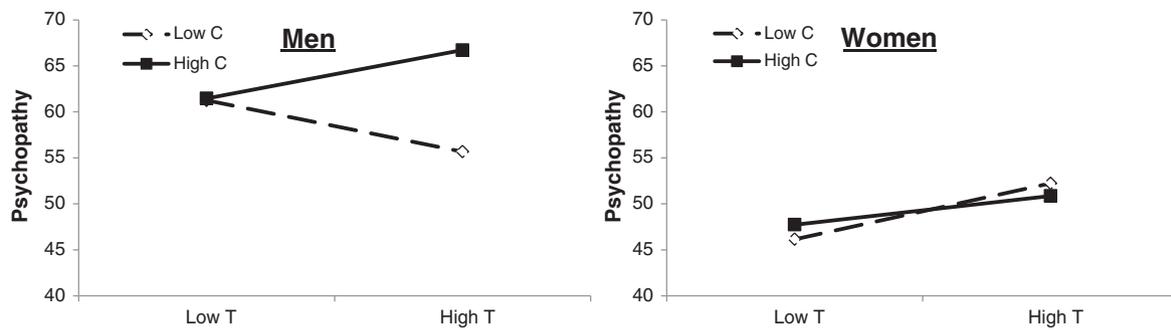


Fig. 2. Psychopathy as a function of testosterone and cortisol within men and women.

cortisol was low. These findings are consistent with recent work and theory suggesting that testosterone and cortisol jointly regulate dominance and antisocial behaviors [8,12,14,20,38,51,58]. Most of these studies indicate that testosterone predicts dominance and behaviors, but only within individuals with low cortisol levels [12,38,51].

However, the current research shows that testosterone is positively related to psychopathy in men when cortisol levels are high. On the other hand, the least psychopathic individuals had low levels of testosterone and cortisol. Although Glenn et al. [20] did not find a significant testosterone \times cortisol interaction in participants, Glenn et al. did not examine whether testosterone \times cortisol interactions occurred specifically in men or women. Although this study suggests a reversal of the dual hormone hypothesis, it is not the first study to show this reversed effect (see [14]). Denson et al. reasoned that this reversal of these effects occurred because high cortisol individuals show increased reactive aggression in response to provocation. This reasoning is consistent with the findings of Geniole, Carré, and McCormick [19], who found that socially excluded men with both high basal testosterone and high basal cortisol show the highest levels of reactive aggression. Since undergraduates show more reactive aggression than instrumental aggression (Falkenbach, Poythress, Creevy, [69]), cortisol may be positively associated with psychopathy in nonclinical samples, but negatively associated with psychopathy in clinical samples (as found by [9,36]). However, other studies have failed to find a relationship between cortisol and psychopathy [16,21]. Notably, both of the studies showing negative associations with psychopathy and cortisol include samples with relatively high levels of psychopathy (i.e. clinical sample, prison sample) compared to nonclinical samples.

The finding that psychopathy was associated with high testosterone and high cortisol in men is consistent with a recent report that adolescent males with high levels of psychopathy have a higher coupling of both testosterone and cortisol [27]. Both this finding and the current research suggest that the mutually-inhibitory effects of the HPA and HPG axes (e.g. [60]) may not occur when males have higher levels of psychopathy. More research is needed to investigate whether individual differences, including psychopathy, modulate cross-talk between the HPA and HPG axes.

The current findings may help to provide further bridging of the interface between psychopathy and testosterone, given that both psychopathy and aggression have several similar neurological correlates. Psychopathy has been linked to alterations in the functioning of the amygdala, ventral striatum, and ventromedial prefrontal cortex [4]. Relatedly, testosterone has been associated with increased amygdala reactivity to angry faces [26], decreased orbital frontal cortex responses to social provocation [37] and heightened ventral striatum reactivity to reward [24]. Carré et al. [6,7] also found that amygdala reactivity to fearful faces was negatively associated to the interpersonal facet of the SRP,

whereas amygdala reactivity to angry facial expression was positively associated with the lifestyle facet of the SRP.

This work is not without limitations. Due to the correlation nature of the study, it is not possible to infer whether testosterone and cortisol can increase psychopathy, are associated with psychopathy, or are by-products of psychopathy. Future experimental research is needed to determine the causal direction of this relationship. Given that pharmacological manipulations of testosterone have found to reduce empathetic behavior [25] generosity [67], and increase amygdala and hypothalamic reactivity to social threats [26], it may be possible that testosterone is also causal agent in heightened psychopathy.

The nonclinical sample used in our study was adequate for testing variability in normal population levels of psychopathic traits. However, because investigating individuals with clinical psychopathy is useful for applied clinical purposes, future research will benefit from assessing the extent to which testosterone and cortisol predict psychopathy dimensions, empathic functioning, and other correlates of psychopathy in a wide variety of samples. Our sample did not contain many individuals with clinically-significant levels of psychopathy. Examining differences between clinical psychopaths and non-psychopaths could potentially reveal more robust effects of testosterone and cortisol interactions. Inclusion of clinical level psychopaths with the population may also reveal a nonlinear relationship between cortisol and psychopathy. In this study, baseline testosterone and cortisol were related to variability in psychopathic traits in men. Building on our findings and those of others, future research is needed to further elucidate the interaction of testosterone and cortisol to co-regulate antisocial behaviors and psychopathy, as well as establish mediating neural and empathic mechanisms of the effects of testosterone and cortisol on psychopathy [68].

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