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An exploratory analysis of testosterone, cortisol, and aggressive behavior type in men and women

Todd Armstrong [a,*], Jessica Wells [b], Danielle L. Boisvert [c], Richard H. Lewis [d], Eric M. Cooke [c], Matthias Woeckener [c], Nicholas Kavish [e]

[a] School of Criminology and Criminal Justice, University of Nebraska Omaha, 6001 Dodge Street, Omaha, NE, 68182, United States
[b] Department of Criminal Justice, Boise State University, Boise, ID, 83725, United States
[c] Department of Criminal Justice and Criminology, Sam Houston State University, Huntsville, TX, 77341, United States
[d] Department of Criminal Justice, University of Arkansas at Little Rock, Little Rock, AR, 72204, United States
[e] Department of Psychology and Philosophy, Sam Houston State University, Huntsville, TX, 77341, United States

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ABSTRACT

Increasing evidence indicates that the interaction between testosterone and cortisol is associated with variation in aggressive behavior. However, results are mixed. The current study further explored the association between testosterone, cortisol, and both reactive and proactive aggression in a large sample of university students. Models considered direct and interactive effects between baseline measures of testosterone and cortisol as well as change in hormones in response to a social stressor. In women, baseline cortisol had a negative direct association with reactive aggression and was further associated with reactive aggression in interaction with baseline testosterone (positive interaction). Hormones were unrelated to reactive aggression in men. Baseline cortisol had a negative direct association with proactive aggression in women. In contrast, the association between change in cortisol and proactive aggression was positive. Cortisol was not associated with proactive aggression in men. In addition, testosterone was not related to proactive aggression either directly or in interaction with cortisol in either men or women. Collectively, these results show that the association between hormones and aggression varies across aggressive behavior type and across sex.

1. Introduction

Growing evidence indicates testosterone and cortisol play a role in the etiology of aggressive behavior. However, the specific nature of the association between testosterone, cortisol, and aggression remains at issue. Testosterone, an androgenic steroid hormone governed by the hypothalamus-pituitary-gonadal (HPG) axis, has long been held to play a role in aggression as a corollary of its more direct association with dominance behavior and competition (Archer, 2006; Carré & Olmstead, 2015; Eisenegger, Haushofer, & Fehr, 2011; Mazur & Booth, 1998). Testosterone has been directly implicated in dominant status driven behavior "to obtain power, influence or valued prerogatives over a conspecific" (Mazur & Booth, 1998, p. 353). This class of behaviors arguably includes some aggressive acts which are commonly defined as acts directed at another and carried out with the intent to cause harm (Anderson & Bushman, 2002; p. 28). Testosterone may be related to aggressive behaviors through genomic and contemporaneous effects on brain structures in the social behavior network (Newman, 1999) that have been implicated in the etiology of aggression (Nelson & Trainor, 2007).

A contemporaneous association between testosterone and aggression may be modulated by increased attention to social threat resulting in increased social threat approach. The potential role of testosterone in aggression in response to threat was originally highlighted by Henry and Stephens (1977). Contemporary research shows that the positive association between testosterone and increased attention to social threat is well established (for a review see Geniole & Carré, 2018). Studies have also shown that exogenous testosterone is positively associated with activity in structures that play a role in the neural processing of social threat (e.g. attention to angry faces) including the amygdala and orbitofrontal cortex (Goetz et al., 2014; Hermans, Ramsey, & van Honk, 2008; Radke et al., 2015), and may serve to decrease connectivity between subcortical and cortical brain areas (Peper, van den Heuvel, Mandl, Pol, & van Honk, 2011). Importantly, covariance between the

* Corresponding author.
E-mail address: toddarmstrong@unomaha.edu (T. Armstrong).

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amygdala and medial prefrontal cortex has been shown to mediate the association between baseline testosterone and aggression, such that significance of the association between testosterone and aggression decreases when amygdala-prefrontal covariance is accounted for (Nguyen et al., 2016). Recently, a number of studies have begun to show that the relationship between exogenous testosterone and social threat may result in greater risk for aggression through increased threat approach and decreased social distance in the context of social threat (Enter, Spinhoven, & Roelofs, 2016; Radke et al., 2015; Wagels, Radke, Goerlich, Habel, & Votinov, 2017).

Testosterone's role in aggression through increased approach to social threat may be supported by an association between testosterone and reward salience/risk tolerance (Eisenegger et al., 2011; Welker, Gruber, & Mehta, 2015; Wood, 2004). Testosterone has a positive association with activity in mesolimbic areas associated with the representation of reward value in decision making including the ventral striatum and nucleus accumbens (Braams, van Duijvenvoorde, Peper, & Crone, 2015; Hermans et al., 2010; Laube, Lorenz, & van den Bos, 2020; Op de Macks et al., 2011; White, Lee, Schlund, Shirtcliff, & Ladouceur, 2020). In line with this evidence, a recent meta-analysis found a small positive correlation ($r(9,112) = .12$) between testosterone and constructs associated with increased risk preference including sensation seeking, impulsivity and risk taking (Kurath & Mata, 2018).

Despite the associations between testosterone and aggression implied in the lines of research reviewed above, meta-analyses of the testosterone-aggression relationship tend to find modest effects (Archer, Graham-Kevan, & Davies, 2005; Book & Quinsey, 2005; Book, Starzyk, & Quinsey, 2001). In a recent contribution to this line of research, Geniole et al. (2019) found that the association between change in testosterone and aggression among men ($r(994) = .162$, $p < .05$) was stronger than the association between baseline testosterone and aggression ($r(12,986) = .071$, $p < .05$). The same pattern was evident among women, however both associations lacked statistical significance (change in testosterone and aggression, $r(469) = .010$, $p = .851$; baseline testosterone and aggression $r(2212) = .002$, $p = .936$).

The weak association between testosterone and aggression has led to speculation that cortisol may modulate the effect of testosterone on aggressive behaviors. Cortisol is a glucocorticoid hormone product of the hypothalamus-pituitary-adrenal (HPA) axis stress response. Cortisol release is part of a broader set of psychological, physiological and behavioral responses to threats to the social self (Dickerson & Kemeny, 2004). Cortisol has a number of physiological effects as part of its role in the stress system including the mobilization of energy, immune and cardiovascular responses (Buckingham, 2006; Sapolsky, Romero, & Munck, 2000). In addition to these physiological effects, there is evidence that cortisol has both acute and more protracted effects on neurological activity in response to stress (Joëls, 2018). A recent review of studies considering associations between cortisol and brain activity found cortisol during and after stress was associated with increased activity in the perigenual anterior cingulate cortex, decreased activity in the ventromedial prefrontal cortex, and altered function (both increases and decreases) in the amygdala and hippocampus (Harrewijn et al., 2020). Collectively, these changes may support increased attention and vigilance as well as a shift away from higher cognitive function towards areas associated with habitual behavioral strategies (Joëls, 2018).

Work on the cognitive effects of cortisol suggests baseline cortisol, cortisol response, and exogenous cortisol are positively associated with fear, punishment sensitivity, anxiety and social avoidance (Brown et al., 1996; Roelofs et al., 2009; Schulkin, Morgan, & Rosen, 2005; Shields, Bonner, & Moons, 2015; van Honk, Schutter, Hermans, & Putman, 2003). Well-developed lines of research also show baseline cortisol and cortisol in response to stress are positively associated with attention to social threat and behavioral inhibition (Roelofs, Bakvis, Hermans, van Pelt, & van Honk, 2007; Roelofs, Elzinga, & Rotteveel, 2005; Roelofs et al., 2009; Tops & Boksem, 2011; van Honk et al., 1998, 2000, 2003). The positive association between cortisol and attention to social threat coupled with increased behavioral inhibition, punishment sensitivity, fear and anxiety suggests a negative relationship between cortisol and aggressive behaviors. However, the pattern of association between cortisol and aggression is somewhat mixed. The majority of evidence indicates a negative association between cortisol and aggressive or antisocial behavior (van Goozen, Fairchild, Snoek, & Harold, 2007). However, there are null findings and significant results that are opposite general trends (Gerra et al., 1997; Kruesi, Schmidt, Donnelly, Hibbs, & Hamburger, 1989; McBurnett et al., 2005; Schulz, Halperin, Newcorn, Sharma, & Gabriel, 1997; van Bokhoven et al., 2005).

Mixed results and weak associations across studies relating cortisol and testosterone individually to aggression may be explained by the interactive effects of these hormones on aggressive behaviors. The dual hormone hypothesis (DHH) suggests that the association between testosterone and dominance behavior including forms of aggression is moderated by cortisol (DHH; Mehta & Josephs, 2010; Mehta & Prasad, 2015). In the DHH, cortisol is thought to exert a countervailing influence on increases in dominance behavior with testosterone so that dominance behaviors occur more frequently when testosterone levels are high and cortisol levels are low. In the context of studies evaluating interactions between testosterone and cortisol the DHH predicts a negative interaction where testosterone is positively associated with aggression when cortisol is low. The interactive effects of testosterone and cortisol on traits and behaviors are suggested by significant cross-talk between the hypothalamus-pituitary-gonadal HPG- and hypothalamus-pituitary-adrenal HPA-axes (Viau, 2002). Research indicates HPA-axis stress response products dampen HPG-axis function (Johnson, Kamilaris, Chrousos, & Gold, 1992). For example, the secretion of gonadotropins is inhibited by corticotropin releasing hormone (Tilbrook, Turner, & Clarke, 2000) and glucocorticoids such as cortisol are associated with decreased androgen receptor synthesis (Mehta & Prasad, 2015; Smith, Syms, Nag, Lerner, & Norris, 1985). Additional evidence indicates that the HPA-axis is inhibited by testosterone at the level of the hypothalamus (Williamson & Viau, 2008) and the adrenal gland (Rubinow et al., 2005).

While research is generally supportive of the DHH, a recent meta-analysis found the magnitude of association between the interaction of testosterone and cortisol and a range of measures potentially implicated in status seeking was statistically significant but small ($r = -.061$, $p = .026$; Dekkers et al., 2019). Specific to aggression, several studies confirm an association between aggression and hormones that is consistent with the DHH, but there are also null findings, and evidence of associations in the opposite direction. Dabbs, Jurkovic, and Frady (1991) reported violent crimes were more common among participants with high basal testosterone and low baseline cortisol in a sample of 113 male adolescent offenders, while Grotzinger et al. (2018) found increases in self-reported aggression with baseline testosterone were stronger at low levels of baseline cortisol in a large community sample of adolescents ($N = 984$).

Other studies provide partial support for the interactive effects of testosterone and cortisol on aggression. Popma et al. (2007) found the interaction of baseline testosterone with baseline cortisol was negatively associated with self-reports of overt aggression, but not covert aggression. Tackett, Herzhoff, Harden, Page-Gould, and Josephs (2014) found a negative association between youth and parent reports of externalizing behaviors and the interaction of baseline testosterone with baseline cortisol only emerged in three-way interactions including either disagreeableness or emotional instability. Platje et al. (2015) found the ratio of baseline testosterone to baseline cortisol had a statistically significant positive association with a self-report measure of aggressive behavior, but the interaction between testosterone and cortisol did not. There are also a number of studies that find the interaction of cortisol and testosterone are not related to aggressive behaviors including laboratory, self-report, and clinician rating measures (Buades-Rotger et al., 2016; Korpel, Varkevisser, Hoppenbrouwers, Van Honk, & Geuze, 2019; Mazur & Booth, 2014; Scerbo & Kolko, 1994). In addition to null

findings, studies have also found evidence of associations opposite of the direction predicted by the DHH. Here, aggression is increased when both testosterone and cortisol are high. For example, Denson, Mehta, and Tan (2013), found that among a small sample (N = 53) of undergraduate women increases in testosterone were associated with increased reactive aggression in response to an insult at high levels of cortisol. Geniole, Carré, and McCormick (2011) reported a directionally similar but not statistically significant association between baseline testosterone, cortisol and reactive aggressive behavior in the Point Subtraction Aggression Paradigm (N = 78).

Differences across findings regarding the association between testosterone, cortisol and aggression can be organized through a consideration of differences between reactive and proactive aggression. Dominance behaviors seem to have considerable overlap with the description of proactive aggression as "cold-blooded" and instrumental/goal directed aggression that is responsive to reinforcement (Dodge, 1991; Raine et al., 2006). In contrast, reactive aggressive behavior is "hot-blooded" aggression in response to real or perceived provocation or threat and is usually accompanied by increased emotional and physiological arousal (Vitaro, Brendgen, & Barker, 2006). The distinction between reactive and proactive aggression is not without controversy. However, evidence shows that reactive and proactive aggression have different physiological, developmental, and psychological correlates (Armstrong et al., 2019; Card & Little, 2006; Cima & Raine, 2009; Hubbard, McAuliffe, Morrow, & Romano, 2010). In work that may be particularly relevant to the issue at hand, studies have found baseline cortisol and cortisol reactivity are positively associated with reactive aggression, but not with proactive aggression (Lopez-Duran, Olson, Hajal, Felt, & Vazquez, 2009; van Bokhoven et al., 2005).

1.1. The current study

Here we further investigate the association between testosterone, cortisol, and self-report aggression using a sample of undergraduate university students. This relatively large sample allows us to conduct analyses separately within women and men. In addition, we also consider interactions between baseline hormone measures, interaction between measures of change in hormones in response to stress, and interactions between baseline measures and change measures. Attention to sex differences and change in hormones in response to stress are important given recent meta-analytical findings that the association between testosterone varies across sex and across baseline hormones measures and measures of change in hormones (Geniole et al., 2019). Analyses within sex are also supported by the organizational-activational model of hormone-based sex difference in brain and behavior (Phoenix, Goy, Gerall, & Young, 1959; Schulz, Molenda-Figueria, & Sisk, 2009). This model specifies both hormone driven sex differences in neurological development and sex differences in the contemporaneous influence of hormones on brain function and behavior. The current work also builds on prior work through the use of the Reactive Proactive Aggression Questionnaire (RPQ; Raine et al., 2006) to capture distinct forms of aggressive behavior. The RPQ is a widely used measure of reactive and proactive aggression with well-established reliability and validity including associations with violent criminal behavior and independent ratings of aggression (Cima, Raine, Meesters, & Popma, 2013; Fossati et al., 2009; Raine et al., 2006; Tuvblad, Dhamija, Berntsen, Raine, & Liu, 2016).

Given the limited scope of work relating both testosterone and cortisol to aggressive behavior type and the varying results within this work, we consider the current study exploratory. However, based on prior research we anticipate testosterone will have a positive direct association with aggressive behaviors, while cortisol will have a negative direct association. Direct associations between cortisol and aggressive behavior may vary somewhat across aggressive behavior type. The preponderance of research indicates a negative association between cortisol and antisocial and aggressive behaviors, but some work has shown a positive association between cortisol and reactive aggression (Lopez-Duran et al., 2009; van Bokhoven et al., 2005). Regarding the interaction of testosterone and cortisol, there is reason to anticipate a negative association between the interaction of testosterone with cortisol and aggressive behavior with increased aggressive behavior occurring at high levels of testosterone and low cortisol (Grotzinger et al., 2018; Popma et al., 2007; Tackett et al., 2014). Partial overlap between proactive aggression and dominance behaviors may suggest that this negative association extends to proactive aggression. However, studies specific to reactive aggression point to a positive association between the interaction of testosterone with cortisol and aggressive behavior where reactive aggressive behavior increases with cortisol behavior when testosterone is high (Denson et al., 2013; Geniole et al., 2011).

2. Methods

2.1. Participants

After Institutional Review Board approval, study participants were recruited from classes at a university in the southern United States. In each class, a research team member provided a brief overview of the study and requested student participation. The research team member also emphasized that participation was voluntary and confidential. Some participants received extra credit at the discretion of the class instructor. After the provision of written informed consent, 862 participants completed a questionnaire and were referred to a laboratory data collection protocol where hormone measures were gathered. Of the 862 participants that completed the in-class survey, 567 participated in the laboratory measurement protocol. Of these, 10 declined to provide saliva samples for analysis and 4 did not complete the protocol. Analyses also omitted data from a single participant who identified as a transgender woman. The final analysis sample included 366 women and 186 men. Participants were an average of 20.34 years of age (SD = 3.05) and self-identified as 13.2 % African American, 37.1 % Caucasian, 39.2 % Hispanic, and 10.5 % other. Sample demographic characteristics within sex are presented in Table 1.

2.2. Measures

Measures included hormone assays capturing response to a social stressor and a questionnaire measuring aggressive behavior.

2.2.1. Hormone assays

Testosterone and cortisol samples were collected via passive drool using Salimetrics LLC Saliva Collection Aids. All samples were stored in a −20 °C freezer until analysis. Samples were analyzed at the university that provided IRB approval via testosterone and cortisol enzyme immunoassay kits of passive saliva samples (Salimetrics LLC). Each sample contained 1.5 mL of saliva or greater and were analyzed in duplicate following Salimetrics assay kit protocol. Analyses were based on the mean concentrations of the two assays. The mean intra-assay coefficient of variation for testosterone was 5.98 %, while the mean intra-assay coefficient for cortisol was 11.10 %. The value for cortisol is high relative to established standards but is similar to values reported in some published work in this area (see for example, Welker, Lozoya, Campbell, Neumann, & Carré, 2014). The inter-assay coefficients of variation for testosterone and cortisol were 7.95 % and 5.91 % respectively. Mean hormone concentrations are presented in Table 1.

2.2.2. Questionnaire measures

Aggressive behaviors were measured with the RPQ. The RPQ includes 11 items capturing reactive aggression and 12 items capturing proactive aggression. Items measuring reactive aggression including "gotten angry when others threatened you" and "yelled at others when they have annoyed you" addressed aggression in response to real or

Table 1
Descriptive Statistics and Tests of Sex Differences for Analyses Variables.

	Women (n = 366)		Men (n = 186)		Test for sex differences		
Demographics					Chi-Square	df	p
% African American	14.3 %		10.2 %		4.80	6	.569
% Caucasian	35.1 %		42.0 %				
% Hispanic	40.0 %		36.9 %				
% Other	10.60 %		10.90 %				
	M	SD	M	SD	t	df	p
Age	20.35	3.39	20.33	2.26	−.070	541	.944
Physiological Measures							
Testosterone Baseline	46.78	21.23	124.09	60.00	17.04	209	.000
Testosterone Post-test	41.58	21.36	115.95	56.35	17.38	212	.000
Testosterone Change	−5.54	11.85	−8.17	34.91	−1.00	207	.194
Cortisol Baseline	.22	.17	.22	.16	−.02	550	.983
Cortisol Post-test	.21	.16	.25	.21	2.00	297	.068
Cortisol Change	−.01	.22	.03	.25	2.01	334	.045
Aggressive Behavior Type							
RA	6.29	3.74	5.98	3.86	−.89	524	.375
PA	.76	1.43	1.32	2.32	2.90	242	.004

Note: Testosterone concentrations are pg/mL and cortisol concentrations are μg/dL. RA = raw reactive aggression scores, PA = raw proactive aggression scores; Cortisol values are for the non-transformed cortisol concentrations; Non-integer degrees of freedom are from corrections when equal variances cannot be assumed.

perceived provocation. Proactive aggression items including "used physical force to get others to do what you want" and "had fights with others to show who was on top" measured instrumental and goal directed aggression. The reliability and validity of the RPQ have been established among children, adolescents, and adults and across nationality/ethnic group (Cima et al., 2013; Fossati et al., 2009; Raine et al., 2006; Tuvblad et al., 2016). Specifically, the RPQ has shown good test-retest stability across two months in high school students (Fossati et al., 2009; reactive aggression $r(347) = .72$; proactive aggression $r(347) = .75$) and across one and two year intervals in childhood arrestees (Cima et al., 2013; $ICC(324) > .041$). In the current sample, reactive and proactive aggression scales had adequate internal consistency (Cronbach's alpha reactive aggression = .83, proactive aggression = .77).

2.3. Procedure

Participants in the laboratory measurement protocol reported to the lab between the hours of 0800 and 1830. The prolonged measurement period was necessary to accommodate the large number of study participants, but introduces variation in hormones due to diurnal variation in both cortisol and testosterone (Dabbs, 1990; Pruessner et al., 1997). To reduce the impact of diurnal variation on results, all analyses linking hormones to aggressive behavior included time of day as a covariate. In addition, models yielding statistically significant associations between hormones and aggressive behavior measures were replicated with the sample restricted to those who participated in the laboratory protocol after 12 pm.

Prior to reporting to the lab, participants were instructed to refrain from a variety of activities that may affect testosterone and cortisol levels for at least one hour (e.g., smoking, eating, exercise). Baseline saliva samples were gathered after participants had been comfortably seated for approximately 30 s. Participants were then instructed that they had two minutes to prepare and deliver a two-minute speech that addressed their biggest faults and weaknesses. Participants were instructed that this speech would be recorded and later analyzed. To increase stress, the two-minute speech was recorded via digital camera recording device. During the speech, the researcher remained in the room with the participant but was concealed behind a partition. Participants were instructed to halt their speech if they attempted to continue past two minutes. Alternatively, if the speech concluded before the two minutes were up, recording continued until the full two minutes passed. Post-stress saliva samples were gathered approximately 15 min after the conclusion of the recording of the speech ($M = 22.26$; $SD = 2.18$). The time between initiation of the stressor is consistent with the time between onset of stress and peak cortisol response (Dickerson & Kemeny, 2004).

2.4. Data analysis strategy

Analyses were based on data collected from participants recruited for a convenience sample drawn from classes at a university in the southern United States. Analyses were conducted separately for women and men in light of evidence for sex differences in the association between hormones and aggressive behavior and evidence for large differences between women and men in testosterone. In the current sample, independent samples *t*-tests showed baseline testosterone concentrations differed significantly for women and men (women $M = 46.78$, $SD = 21.23$; men $M = 124.09$, $SD = 60.00$; $t(209) = 17.04$, $p < .001$). Therefore, transformations to induce normality in distributions, as well as the identification and rescoring of outliers, were also done separated for women and men.

Cortisol concentrations were log 10 transformed to account for skewness. Log transformations induce normality in the distribution of a variable by accounting for different distances between scale values. After log transformation there was one univariate (female) outlier in baseline cortisol scores and two univariate outliers in post-stress cortisol scores (one female and one male). Hormone outliers were windsorized to three standard deviations from the mean (Wilcox, 2010). Testosterone concentrations did not exhibit significant skewness and were not transformed. At baseline, there were 2 univariate outliers among testosterone scores for women and one univariate outlier among testosterone scores for men. At post-stress there were 2 univariate outliers among scores for both women and men.

Preliminary analysis compared hormone levels and aggressive behaviors across sex with independent samples *t*-tests. Following this, change in hormone concentrations from baseline (Time 1) to post-test (Time 2) was assessed with paired samples *t*-tests. Next, bivariate correlations (Pearson's *r*) between hormones and aggressive behavior measures were estimated. Multivariate associations between hormones and aggressive behaviors were then tested with regression models with

multiple imputation for missing data in Stata 15 (StataCorp, 2017).[1] Inference in multiple imputation was based on average associations across ten imputed data sets with 100 iterations between draws. Imputations were based on analyses variables.

Measures of hormones and aggressive behaviors were standardized for regression models. Initial regression models considered the simultaneous direct association between Time 1 (baseline) measures of testosterone and cortisol and aggressive behavior type, net of controls for time of day, age of the respondent, and dummy variables for the two largest race groups in the sample: Caucasian (1 = Caucasian and 0 = other) and Hispanic (1 = Hispanic and 0 = other). Age and race were included as covariates due to work showing these characteristics are associated with variation in aggressive behaviors (Coie & Dodge, 1998). Time of day was represented as an integer corresponding to the hour of the day when data collection began and two values after the decimal point indicating the fraction of minutes past the hour. A subsequent regression model added the interaction term between the hormone measures. This set of models was repeated for hormone change scores and for models including baseline measures and change scores (baseline cortisol and change in testosterone, and baseline testosterone and change in cortisol). This results in four sets of models within aggressive behavior type within sex. Significant interactions were probed using simple slopes analysis (Bauer, Preacher, & Curran, 2007). Simple slopes analysis of statistically significant interactions were framed by direct associations between hormones and aggressive behavior measures. If either testosterone or cortisol had a direct association with a given aggressive behavior measure, we considered the association between that hormone and aggressive behavior at different levels of the other hormone (−1 *SD*, +1 *SD*).

Sensitivity analyses were conducted for all regression models yielding statistically significant results. Sensitivity analyses included re-estimating regression models with the sample restricted to those who began the laboratory protocol after 12 pm and re-estimating regression models without multiple imputation for missing data. The estimation of models without multiple imputation for missing data also supported the estimation of partial eta-squared as an indicator of effect size. The strength of associations between hormones and aggressive behavior measures were further contextualized with a consideration of the impact of multiple comparisons. In the current study, multiple hormone measures gathered at different points in time, necessitated a large number of comparisons, particularly when analyses were disaggregated by sex and aggression type. A correction for multiple comparisons dividing the traditional threshold for statistical significance by the number of initial comparisons made results in a threshold for statistical significance that is slightly above .001. At this threshold, none of the associations in the current work were statistically significant. In the discussion of results below, we present results at the traditional threshold of statistical significance but also discuss results in the context of the application of a more modest correction for multiple comparisons ($p < .0125$) that accounts for the number of final regression models within aggressive behavior type by sex (four).

3. Results

3.1. Sample characteristics

The sample was largely comprised of young adult University students who were predominantly Caucasian and Hispanic. Information regarding age and race/ethnicity within sex group is presented in Table 1. Sex differences in race/ethnicity were not statistically significant (X^2 (6, $N = 526$) = 4.80, $p = .569$), nor were differences in age (t (542) = −.706, $p = .944$).

3.2. Change in hormones in response to stress

There was a statistically significant decrease in testosterone in response to stress among women ($M_{baseline} = 46.78$, $SD = 21.23$; $M_{post\ stress} = 41.58$, $SD = 21.36$; $t(365) = 6.37$, $p < .001$) and among men ($M_{baseline} = 124.09$, $SD = 60.00$; $M_{post\ stress} = 115.95$, $SD = 56.35$; $t(185) = 3.18$, $p = .002$). Women showed a slight decrease in cortisol in response to stress that was not statistically significant ($M_{baseline} = .22$, $SD = .17$; $M_{post\ stress} = .21$, $SD = .16$; $t(365) = .63$, $p = .538$), while men showed a statistically significant increase in cortisol from pre- to post-test ($M_{baseline} = .22$, $SD = .16$; $M_{post\ stress} = .25$, $SD = .21$; $t(185) = -2.17$, $p = .031$).

3.3. Sex differences in hormones

Means and standard deviations for hormone measures and aggressive behavior type across sex are presented in Table 1. Testosterone concentrations were significantly higher among men than women at both baseline and post-test. There were no sex differences in testosterone change. Baseline cortisol concentrations did not differ across sex. Sex differences in post-test cortisol concentrations showed a trend towards statistical significance, with concentrations higher among men ($p = .068$). Change in cortisol was greater among men than women. Men reported significantly higher proactive aggression. Women and men were not different with regard to reactive aggression. Mean aggressive behavior scores and sex differences in aggressive behaviors reported here are consistent with prior work (Tuvblad et al., 2016).

3.4. Correlations among hormone measures, time of data collection and aggressive behavior

Correlations among hormone measures, time of data collection, and aggressive behavior are presented in Table 2. Correlation coefficients for women are below the diagonal, while coefficients for men are above. Across both women and men, baseline hormone measures were positively correlated with each other and negatively correlated with within hormone change scores (e. g. testosterone negatively correlated with change in testosterone). Baseline cortisol measures were also negatively correlated with change in testosterone across both women and men. The association between baseline testosterone and change in cortisol was negative and approached statistical significance in women ($p = .069$) but was attenuated in men ($p = .131$). Time of sample collection was negatively associated with both baseline testosterone and cortisol among men and women and was positively related to change in testosterone and change in cortisol among both men and women.

Baseline testosterone and change in testosterone were not correlated with any of the aggressive behavior measures. Baseline cortisol was negatively correlated with reactive aggression scores among women, but not men. Change in cortisol was not related to reactive aggression among women or men. All cortisol measures were not related to proactive aggression measures among women or men.

3.5. Multivariate associations between hormones and reactive aggressive behavior

Results from regression models testing the association between hormone measures and reactive aggression in women are presented in Table 3. Model 1 tested the direct association between two hormone measures and reactive aggression net of controls. Model 2 added the interaction between the two hormone measures. Baseline cortisol had a negative direct association with reactive aggression and was further associated with reactive aggression in interaction with both baseline

[1] Of those completing the laboratory protocol with complete hormone data, 5.3% (n=29) were missing a measure of either age or race/ethnicity, 8.3% (n=46) were missing at least one of two the RPQ measures. In more complex models, up to 13.0 % (n=72) of the sample were missing values for at least one variable.

Table 2
Correlations Among Hormone Measures, Time of Data Collection and Aggressive Behavior.

	1	2	3	4	5	6	7
1 Baseline T	–	−.367**	.325**	−.111	−.285**	−.016	−.026
1 Change T	−.294**	–	−.231**	.159*	.182*	.030	.056
1 Baseline C	.300**	−.225**	–	−.385**	−.245**	−.092	−.096
1 Change C	−.095†	.287**	−.404**	–	.146*	−.015	−.112
1 Time	−.111*	.121*	−.363**	.258**	–	.098	.078
1 RA	−.026	−.033	−.111*	.078	.052	–	.548**
1 PA	.051	−.023	−.089	.072	−.080	.521**	–

Notes: Correlation coefficients for women are below the diagonal, while correlation coefficients for men are above. T = testosterone; C = cortisol, Time = time of the initiation of the laboratory data collection protocol, RA = reactive aggression, PA = proactive aggression.
* $p < .05$.
** $p < .01$.
† $p < .010$.

Table 3
The Association between Baseline Hormone Measures and Reactive Aggression in Women (n = 366).

	Model 1			Model 2		
	B	p	95 % CI	B	p	95 % CI
T Baseline	.061	.771	−.352, .474	−.088	.680	−.506, .331
C Baseline	**−.513**	**.026***	**−.963, −.062**	**−.542**	**.018***	**−.990, −.095**
Interaction	–	–	–	**.512**	**.007****	**.138, .886**
T Change	−.240	.269	−.668, .187	−.232	.287	−.660, .196
C Change	.327	.122	−.088, .743	.328	.121	−.088, .745
Interaction	–	–	–	.092	.570	−.227, .412
T Baseline	−.063	.262	−.174, .048	−.072	.199	−.182, .038
C Change	.032	.561	−.077, .142	.043	.449	−.068, .153
Interaction	–	–	–	−.075	.153	−.177, .028
T Change	−.309	.151	−.732, .113	−.191	.382	−.622, .239
C Baseline	**−.574**	**.012***	**−1.024, −.125**	**−.587**	**.010***	**−1.035, −.139**
Interaction	–	–	–	**−.392**	**.042***	**−.769, −.014**

Note: Models also included race dummy variables, age, and time of day (hour and fraction of minutes). * $p < .05$, ** $p < .01$.

testosterone and change in testosterone. The association between the interaction of baseline cortisol with baseline testosterone and reactive aggression was positive, while the association between the interaction of baseline cortisol with change in testosterone was negative. At the revised threshold for statistical significance accounting for multiple comparisons defined earlier ($p < .0125$), baseline cortisol remained associated with reactive aggression when change in testosterone was a covariate. The interaction of baseline cortisol with baseline testosterone also remained statistically significant after correction for multiple comparisons.

Given relatively strong direct associations between cortisol and aggressive behavior measures and no direct association between testosterone and aggression, simple slopes analyses consider the moderation of cortisol's effects on aggression by testosterone. In simple slopes analysis of the positive interaction between baseline cortisol with baseline testosterone among women, baseline cortisol had a negative association with reactive aggression ($B = -3.514$, $p = .001$, 95 % CI [−5.584, −1.445]) when testosterone scores were low (−1*SD*), but baseline cortisol was not associated with reactive aggression ($B = .290$, $p = .777$, 95 % CI [−1.728, −2.309]) when testosterone scores were high (+1*SD*). The association between baseline cortisol, baseline testosterone, and reactive aggression in women is graphed in Fig. 1. Reactive

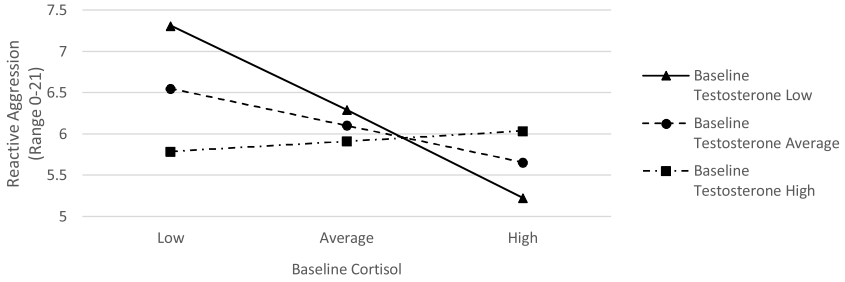

Fig. 1. Reactive Aggression and the Interaction of Baseline Cortisol with Baseline Testosterone Among Women (n = 366).

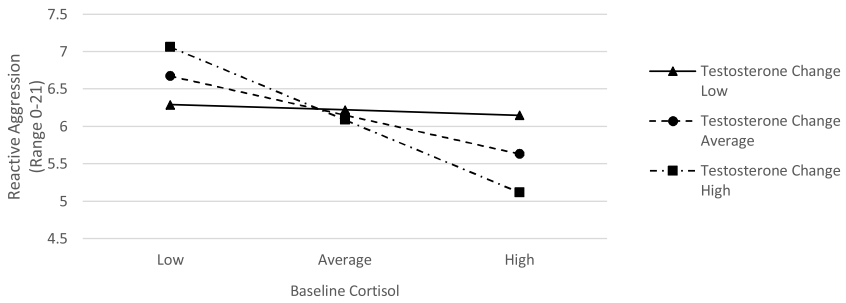

Fig. 2. Reactive Aggression and the Interaction of Cortisol with Testosterone Reactivity in Women (n = 366).

aggression scores are highest when both baseline cortisol and baseline testosterone are low. In contrast, simple slopes analysis of the negative interaction between baseline cortisol and change in testosterone in women found that baseline cortisol had a negative association with reactive aggression when change in testosterone was high ($B = -.211$, $p = .008$, 95 % CI [−.368, −.055]), but not when change in testosterone was low ($B = -.003$, $p = .837$, 95 % CI [−.010, −.008]). The association between baseline cortisol, change in testosterone and reactive aggression in women is graphed in Fig. 2. Here reactive aggression scores are highest when baseline cortisol is low but change in testosterone is high.

Baseline measures and changes scores for both testosterone and cortisol were not related to reactive aggression among men either directly or in interaction. Regression models results for men and for models discussed in text but not tabled are available in the online Supporting Materials for this manuscript.

3.6. Multivariate associations between hormones and proactive aggressive behavior

The results for multivariate regression models considering direct and interactive associations between hormones and aggressive behaviors among women are presented in Table 4. Cortisol was directly associated with proactive aggression, but the direction of this association varied across sex and with the characterization of cortisol (baseline or change). Among women, baseline cortisol had a negative direct association with proactive aggression with baseline testosterone as a covariate (top of Table 4) and with change in testosterone as a covariate (bottom of Table 4). In direct contrast, change in cortisol had a positive direct association with proactive aggression among women in the model with baseline testosterone as a covariate and in the model with change in testosterone as a covariate. At the revised threshold for statistical significance accounting for multiple comparisons ($p < .0125$), baseline cortisol remained negatively associated with proactive aggression when baseline testosterone was included as a covariate. Baseline cortisol was also negatively associated with proactive aggression after correction for multiple comparisons when change in testosterone was included as a covariate, but only prior to the inclusion of the interaction term.

In models testing multivariate associations between hormone measures and proactive aggression among men, the association between change in cortisol and proactive aggression was negative and showed a trend towards statistical significance ($B = -.349$, $p = .057$, 95 % CI [−.709, .011]) with change in testosterone as a covariate. The association between change in cortisol and proactive aggression was again negative with a trend towards statistical significance ($B = -.341$, $p = .062$, 95 % CI [−.699, .017]) when baseline testosterone was included as a covariate. Baseline cortisol was not related to proactive aggression among men. Proactive aggression was not directly associated with either testosterone measures or the interaction of testosterone measures with cortisol measures among both women and men.

3.7. Sensitivity analyses

To examine the implications of the extended data collection window for hormone measures all models yielding statistically significant associations between hormone measures and aggressive behaviors were replicated with the samples restricted to participants who began the laboratory protocol after 12 pm. Additional sensitivity analysis models explored the impact of multiple imputation for missing data by re-estimating models with listwise missing data deletion. Regression models with listwise deletion facilitated the estimation of partial eta-squared as a measure of effect size. Full results for sensitivity analyses models are available in the online Supporting Material for this manuscript. Across all alternative model specifications, the negative association between reactive aggression and the interaction of baseline cortisol with change in testosterone remained statistically significant among women ($p < .05$). Despite this robust statistical association, the effect size for the interaction of baseline cortisol and change in testosterone estimated with the model using listwise deletion was small (partial eta-squared = .013). In contrast, the positive association between reactive aggression and the interaction of baseline cortisol and baseline

Table 4
The Association between Hormone Measures and Proactive Aggression Among Women (n = 366).

	Model 1			Model 2		
	B	p	95 % CI	B	p	95 % CI
T Baseline	.117	.137	−.038, .272	.112	.171	−.049, .273
C Baseline	**−.258**	**.003****	**−.426, −.091**	**−.259**	**.003****	**−.427, −.092**
Interaction	–	–	–	.017	.812	−.123, .157
T Change	−.078	.337	−.237, .081	−.076	.347	−.235, .083
C Change	**.173**	**.033***	**.014, .332**	**.173**	**.033***	**.014, .332**
Interaction	–	–	–	.017	.776	−.103, .138
T Baseline	−.060	.437	−.091, .210	−.049	.529	−.103, .200
C Change	**.154**	**.050***	**.000, .307**	**.166**	**.035***	**.011, .321**
Interaction	–	–	–	−.091	.209	−.233, .051
T Change	−.097	.221	−.254, .059	−.084	.309	−.246, .078
C Baseline	**−.250**	**.003****	**−.416, −.083**	**−.251**	**.003****	**−.418, .084**
Interaction	–	–	–	−.045	.526	−.186, .095

Note: Models also included race dummy variables, age, and time of day. * $p < .05$, ** $p < .01$.

testosterone was attenuated with the sample restricted to participants with hormone measures collected in the afternoon ($B = .524$, $p = .119$, 95 % CI [−.136, 1.185]), but remained statistically significant in replication models with listwise deletion. The effect size for the interaction of baseline cortisol and baseline testosterone in the listwise deletion model was again small (partial eta-squared = .030).

The direct negative association between baseline cortisol and aggression was fairly robust. In models with reactive aggression as the endogenous variable, the direct negative association between baseline cortisol and reactive aggression remained statistically significant among women in 3 of the 4 models. This association was attenuated somewhat in the listwise deletion replication of the model with both baseline hormone measures ($B = -.405$, p = .059, 95 % CI [−.630, .242]). In models with proactive aggression as the endogenous variable the direct association between baseline cortisol and proactive aggression remained negative and statistically significant among women in all replication models. Effect sizes for the negative associations between baseline cortisol measures and aggressive behavior measures were small. Partial eta-squared ranged from .010 to .014 for associations between baseline cortisol and reactive aggression, and from .024 to .026 for the association between baseline cortisol and proactive aggression.

Additional models explored the sensitivity of associations between change in cortisol and proactive aggression that showed a trend towards statistical significance among men. These associations emerged as statistically significant with the sample restricted to men with hormone measures collected in the afternoon ($B = -.679$, $p = .025$, 95 % CI [−1.270, .088]; $B = -.668$, $p = .026$, 95 % CI [−1.254, −.082]), but remained short of the traditional threshold of statistical significance when models were replicated with listwise deletion for missing data ($B = -.330$, $p = .079$, 95 % CI [−.698, .039]; $B = -.319$, $p = .088$, 95 % CI= [−.685, .048]).

4. Discussion

The present study built on prior work by testing associations between testosterone, cortisol, and aggressive behaviors in a relatively large sample, facilitating the estimation of models separately among men and women. The current study also extended prior work by considering differences across aggressive behavior type. Results indicated that reactive aggression was positively associated with the interaction of baseline cortisol with baseline testosterone among women. Simple slopes analyses of the association between reactive aggression and the interaction of baseline cortisol with baseline testosterone revealed that baseline cortisol had a negative association with reactive aggression when testosterone scores were low. The initial strength of the association between reactive aggression and the interaction of baseline cortisol with baseline testosterone survived correction for multiple comparison, but the association was attenuated when the sample was restricted to participants attending data collection in the afternoon. Given comprehensive evidence for diurnal cortisol and testosterone cycles (Dabbs, 1990; Pruessner et al., 1997), this leaves the possibility that the positive association between reactive aggression and the interaction of baseline cortisol with baseline testosterone found among women is an artifact of the data collection. However, Denson et al. (2013) also found a positive association between reactive aggression and the interaction between cortisol and testosterone in women. In this case, simple slopes analysis indicated the positive interaction was a function of increased reactive aggression when both baseline cortisol and testosterone were high.

In the current study, the positive association between reactive aggression and the interaction of baseline cortisol with baseline testosterone was indicated by increased reactive aggressive behavior scores among women with low baseline testosterone and low cortisol (Fig. 1). Here it is possible that decreases in status, prosocial behavioral strategies, and social cooperation occurring at low testosterone (Bird et al., 2019; Casto & Edwards, 2016; Dreher et al., 2016; Edwards & Casto, 2013; Eisenegger, Naef, Snozzi, Heinrichs, & Fehr, 2010; Harris, Rushton, Hampson, & Jackson, 1996; Sherman, Lerner, Josephs, Renshon, & Gross, 2016) compound decreases in fear, punishment sensitivity, and anxiety that are associated with lower levels of cortisol (Brown et al., 1996; Roelofs et al., 2009; Schulkin et al., 2005; Shields et al., 2015; van Honk et al., 2003). Fig. 1 also indicates that at high baseline cortisol it is high testosterone that is associated with increased risk for reactive aggression. Thus, both low baseline cortisol/low baseline testosterone and high baseline cortisol/high baseline testosterone are indicative of increased risk for reactive aggression among women. The suggestion that hypo- and hyper-activity in biological substrates may both be associated with antisocial behavior is not unique (see for example Yildirim & Derksen, 2013), but does complicate theoretical inference. In this case, low testosterone and cortisol at baseline arousal may result in increased risk for reactive aggression among women in the manner outline above, while high baseline testosterone and cortisol result in increased risk for reactive aggression when the increased attention to social threat occurring at higher levels of baseline cortisol (van Honk et al., 1998) are compounded by similar increased attention to social threat occurring at higher levels of testosterone (Geniole & Carré, 2018).

In the current study, baseline cortisol also interacted with change in testosterone to predict reactive aggression among women, but this reaction was negative. Here, reactive aggression was increased when change in testosterone was high and cortisol was low (see Fig. 2). Increases in aggression when change in testosterone is high and cortisol low are somewhat consistent with the association between testosterone, cortisol and dominance behaviors proposed in the DHH (Mehta & Josephs, 2010; Mehta & Prasad, 2015). However, the DHH tends to emphasize associations applicable to baseline hormone levels not change in hormones. It is somewhat surprising that we see reactive aggression scores are highest when cortisol is low and change in testosterone is high, but this association does not extend to the proactive aggressive behaviors that are more closely allied with the status striving dominance that is thought to be the direct results of the interaction of high testosterone and low cortisol according to the DHH. While the theoretical significance of this interaction is somewhat undermined by its relatively weak strength of association in initial analyses (p = .042), it is possible the interaction indicates that the effect on reactive aggression of decreases in fear, punishment sensitivity, and anxiety occurring at lower levels of cortisol (Brown et al., 1996; Roelofs et al., 2009; Schulkin et al., 2005; Shields et al., 2015; van Honk et al., 2003) are increased through increased attention to social threat and reward salience/risk tolerance that have been shown to accompany the administration of exogenous testosterone (Radke et al., 2015; Welker et al., 2015; Wood, 2004).

In addition to conditional evidence for the association between the interaction of testosterone and cortisol and reactive aggression, the current results also provided fairly consistent evidence for a negative association between cortisol and reactive aggression among women. The negative association between baseline cortisol and aggression found herein is consistent with the balance of work relating cortisol to aggressive and antisocial behavior (van Goozen et al., 2007), and may be explained at least in part by decreases in punishment sensitivity and behavior inhibition occurring at low baseline cortisol (Tops & Boksem, 2011; van Honk et al., 2003). However, some prior work has found a positive association between reactive aggression and both baseline cortisol and cortisol reactivity (see Lopez-Duran et al., 2009, and van Bokhoven et al., 2005). Differences between the current results and earlier studies finding a positive association between cortisol and reactive aggression may be due to differences in sample and measurement. Earlier studies showing a positive association between cortisol and reactive aggression both used a subset of teacher-report items from the Aggression Behavior Teacher Checklist to capture reactive and proactive aggressive behaviors in samples of children (Lopez-Duran et al., 2009) and adolescents (van Bokhoven et al., 2005). Inconsistent findings show parsing the association between cortisol and aggressive behavior will

take further research. In future work, it may be worthwhile to pay careful attention to age differences in the association between testosterone and aggression. Both sexes experience a surge in testosterone around the ages of 18/19. Testosterone levels then remain high and don't start to decline until around the age of 30 for men and during menopause for women (Sternbach, 1998).

The current work did find a positive direct association between change in cortisol and proactive aggression among women. This association was statistically significant in analyses with all women and with women who participated in the study during the afternoon, but was attenuated somewhat in sensitivity analyses employing listwise deletion for missing data. The positive association between change in cortisol and proactive aggression is surprising given evidence that greater cortisol responding is associated with increased attention to angry faces and avoidance in social phobia (Roelofs et al., 2005, 2007; Roelofs et al., 2009; van Honk et al., 2000). However, van Honk and Schutter (2007) argue that vigilance for angry facial expression is associated with dominance motives and increased anger, but not anxiety. With increased attention to angry faces characterized in this way, it seems reasonable to suggest that greater change in cortisol may be associated with increased proactive aggression when proactive aggressive behaviors reflect dominance.

Associations between hormones and aggressive behaviors among men in the current study were very limited. A negative association between change in cortisol and proactive aggression in men that approached statistical significance in the main analyses emerged as statistically significant when the sample was restricted to men who participated in the laboratory measurement protocol during the afternoon. The lack of association between hormones and aggressive behaviors among men in the current work is somewhat surprising. However, other studies in this area using community samples have found a similar pattern of results with statistically significant associations between hormones and aggression among females, but not among males (Grotzinger et al., 2018; Platje et al., 2015).

Beyond their implications for the specification of the role of hormones in the etiology of aggressive behaviors, the current results also join studies showing that reactive and proactive aggressive behaviors have distinct physiological correlates (Armstrong et al., 2019; Bobadilla, Wampler, & Taylor, 2012; Hubbard et al., 2002; Murray-Close, Holterman, Breslend, & Sullivan, 2017). While the bulk of research in this area has focused on differences in autonomic nervous system function, the current study suggests that the pattern of association between testosterone, cortisol and proactive aggressive behavior differs from that between testosterone, cortisol and reactive aggressive behavior. These results extend work showing that reactive and proactive aggression have unique socio-environmental, psychological and physiological correlates. However, strong conclusions regarding the precise nature of the association between hormones and both reactive and proactive aggression clearly awaits additional research.

The implications of the current results for the characterization of associations between hormones and aggressive behaviors are conditioned by both the nature of these results and by the methodology of the current study. The current results regarding the association between change in hormones and aggressive behaviors may be specific to the social stressor used in the current study which elicited fairly pronounced decreases in testosterone among both women and men, and increased cortisol in men, but not women. The decreases in testosterone found in the current study in the context of developing and reciting a speech addressing one's biggest faults parallels decreases testosterone reported among those losing a competition (see Casto & Edwards, 2016). With regard to changes in cortisol, the lack of significant changes among women may be attributable to the nature of the stressor. In work considering the neuropsychological and behavioral correlates of cortisol response, such response is often induced with the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993). Core elements of the TSST include a speech task and mental arithmetic in front of an audience whose members do not provide feedback. For the social stressor in the current work, participants were asked to prepare and deliver a speech about their greatest faults. Participants were also instructed that their speeches would be recorded and later evaluated, but there was no direct socially evaluative component. The lack of significant change among women in the current study may then be attributable to the lack of direct social evaluation. Dickerson and Kemeny (2004) found cortisol response to social stress was greatest when stressors include a social evaluative threat in which participant performance could be negatively evaluated. It is possible a stressor with direct threat of negative social evaluation may induce larger increases in cortisol, potentially strengthening associations between change in cortisol and aggressive behaviors. The implications of choice of stressor for study outcomes would have potentially been informed by self-reports of participant stress. Unfortunately, self-reports of stress were not part of the data collection protocol and a lack of such reports are a limitation of the current work. Self-reports of stress may have also informed differences in the response to stress between men and women.

The pattern of association between hormones and aggressive behavior found in the current work may also be influenced by the use of a social stressor to elicit change in hormones rather than behavioral provocation (e.g., Denson et al., 2013). It is likely that the change hormones in response to an anxiety provoking social stressor differ in important ways from those elicited by aggression itself. Thus, associations between aggressive behavior and changes in hormones resulting from aggressive provocation may be stronger than those between aggressive behavior and hormone response to a social stressor such as the one used in the current work.

Other aspects of the study methodology potential influencing the current findings include the use of enzyme linked immunoassays to determine hormone levels, the short period of rest before initial saliva samples were collected, and the protracted window for data collection. While the use of enzyme linked immunoassays to determine testosterone and cortisol levels is fairly common in work relating hormones to traits and behaviors, there is evidence that the correlation between testosterone concentrations estimated with immunoassays and testosterone concentrations measured with liquid chromatography tandem mass spectrometry is weaker than the correlation between cortisol concentrations assayed measured immune assays and cortisol concentrations measured with mass spectrometry (Taieb et al., 2003; Welker et al., 2016). The use of immunoassays may then be regarded as a limitation of the current study. In addition, baseline measures of hormone concentrations may have been influenced by the short period of time between arrival for the laboratory protocol and the collection of the saliva sample for baseline hormones. Initial saliva samples were collected approximately 30 s after arrival for the laboratory protocol. While there is some precedence for the collection of saliva samples for hormonal assay of baseline measures immediately after arrival at a laboratory (see van Bokhoven et al., 2005), baseline hormone levels may have been elevated by activity and experiences occurring before arrival.

In the current study, saliva samples were collected between the hours of 0800 and 1830. While this prolonged window for data collection had the virtue of supporting a large sample size it may have influenced study results due to diurnal variation in both cortisol and testosterone (Dabbs, 1990; Pruessner et al., 1997). To directly assess the influence of time of day on associations, models were re-estimated with the sample restricted to those who participated in the laboratory protocol after 12 pm as discussed earlier. The potential influence of time of day on results is also informed by correlations between time of day and hormone measures (see Table 2). In general, time of day was negatively correlated with baseline hormone measures, but positively correlated with measure of change in hormones. This pattern of association is consistent with the law of initial values which holds that higher baseline values are associated with decreased change, leading to the expectation that decreases in hormone levels with time of day will be paralleled by increases in hormone reactivity. Associations between hormone measures and time

at which data were collected suggest that the time of data collection may be associated with membership in groups defined for simple slopes analysis. However, there is reason to anticipated that the potential influence of this association on results is minimal as time of day was not associated with aggressive behavior measures.

The pattern of findings in the current study may also be influenced by the measurement of aggressive behavior. The proactive aggressive behaviors captured by the RPQ occur relatively infrequently in community samples and with even less frequency among women in community samples. Our understanding of the association between hormones and aggressive behavior type may then be advanced through the use of measures capturing proactive aggressive behaviors that occur with increased frequency including proactive relational aggression (see Murray-Close, Ostrov, Nelson, Crick, & Coccaro, 2010). Future work should also attend to potential differences in association between hormones and aggression across self-report, other report, and task-based measures of aggression (see Dekkers et al., 2019). It is possible that social desirability bias associated with self-report measures influences associations between hormones and aggression (Baumeister, Vohs, & Funder, 2007). This issue may be particularly important for associations between testosterone and aggression as there is some evidence that associations between testosterone and aggressive behavior are stronger in studies using behavioral measures of aggression (Archer et al., 2005). However, a recent meta-analysis of the association between testosterone and aggression found that the association between testosterone and aggression was not moderated by the use of self-report measures (Geniole et al., 2019). Nonetheless, careful attention to the measurement of aggression may help to reconcile disparate findings. The implication of the results of the current work are also limited to a certain extent by sample size. While the overall sample size of the current study is large relative to earlier work, the final analytical sample for men is small, particularly for work testing interactions and in sensitivity analyses. Even if interactions explain a modest amount of variance (2 %) sample sizes of approximately 400 are required to achieve 80 % statistical power. Thus, the confidence given the results of the current work are somewhat tempered by sample size.

5. Conclusions

Strong conclusions regarding the nature of the association between hormones and aggressive behavior clearly await further study. However, a number of studies have now shown that low cortisol and high testosterone (reactivity) are associated with increased risk for aggressive behaviors. Along with this growing evidence there are also numerous null findings and findings in the opposite direction, including the current work where there was some evidence that reactive aggression may also be increased at low cortisol and low testosterone. Differences in results across studies are likely heavily influenced by key methodological differences including the measurement of aggression, the composition of the sample, the characterization of hormones (baseline vs. change), and the nature of the stressor in studies considering hormone change in response to stress. Therefore, it seems reasonable to suggest that systematic attention to the effect of variation in these aspects of methodology on results may yield substantial contributions to our understanding of the role of hormones in the etiology of aggressive behaviors. Similarly, our understanding of the nature of hormone interactions may be furthered by specific attention to the mechanisms that underpin these interactions. Growing evidence shows that the interaction of cortisol and testosterone plays a role in variation in risk for aggression but, it remains unclear if these interactions occur at the neuroendocrinological, neurological, or psychological level. To parse the association between hormones and aggression, studies should adopt a systematic focus on the association between variation in methodology and results in studies relating hormones to aggressive behaviors and extend to the consideration of the traits and biological processes that may mediate the association between hormones and aggressive behaviors.

Declaration of Competing Interest

The authors report no declarations of interest.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.biopsycho.2021.108073.

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