Basal cortisol's relation to testosterone changes may not be driven by social challenges

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Word count: 5030

Note: This research was supported by a National Science Foundation grant awarded to PHM (Award Number: 1451848). This funding sponsor was not involved in study design; in the collection, analysis and interpretation of data; in the writing of the report; or in the decision to submit the article for publication.

Abstract

Multiple studies show a negative correlation between basal cortisol and testosterone changes in the presence of competition and social-evaluative stressors. These negative associations are proposed to be derived from psychological responses to competition and social-evaluative stress. However, we argue that the association between basal cortisol and testosterone change may instead be a statistical consequence of positively associated variables. In this paper, we present a mathematical rationale for this alternative explanation and examples from two studies that are consistent with this alternative explanation. Both studies show that the associations between basal cortisol and testosterone change have covariance patterns consistent with this alternative possibility. We conclude that the often-found positive association between basal cortisol and basal testosterone opens the door for alternative explanations of the basal cortisol-testosterone change association rooted in the patterns of associations between hormones measured over time. We also suggest future research directions and methods for testing alternative explanations.

Keywords: *testosterone, cortisol, competition, stress, cross-talk*

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The functional "cross-talk" (e.g., Viau, 2002) between the hypothalamic-pituitary-gonadal (HPG) and hypothalamic-pituitary-adrenal (HPA) axes is an increasingly studied area of research in the field of psychoneuroendocrinology. Together, testosterone and cortisol may jointly regulate social behaviors and traits (see Mehta & Prasad, 2015 for a review). Additionally, simultaneous, coupled activation of testosterone and cortisol may characterize psychopathology and responses to stress (e.g., Marceau et al., 2015; Shirtcliff et al., 2015). Overall, research on cross-talk and interactions between these axes is being increasingly used to understand social behaviors and personality traits.

A recent line of research investigating HPA-HPG cross-talk has examined correlations between basal cortisol and changes in testosterone from before to after a competition or social-evaluative stressor. Testosterone often increases in the presence of competition or when status is challenged (Archer, 2006; Casto & Edwards, 2016; Geniole et al., in press). These testosterone increases tend to facilitate dominant behavioral responses in the face of social challenges (Wingfield et al., 1990). Moreover, because of evidence for cross-talk between the HPA and HPG axes, researchers have investigated whether basal cortisol is associated with the observed testosterone changes during social challenges. Bedgood and colleagues demonstrated a negative correlation between basal cortisol and testosterone reactivity during a socially evaluative stressor (Bedgood, et al., 2014). Specifically, lower basal cortisol levels predicted increased testosterone reactivity to the stressor. Additionally, Edwards and Casto (2015) extended these results by reporting a similar negative relation between basal cortisol and testosterone reactivity during athletic competitions. These findings are parallel to those of Mehta and Josephs (2006) and Maestripieri and colleagues (2010), that also show moderate negative associations between basal

cortisol and testosterone changes in response to laboratory competitions and psychosocial stressors.

Based on these results, an elegant conclusion is that the association between basal cortisol and testosterone reactivity during competitions or stressors corresponds to psychological states and behaviors in stressful and competitive contexts (Bedgood et al., 2014; Edwards and Casto; 2015). In particular, Bedgood et at al. (2014) proposed that individuals with high basal cortisol levels perceive social evaluative situations as a threat to status rather than as a dominance challenge, leading to a blunted testosterone response (Bedgood et al., 2014). A similar psychological mechanism during athletic competition may explain the negative association between basal cortisol and testosterone change in the Edwards & Casto (2015) study. For brevity in this article, we refer to this potential explanation as the *"social challenge explanation"* of the association between basal cortisol and testosterone changes. The social challenge explanation claims that the negative association between basal cortisol and testosterone reactivity is a function of HPA-HPG cross-talk in social-evaluative stress and competitive contexts, particularly where status is contested.

This compelling social challenge explanation of the basal cortisol-testosterone change correlation would indeed be influential in moving psycho-neuroendocrine theory of competition and status forward. However, we argue it is premature to conclude that this correlation is produced by psychological factors. We propose another possibility for the association between basal cortisol and testosterone changes. A critical step in establishing the veracity of the social challenge explanation involves ruling out potential alternative explanations. We propose that the association between basal cortisol and testosterone changes is a direct consequence of covariance patterns between hormones measured over time and provide empirical evidence consistent with

this alternative explanation. At this point, it is unknown what factors may create these covariance patterns, and if the factors are social psychological or physiological in nature. Therefore, it is necessary to explain what factors lead a negative basal cortisol and testosterone change association when measured over time. Although we refer to this as an "alternative possibility" or "explanation", there may be many other? possibilities that enable the conditions we identify contributing to a negative basal cortisol-testosterone change association. In this paper, we describe a rationale for this alternative statistical possibility and present data that are consistent with this alternative explanation. If this possibility is true, a critical next step is to determine what factors produce the covariance patterns that lead to a negative correlation between basal cortisol and testosterone changes. To do so, we also provide researchers with directions moving forward to investigate this alternative possibility.

1. The Alternative Statistical Possibility

With this alternative possibility, we propose that the negative association between basal cortisol and testosterone reactivity may be a statistical consequence of positively associated variables (i.e., basal testosterone and basal cortisol concentrations; Mehta & Josephs, 2010; Mehta et al., 2015; Popma et al., 2007; Welker et al., 2016). More specifically, this association could be a consequence of the mathematics behind a correlation between two variables—in this case testosterone and cortisol—measured at different times. Here, we present the argument that a negative covariance (and also a correlation) between basal cortisol and testosterone changes (assessed as difference scores) is inevitable, provided that the association between basal cortisol and basal testosterone (i.e. cortisol and testosterone measured at the same time) is stronger than the association between basal cortisol and testosterone measured at a later point. To illustrate

this, we draw from the equation of a covariance between two variables, denoted here as X and Y in Equation 1:

$$cov(\mathrm{Y},\mathrm{X}) = \frac{\sum_{i=1}^{n}(Y_i-\bar{Y})(X_i-\bar{X})]}{n-1} \quad (1)$$

In this equation, we substitute basal cortisol and testosterone changes into the values of Y and X, respectively. Equation 2 presents an algebraic statement of the covariance between basal cortisol (C1) and the change from basal testosterone (T1) to a later measure of testosterone (T2) as (*cov*(C1, [T2 – T1])). From this, we derive (Equation 2) that the covariance of basal cortisol (C1) and testosterone changes from baseline (T2-T1) is equal to the covariance of basal cortisol and post-competition testosterone (*cov*(C1, T2)) minus the covariance of basal testosterone and cortisol (*cov*(C1, T1)).

$$cov(\mathrm{C1},[\mathrm{T2}-\mathrm{T1}]) = \frac{\sum_{i=1}^{n}(C1_i-\overline{C1})[(T2_i-T1_i)-(\overline{T2}-\overline{T1})]}{n-1} \quad (2)$$

$$cov(\mathrm{C1},[\mathrm{T2}-\mathrm{T1}]) = \frac{\sum_{i=1}^{n}(C1_i-\overline{C1})[(T2_i-\overline{T2})-(T1_i-\overline{T1})]}{n-1}$$

$$cov(\mathrm{C1},[\mathrm{T2}-\mathrm{T1}]) = \frac{\sum_{i=1}^{n}[(C1_i-\overline{C1})(T2_i-\overline{T2})-(C1_i-\overline{C1})(T1_i-\overline{T1})]}{n-1}$$

$$cov\big(\mathrm{C1},\big[\mathrm{T2}-\mathrm{T1}\big]\big) = \frac{\sum_{i=1}^{n}[(C1_i-\overline{C1})(T2_i-\overline{T2})]}{n-1} - \frac{\sum_{i=1}^{n}[(C1_i-\overline{C1})(T1_i-\overline{T1})]}{n-1}$$

cov(C1, [T2 – T1]) = *cov*(C1, T2) – *cov(*C1, T1)

Based on this equation, if the covariance between basal testosterone and basal cortisol (*cov*(C1, T1)) is greater than the covariance between basal cortisol and post-competition testosterone (*cov*(C1, T2)), it follows that a negative covariance between testosterone changes and basal cortisol will occur. This is because this pattern of covariances will result in a negative value for *cov*(C1, [T2 – T1])). More simply, subtracting a larger positive quantity from a smaller positive quantity provides a negative quantity.

A correlation is mathematically linked to a covariance, as bivariate correlation coefficients in the form of Pearson's *r* are computed by dividing a covariance (*cov*(Y,X)) by the product of the standard deviations (σY and σX) of both variables (Equation 3).

$$r = \frac{cov(Y,X)}{\sigma Y \sigma X} \quad (3)$$

Indeed, statistically, a correlation can be interpreted as a "standardized covariance" (Rogers & Nicewander, 1988). Therefore, this potential statistical explanation will influence correlational analysis in the same way provided that the variances are not changing over time.[1,2]

2. Evidence consistent with the statistical explanation

According to the social challenge explanation, competition or stressors lead to a negative association between basal cortisol and testosterone reactivity. However, the alternative statistical possibility predicts that the negative association between basal cortisol and testosterone reactivity should emerge across a variety of contexts, provided the condition basal testosterone and basal cortisol are positively associated and simultaneously measured hormones are more strongly correlated than measures of hormones from different times. Although the circumstances arising to this condition need to be explained by future research, basal testosterone and basal cortisol have a well-established positive correlation from previous literature (e.g., Mehta & Josephs, 2010; Mehta et al., 2015; Popma et al., 2007; Welker et al., 2016, Zilioli & Watson, 2013). Furthermore, simultaneously measured variables that are correlated across time often have stronger synchronous correlations compared to when multiple measurements of variables are correlated across time in lagged correlations (Kenny, 1975; Kenny & Campbell, 1989),

[1] Bedgood and colleagues (2014) and Edwards and Casto (2015) do not report correlations between individual testosterone and cortisol samples in either paper. Thus, it is not possible to compare correlations between simultaneously sampled testosterone and cortisol to cortisol and testosterone sampled at different times.

[2] For a related statistical explanation based on regression to the mean, see Maestripieri et al (2010).

which is often the case with hormones (e.g., Liening et al., 2010; Welker et al., in press). Indeed, many cross-lagged analyses show this pattern of correlations where synchronous correlations between two variables are stronger than lagged correlations (e.g., Racine et al., 2016; Roest et al., 2016; Spurk & Abele, 2014; Wirtz et al., 2014). Therefore, if testosterone and cortisol have a sizeable synchronous correlation and the lagged correlations between testosterone and cortisol are weaker than the synchronous correlation, then a relationship between basal cortisol and testosterone change would likely be negative.

In investigating this alternative possibility, we analyzed archival data to test these associations across social contexts. To show data consistent with the alternative statistical possibility, we examined the patterns of covariance between basal cortisol and testosterone change to determine whether the data conform to Equation 1 mentioned above. Investigating both correlations and covariances, we also examined whether the association between testosterone and cortisol concentrations measured at the same time (i.e., *cov*(C1, T1), *cov*(C2, T2)) are stronger than those of the testosterone and cortisol measured at different times (i.e., *cov*(C1, T2), *cov*(C2, T1)).

In a recently published paper (Mehta et al., 2015a), we measured testosterone and cortisol reactivity during a face-to-face negotiation task (Study 1) and across multiple rounds of the Ultimatum Game (Study 2). In Study 1, participants (N = 70, 39% Women) provided a baseline saliva sample, were randomly paired with a partner, and assigned the role of a buyer or seller. Participants then engaged in a negotiation task involving selling a hypothetical pharmaceutical plant (the Synertech-Dosagen case; Galinksy & Mussweiler, 2001). Participants negotiated for approximately 15 minutes and provided a second saliva sample 20 minutes after the negotiation. In Study 2, participants (N = 115, 53% Women) provided a saliva sample and then played 30

one-shot rounds of the Ultimatum Game (Güth, Schmittberger, & Schwarze, 1982) for approximately 10 minutes. The Ultimatum game is an economic decision-making paradigm assessing whether people will retaliate against unfair treatment. In the game, a participant can pay a financial cost to punish the other participant for making an unfair offer. After the game, participants provided a second saliva sample to assess testosterone and cortisol roughly 20 minutes after the completion of the Ultimatum Game.

We took several measures to calculate changes in hormones. Cortisol values were log transformed to correct for skewness before this difference was calculated and then standardized, whereas testosterone was standardized within men and women separately. Absolute change in testosterone and cortisol were calculated by subtracting transformed Time 2 hormone concentrations from transformed Time 1 hormone concentrations (after the previously mentioned transformations were applied; See Mehta et al., 2015a).

Correlations in both studies between testosterone and cortisol levels at the corresponding time points, as well as testosterone and cortisol change, are presented in Table 1. To demonstrate the alternative statistical possibility, we examined the difference between the covariance between testosterone and cortisol at Time 1 and the covariance between Time 1 cortisol and post-negotiation testosterone (measured at Time 2) for both studies. Below, we present the covariances between Time 1 cortisol and testosterone changes (σ(C1, [T2 – T1])) along with the covariance between Time 1 cortisol and testosterone (σ(C1, T1)) and Time 1 cortisol and time 2 testosterone (σ(C1, T2)). Cases were excluded listwise so that covariances represented only those with no missing data for C1, T1, and T2. These covariances also illustrate the principle that σ(C1, [T2 – T1]) is derived from σ(C1, T2) – σ(C1, T1):

Study 1: σ(C1, [T2 – T1]) = σ(C1, T2) – σ(C1, T1) = .06 – .21 = -.15

Study 2: σ(C1, [T2 – T1]) = σ(C1, T2) – σ(C1, T1) = .14 –.30 = -.16

Consistent with the alternative statistical possibility, these results show that in both studies that σ(C1, T1) is indeed greater than σ(C1, T2), which results in a negative σ(C1, [T2 – T1]).

To supplement this primary evidence for our alternative explanation, we also examined correlations, excluding missing cases listwise. Expectedly, the pattern of correlations was similar to pattern of covariances. Similar to the findings of Bedgood and colleagues (2014) and Edwards and Casto (2015), basal cortisol was negatively associated with testosterone change in Study 1 (r(67)= -.24, p = .047) and Study 2 (r(109)= -.26, p = .007). Additionally, the basal testosterone and cortisol correlations (r(67) = .23, p = .057. and r(109) = .30, p = .002 for Studies 1 and 2, respectively) were generally of more robust magnitude than testosterone and cortisol correlations from measures at different times (rs from .04 to .24, See Table 1). For interested readers, we also examined the pattern of correlations and covariances separately for men and women, noting that they are of similar magnitude as the pattern of findings across men and women (see Tables S1 and S2 in the Online Supplemental Materials). Furthermore, since basal testosterone was correlated with testosterone changes in Studies 1 (r(67) = -.30, p = .016) and 2 (r(109) = -.32, p = .001), we also controlled for basal testosterone when examining the basal cortisol and testosterone change association. Doing so resulted in a negative, albeit nonsignificant, partial correlation in Study 1 (r_p(67)= -.19, p = .131) and a marginally-significant negative correlation in Study 2 (r_p(109) = -.18, p = .058). Although the differences in these correlations was nonsignificant (ps ≥ .276) and our data was likely under-powered to detect such a change, this relationship was slightly attenuated but still in the same negative direction, when accounting for both basal hormone measurements.

We note that the association between post-interaction testosterone and cortisol was diminished ($r(67) = -.02$, $p = .855$ and $r(109) = .15$, $p = .120$ for Studies 1 and 2, respectively). This reduced association between post-task testosterone and cortisol may exist because post-task testosterone and cortisol are influenced by more situational and individual difference factors in addition to the interaction. For instance, a growing body of work suggests several factors that may promote hormonal reactivity to competitive and stressful interactions, including enjoyment of the activity (Mehta et al., 2015b), competitive outcomes (e.g., Carré et al., 2013; Mehta et al., 2015b), and aggressiveness (e.g., Carré et al., 2010; Carré et al., 2013). These factors may have contributed to more unexplained variation in post-interaction hormones, relative to basal hormones. We return to this point later.

3. Discussion

Overall, our calculations and the patterns in our data suggest that the negative association between basal cortisol and testosterone changes may be a statistical consequence of positive correlations between basal cortisol and basal testosterone. Therefore, it is plausible that observations of HPA-HPG cross-talk (i.e., associations between basal cortisol and testosterone change) may not be relevant to adapting to social challenges. With the goal of moving theory forward, we propose several future directions to further establish whether the social challenge explanation may be true. We also speculate about the possible reasons that may give rise to the covariance patterns between hormones measured at different times that lead to the negative correlation between basal cortisol and testosterone changes.

First, a critical step forward to test the social challenge explanation is to experimentally manipulate the presence of social challenges, such as randomly assigning participants to a stressful/competitive condition or a control condition. Although concerns over this limitation have been raised (Bedgood et al., 2014), no research, including data presented from the present

research, has yet compared the relationship between basal cortisol and testosterone reactivity in the presence and absence of social challenges. Such a study might use moderated regression analysis to test for a significant interaction between basal cortisol and a variable coding for the experimental condition predicting testosterone responses. This approach would be superior to examining correlations between basal cortisol and testosterone changes separately within different experimental conditions (for an example of the latter approach, see Mehta & Josephs, 2006). Indeed, it would provide a proper test of whether the experimentally manipulated social context significantly changes (that is, *moderates*) the relationship between basal cortisol and testosterone responses[3]. If the social challenge explanation is true, then researchers will find significantly more pronounced negative associations between basal cortisol and testosterone in competition or stressful situations compared to control conditions (as Bedgood et al., 2014 suggest). Future research with this approach will help determine if the presence of a social challenge or competition alters the association between basal cortisol and testosterone change. However, if the social challenge explanation were not true, this association would be constant regardless of the psychological or social context. It is also important that research testing this explanation be fueled by studies with larger sample sizes, as the empirical data presented in this paper and by previous researchers (Bedgood et al., 2014, Edwards & Casto, 2015) are likely underpowered for detecting statistical moderation. Future work with larger sample sizes will provide adequate statistical power to investigate whether social contexts and challenges systematically alter how basal cortisol and testosterone changes are associated.

[3] Both Bedgood et al. (2014) and Edwards & Casto (2015) used the terms "moderator" or "moderates" in their discussion of social contexts without sufficient evidence. We caution against the use of these terms in study designs that are not designed to test for moderation. Instead, we recommend terms such as association or relationship for correlational studies like Bedgood et al. (2014) and Edwards & Casto (2015).

By experimentally manipulating the social context, researchers might be able to parse out the psychologically-meaningful variability in the basal cortisol and testosterone change relationship from the variability that is a by-product of statistical covariances. It is possible that by controlling for basal hormones in the analyses of hormone changes, either through partial correlations, regression models, or lagged analyses using repeated measures (e.g., multilevel modeling, lagged path analysis), researchers might estimate variability in hormone changes independent of the influence of basal hormones. Other researchers have used residualized values of change to index hormonal changes created from regressing a later measure of hormones on an earlier or basal hormone measurement (e.g., Smith & Apicella, in press; Welker et al., in press). However, this approach may not fully resolve this problem. In the current research, doing this resulted in smaller negative partial correlations between basal cortisol and testosterone changes, suggesting that basal testosterone accounts for some of the variability in this relationship. However, controlling for these factors did not substantially or significantly decrease the negative association between testosterone changes and basal cortisol. Although these approaches may not fully mitigate the statistical explanation we raise, they will aid in examining hormone changes independent of the influence basal hormones.

Another direction is for researchers to share the correlations between basal hormones and hormone changes in future publications, or from previously collected data through data sharing initiatives such as the open science framework (https://osf.io/), when possible. As research on HPG-and HPA cross-talk unfolds, researchers might begin investigating meta-analytic associations between basal hormones and changes. The availability of this data will help researchers compare studies presenting basal and hormone change correlations across a variety of social and experimental contexts. Moreover, meta-analytically analyzing the differences in

basal and hormone change correlations across different contexts would also help determine the extent to which specific contextual factors moderate the association between basal cortisol and testosterone changes.

This paper helps demonstrate why a negative correlation between basal cortisol and testosterone changes would occur given the correlations between two hormones measured at baseline compared to lagged correlations. However, a larger remaining issue needs to be addressed: Why is the covariance between basal cortisol and testosterone greater than the covariance between basal cortisol and a lagged measure of testosterone? We speculate that many of the laboratory methods that researchers employ when assessing hormonal changes (e.g., stress-induction tasks, competitive interactions) may induce a variety of differential hormone responses depending on the person. This may lead to reduced correlations between hormones measured at different times or after the laboratory task, relative to the baseline hormones. Indeed, our data followed this pattern, with correlations being the strongest between single measures of testosterone and cortisol at baseline. However, by comparing data from many different research approaches and hormonal changes measured in the absence of a psychologically-relevant task, researchers will have a better sense of why correlations decrease when using lagged hormone measures compared to baseline measures. Furthermore, researchers will be able to detect whether the negative correlation between basal cortisol and testosterone changes occurs across many different social contexts.

Finally, an additional next step is to employ pharmacological manipulations of cortisol (e.g., Metyrapone or Dexamethasone) to examine whether cortisol can causally alter testosterone reactivity. Such a design could test whether exogenously-administered cortisol diminishes testosterone reactivity to social challenges, supporting the presence of social challenge-

dependent HPA-HPG crosstalk. These new directions in research may shed new light on HPA and HPG cross-talk in the presence of social challenges, and its potential influence on behavior.

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Table 1. Associations between testosterone, cortisol, and hormonal changes in Mehta et al (2015a).

Correlations						
Study 1	1	2	3	4	5	6
1. Basal Cortisol (lg10, standardized)	—					
2. Basal Testosterone (standardized within sex)	.23†	—				
3. Post-interaction Cortisol (lg10, standardized)	.53***	.04	—			
4. Post-interaction Testosterone (standardized within sex)	.06	.77***	-.02	—		
5. Cortisol Change	-.60***	-.22*	.37**	-.09	—	
6. Testosterone Change	-.24*	-.30*	-.09	.39***	.18	—
Study 2	1	2	3	4	5	6
1. Basal Cortisol (lg10, standardized)	—					
2. Basal Testosterone (standardized within sex)	.30**	—				
3. Post-interaction Cortisol (lg10, standardized)	.76***	.24*	—			
4. Post-interaction Testosterone (standardized within sex)	.14	.81***	.15	—		
5. Cortisol Change	-.37***	-.10	.32***	.01	—	
6. Testosterone Change	-.26**	-.32***	-.14	.30***	.18†	—
Covariances						
Study 1	1	2	3	4	5	6
1. Basal Cortisol (lg10, standardized)	.91					
2. Basal Testosterone (standardized within sex)	.21	.88				
3. Post-interaction Cortisol (lg10, standardized)	.42	.03	.68			
4. Post-interaction Testosterone (standardized within sex)	.06	.70	-.02	.95		
5. Cortisol Change	-.50	-.18	.26	-.08	.76	
6. Testosterone Change	-.15	-.18	-.05	.25	.10	.43
Study 2	1	2	3	4	5	6
1. Basal Cortisol (lg10, standardized)	1.02					
2. Basal Testosterone (standardized within sex)	.30	1.02				
3. Post-interaction Cortisol (lg10, standardized)	.76	.24	.97			
4. Post-interaction Testosterone (standardized within sex)	.14	.82	.15	1.01		
5. Cortisol Change	-.26	-.07	.21	.01	.47	
6. Testosterone Change	-.16	-.20	-.09	.19	.08	.39

Note: †$p < .10$ *$p < .05$, **$p < .01$, ***$p < .001$. Outliers in Study 1 were Winsorized to 3 SDs after transformations were applied. There were no outliers identified in Study 2. Covariances and correlations are presented excluding cases listwise. Because the standardized variables had

outliers Winsorized after standardizing the variables, the diagonal covariances are not precisely equal to 1.