Hormones and Ethics: Understanding the Biological Basis of Unethical Conduct
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CITATION
BRIEF REPORT

Hormones and Ethics: Understanding the Biological Basis of Unethical Conduct

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Globally, fraud has been rising sharply over the last decade, with current estimates placing financial losses at greater than $3.7 trillion annually. Unfortunately, fraud prevention has been stymied by lack of a clear and comprehensive understanding of its underlying causes and mechanisms. In this paper, we focus on an important but neglected topic—the biological antecedents and consequences of unethical conduct—using salivary collection of hormones (testosterone and cortisol). We hypothesized that preperformance cortisol levels would interact with preperformance levels of testosterone to regulate cheating behavior in 2 studies. Further, based on the previously untested cheating-as-stress-reduction hypothesis, we predicted a dose–response relationship between cheating and reductions in cortisol and negative affect. Taken together, this research marks the first foray into the possibility that endocrine-system activity plays an important role in the regulation of unethical behavior.

Keywords: hormones, cortisol, testosterone, behavioral ethics, cheating

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According to the most recent Global Fraud Survey by the Association of Certified Fraud Examiners (ACFE, 2014), fraud is a global phenomenon, affecting nearly 100 countries on six continents, with financial losses estimated at more than $3.7 trillion annually. These troubling data may explain why scholars across disciplines, from law and economics to psychology and management, have become invested in understanding why people—even those who report placing a high value on morality—often end up behaving unethically. To date, research has focused on identifying situational pressures that can sway a person’s moral compass (Bazerman & Gino, 2012). In part because of the immense social and economic costs of financial fraud (Ribstein, 2002), the untested possibility that endocrine-system activity might play a role in encouraging dishonesty in pursuit of financial reward is timely, novel, and potentially very important.

In this study, we have introduced the novel hypothesis that cross talk between the reproductive and stress axes of the human endocrine system (Viau, 2002) predicts unethical behavior. The primary end product of the body’s reproductive axis (hypothalamic–pituitary–gonadal) is testosterone, and the primary end product of our stress axis (hypothalamic–pituitary–adrenal) is cortisol. Based on the dual-hormone hypothesis (Mehta & Josephs, 2010), which states that testosterone’s influence on a person’s drive to acquire status-bearing resources (e.g., money, power, leadership) is stymied by high levels of cortisol, we proposed that elevated testosterone encourages individuals to cheat for financial gain, but only when cortisol concentrations are low. We also tested another novel hypothesis: that changes in cortisol distinguish those who engage in unethical behavior from those who remain honest.

Testosterone encourages the pursuit of social-approach strategies. These include a wide palette of behaviors and tendencies, including status seeking and power seeking (Mazur & Booth, 1998), reward seeking (Op de Macks et al., 2011), risk taking (Coates, Gurmel, & Sarnyai, 2010), a preoccupation with status (Josephs, Sellers, Newman, & Mehta, 2006), fear reduction (Hermans, Putman, & van Honk, 2006; Putman, Hermans, & van Honk, 2007; van Honk, Peper, & Schutter, 2005), and an insensitivity to punishment (van Honk et al., 2004).
It is interesting to note, testosterone can also encourage pursuit of antisocial-approach strategies, including rule violations, violence, and deviant behavior (Dabbs, Carr, Frady, & Riad, 1995; Popma et al., 2007). Because unethical behavior in its many forms, from academic cheating to sexual infidelity to business misconduct, generally involves risk taking and rule breaking in pursuit of a reward, goal, or competitive advantage, it is logical that testosterone would play a role in unethical behavior.

We designed the current study to investigate the novel hypothesis that testosterone and cortisol jointly influence the pursuit of dishonest financial gain. Because testosterone is positively associated not only with social approach, but also with deviant social approach when cortisol is low (Dabbs, Jurkovic, & Frady, 1991; Popma et al., 2007; Tackett, Herzhoff, Harden, Page-Gould, & Josephs, 2014), we proposed that if testosterone is connected to unethical behavior, this connection should be observed primarily when cortisol levels are low, with higher cortisol levels weakening the connection.

Against a majority of studies supporting the dual-hormone hypothesis are several null-effect studies and several reversals: Testosterone effects only at high levels of cortisol (Hamilton, Carré, Mehta, Olmstead, & Whitaker, 2015). To date, although it is not understood why elevated cortisol might promote testosterone-associated behaviors, there is emerging evidence that elevated cortisol has behavioral influences over and beyond cortisol’s influence on testosterone, including the possibility that elevated cortisol might play a cardinal role in cheating.

The possibility that elevated cortisol might encourage cheating derives in part from the view that cheating has been conceptualized as a reaction to the stress and uncertainty associated with performance. According to this view, anxiety evoked by scholastic uncertainty encourages cheating, presumably as a means of reducing uncertainty and thus anxiety (Anderman, Griesinger, & Westerfield, 1998). In support of this idea, anxiety was reported to increase the likelihood of cheating among college students (Berger, Levin, Jacobson, & Millham, 1977). Incidental anxiety has also been reported to increase the likelihood of other unethical behaviors (Kouchaki & Desai, 2015). A recent study reports that cortisol mediates the association between experimentally manipulated unfairness and ethically questionable behavior, with higher levels of cortisol associated with a greater likelihood of ethically questionable behavior (Yang, Bauer, Johnson, Groer, & Salomon, 2014).

Even if anxiety and/or elevated cortisol plays a role in unethical behavior, clearly not all anxious or stressed individuals engage in unethical behavior; thus, elevated cortisol—even if it is an anxiety marker and linked to unethical behavior—cannot explain completely the connection between anxiety/stress and cheating. Here, testosterone may play an important role. What may distinguish the anxious cheater from his anxious but honest counterpart is the constellation of behaviors and tendencies associated with elevated testosterone (e.g., fearlessness, recklessness, reward sensitivity, and so on). When provided with the opportunity to cheat, a person with elevated cortisol—who presumably is suffering from heightened levels of anxiety/stress—may assume that cheating will reduce these feelings. Furthermore, if this same person also has elevated testosterone—thus shifting in favor of reward-seeking behaviors—then perhaps the endocrine profile that maximizes the likelihood of unethical behavior is not high testosterone coupled with low cortisol, as predicted by the dual-hormone hypothesis, but rather high testosterone with high cortisol. We designed a pilot study to test these competing hypotheses.

**Pilot Study**

**Method and Materials**

We recruited 101 participants ($M_{\text{age}} = 21.85$, $SD_{\text{age}} = 2.70$, 38.6% male) for an hour-long study and paid each $10. Participants could earn up to an additional $20 depending on their self-reported progress on a performance task. Seventeen participants were excluded because we were unable to analyze their saliva samples for hormones levels, and two were excluded for not following the experimenter’s instructions. This resulted in a total of 82 participants ($M_{\text{age}} = 21.93$, $SD_{\text{age}} = 2.81$, 41% male).

Before the performance task, we collected a saliva sample via passive drool (see online supplemental materials for more details on the method). To measure cheating, we used a previously validated pencil-and-paper performance task designed to reward participants financially for being dishonest (Gino, Schweitzer, Mead, & Ariely, 2011; Mazar, Amir, & Ariely, 2008). The task provided an opportunity to cheat by instructing participants to dispose of the evidence of their actual performance, creating the perception that the experimenter could not verify their performance. Unbeknownst to participants, the last two digits on each of their discarded paperwork matched digits provided on the slip of paper they gave to the experimenter prior to starting the task. This allowed the experimenter to connect the discarded paperwork to a particular participant and evaluate the participant’s actual performance against his or her stated performance.

Overstating the number of correctly solved matrices was used as the measure of unethical behavior. Magnitude of unethical behavior was determined simply by subtracting the actual from the self-reported number of correctly solved matrices. At the end of the experiment, participants answered a series of health-related questions. None of these health-related questions was significantly correlated with hormone levels or unethical behavior, all $ps > .21$; thus, we did not use them in the analyses.

**Results**

Participants solved an average of 8.29 out of 20 matrices ($SD = 3.98$; minimum = 0, maximum = 19). Of the participants, 36% ($SD = 0.48$) cheated, which was defined as overstating the number of correctly solved matrices. The average cheater claimed to have solved 1.9 more matrices than she or he actually solved ($SD = 2.86$; minimum = 0, maximum = 12). Zero-order correlations between the key variables are reported in Table S1 in the online supplemental materials.

**Actual task performance.** We first tested whether hormone levels and unethical behavior predicted actual performance (measured by the number of correctly solved matrices). Because actual performance was composed of discrete count data and described by a skewed, dispersed distribution, we used negative binomial regression to predict actual performance. Our predictors were preperformance hormone levels and unethical behavior, with par-
participant’s age as a covariate. Neither preperformance hormone levels nor age predicted actual performance, ps > .20. However, unethical behavior (operationalized as the number of overstated matrices) was negatively correlated with the number of correctly solved matrices (actual performance), \( B = 0.03, SE = 0.01, p = .03, 95\% \text{ CI } [-.06, -00]. \)

**Unethical behavior as a consequence of preperformance hormones.** Because the modal cheating score was 0 (61% of participants didn’t cheat; thus the difference between actual and stated performance was 0), we used a Poisson regression to model cheating as a function of hormone levels. In all subsequent analyses, actual performance (number of correctly solved matrices) was covaried to ensure that any observed influence of hormones on unethical behavior was not due to performance levels. We also covaried age and time of day.

We first tested the general hypothesis that testosterone and cortisol exert a joint influence on unethical behavior. In support, we found a significant interaction, \( B = .58, SE = .26, p = .03, 95\% \text{ CI } [.06, 1.09]. \) Adding or removing the covariates such as actual performance, time of day, and age did not change the nature and significance of the primary results.

We next tested our two primary competing hypotheses that the association between endogenous testosterone level and unethical behavior would (a) only occur when accompanied by low cortisol concentrations or (b) would be amplified by high cortisol concentrations (see Table 1). A simple-slopes analysis supported the second hypothesis—that the association between testosterone and unethical behavior was amplified by the presence of high levels of cortisol. Under low cortisol (−1 SD), testosterone did not predict unethical behavior, \( B = .17, SE = .93, p = .85. \) However, testosterone was a marginally significant predictor of unethical behavior under high cortisol (+1 SD), \( B = 1.72, SE = .92, p = .07. \) Figure 1 depicts this relationship.

**Discussion**

This study has provided promising tentative support for the possibility that unethical behavior is encouraged under elevated levels of testosterone and cortisol. This finding, if replicable, sets a precedent for future empirical investigations of the biological basis for unethical behavior. Given the potential importance of this finding, we conducted a new study as an attempt at replication.

**The Main Study**

This study differed from the pilot study in two important ways. First, we increased the sample size by 50%, substantially increasing statistical power and thus the probability of detecting an effect. Second, in addition to collecting a pretask saliva sample, analyzed for testosterone and cortisol concentrations, we collected a post-task saliva sample and analyzed it for cortisol concentrations. This allowed us to test the intriguing and yet untested hypothesis that there may exist a dose–response relationship between cheating and reductions in stress and negative affect. Coping theorists argue that stressors trigger behaviors that are aimed at coping (Lazarus & Folkman, 1984), and an act of cheating may be used to reduce stress and anxiety. Although direct empirical tests of cheating-as-a-stress-reduction mechanism are largely missing from the literature, there is evidence that cheating can evoke positive affect, a phenomenon termed “the cheater’s high” (Ruedy, Moore, Gino, & Schweitzer, 2013). Thus, demonstrating a dose–response relationship between cheating and reductions in psychological and/or physiological distress strikes us as being a critically important linchpin in the etiology of fraud, cheating, and other unethical behaviors.

**Table 1**

Hierarchical Poisson Regression Models: Frequency of Cheating as a Dependent Variable, Pilot Study

<table>
<thead>
<tr>
<th>Predictor variables</th>
<th>Frequency of cheating</th>
<th></th>
<th></th>
<th></th>
<th></th>
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<th></th>
<th></th>
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<tr>
<td></td>
<td>Step 1</td>
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<td>Step 2</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Actual performance</td>
<td>(-.07^{**})</td>
<td>.02</td>
<td>[(-.12, -.03)]</td>
<td>(-.07^{**})</td>
<td>.02</td>
<td>[(-.12, -.03)]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Time of day</td>
<td>.07</td>
<td>2.47</td>
<td>[(-.70, .80)]</td>
<td>5.28</td>
<td>2.58</td>
<td>[(-2.18, 10.30)]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>.00</td>
<td>.03</td>
<td>[(-.06, .05)]</td>
<td>.01</td>
<td>.03</td>
<td>[(-.07, .05)]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preperformance testosterone</td>
<td>(.42^{*})</td>
<td>.17</td>
<td>[(.09, .75)]</td>
<td>.19</td>
<td>.18</td>
<td>[(-.55, .17)]</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Preperformance cortisol</td>
<td>(N)</td>
<td>82</td>
<td></td>
<td>82</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Interaction: Testosterone \times Cortisol</td>
<td>(\chi^2)</td>
<td>13.34</td>
<td></td>
<td>26.4</td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Prob &lt; (\chi^2)</td>
<td>.004</td>
<td></td>
<td></td>
<td>&gt;.001</td>
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</tr>
</tbody>
</table>

*Note.* LR = likelihood ratio; Prob = probability.

\* \(p < .05. \) ** \(p < .01. \) *** \(p < .001. \)
Method and Materials

Relative to the pilot study, sample size in the main study was increased by 50% to enhance statistical power. We recruited 120 adults ($M_{age} = 21.58, SD_{age} = 2.52, 58\% \text{ male}$) who participated in an hour-long study and were each paid $10. Again, participants could earn up to an additional $20, depending on their self-reported progress on a performance task. Three participants were excluded from further analysis because they did not follow instructions during the experiment, resulting in a total of 117 individuals ($M_{age} = 21.57, SD_{age} = 2.53, 57\% \text{ male}$). Participants completed the pencil-and-paper performance task from the pilot study by solving math matrices and self-reporting the number of matrices completed to the experimenter.

We used the method from the pilot study to measure cheating (Gino et al., 2011; Mazar et al., 2008). In addition to the measures used in the pilot study, participants twice completed a 10-item scale of negative affect from the Positive and Negative Affect Scales (PANAS; Watson, Clark, & Tellegen, 1988), indicating the extent to which they experienced these emotions: distressed, upset, guilty, ashamed, hostile, irritable, nervous, jittery, scared, and afraid ($1 = \text{not at all}, 7 = \text{very much}$). Participants completed this scale before the performance task (i.e., at baseline, $\alpha = .86$) and after it ($\alpha = .89$). Similarly, we collected saliva samples from participants via passive drool before the performance task to assess pretask testosterone and cortisol concentrations, and 15 min after the performance task to assess posttask cortisol levels.

We then asked participants to answer the same health-related questions as well as to give their age and gender. None of the health variables were significantly correlated with hormone levels or unethical behavior, all $p > .23$. Thus, we excluded them from further analyses. Last, we used the 19-item State–Trait Anxiety Inventory to measure participants’ trait anxiety ($\alpha = .93$; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). Participants indicated the extent to which they felt generally anxious by endorsing 19 statements (e.g., “I get in a state of tension or turmoil as I think over my recent concerns and interests;” $1 = \text{almost never}$ to $4 = \text{almost always}$).

Results

Participants solved an average of 8.02 out of 20 matrices ($SD = 3.87$; minimum $= 0$, maximum $= 19$). Of the participants 44% ($SD = .50$) cheated, defined as overstating the number of correctly solved matrices. The average cheater claimed to have solved 1.9 more matrices than s/he actually solved ($SD = 3.44$; minimum $= 0$, maximum $= 16$). Zero-order correlations between the key variables are reported in Table S3 in the online supplemental materials.

Actual task performance as a consequence of preperformance hormones. As in our pilot study, our dependent measure was composed of discrete count data, and was described by a skewed, dispersed distribution. Thus, we used negative binomial regression to predict actual performance. Our predictors were preperformance hormone levels and unethical behavior, with participant’s age plus state and trait anxiety entered as covariates. Neither preperformance hormone levels nor any of the covariates predicted actual performance, $ps > .05$. We found it interesting that the number of overstated matrices negatively predicted actual performance, $B = -.04$, $SE = .01$, $p = .003$, 95% CI $[-.07, -.01]$.

Unethical behavior as a consequence of preperformance hormones. We used hierarchical negative binomial regression to model the frequency of cheating as a function of hormonal predictors. The first model tested the relationship between our covariates (actual performance, time of day, age, state anxiety, and trait anxiety) and frequency of cheating (see Model 1 in Table 2). Our second model tested the joint influence of testosterone and cortisol on cheating: we found a significant preperformance Testosterone $\times$ Cortisol interaction, $B = .25$, $SE = .12$, $p = .04$, 95% CI $[.01, .50]$ (see Model 2 in Table 2), indicating that the association between preperformance testosterone levels and unethical behavior was moderated by cortisol concentrations.2

A simple-slopes analysis replicated the previous finding that the association between preperformance testosterone and unethical behavior is amplified by the presence of high levels of preperformance cortisol. Under low cortisol ($-1 SD$), testosterone did not predict unethical behavior, $B = .75$, $SE = .42$, $p = .08$. However, testosterone significantly predicted unethical behavior under high cortisol ($+1 SD$), $B = 1.73$, $SE = .40$, $p < .001$. Figure 2 depicts this relationship.

Acute changes in cortisol and negative affect after cheating. To test our second hypothesis—the existence of a dose–response relationship between cheating and reductions in physiological and psychological distress—we analyzed the association between cheating and cortisol change (preperformance to postperformance). In support of the hypothesis, we found a significant negative correlation, $r = -.27$, $p = .003$. Among those who cheated, the more a participant cheated, the greater the participant’s decrease in cortisol, $B = -.06$, $SE = .02$, $p = .005$, 95% CI $[-.11, -.02]$, controlling for preperformance hormone levels, trait anxiety, time of day, age, and actual performance.

Next, we examined the effect of cheating on self-reported change in state negative affect. We found a significant negative correlation: The more a participant cheated, the greater the decrease in negative affect, $B = -.07$, $SE = .02$, $p = .003$, 95% CI $[-.11, -.03]$, controlling for preperformance hormone levels, trait anxiety, time of day, age, and actual performance.

General Discussion

Our two studies supported two previously untested but potentially important hypotheses: (a) endocrine activity possesses a regulatory function in unethical behavior, and (b) cheating lowers cortisol and negative affect.

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1 We used negative binomial regressions in the main study instead of Poisson regressions in the pilot study to account for overdispersion in our outcome measure. For both studies, we also report the additional ordinary least squares (OLS) regression analyses in Table S2 and Table S4 in the online supplemental materials.

2 We found it interesting that change in cortisol levels between pre- and postperformance did not interact with preperformance testosterone to predict cheating ($p > .48$). Thus, stress—as indicated by preperformance cortisol—moderated testosterone, but change in stress—as indicated by change in cortisol—did not.
The first hypothesis predicted the joint influence of two behaviorally important hormones, testosterone and cortisol, on unethical behavior. Over two studies, this hypothesis was supported. Elevated concentrations of cortisol and testosterone encouraged cheating (false reporting on a performance test). Neither hormone without the other predicted cheating behavior (a main effect of testosterone in the pilot study was qualified by the Testosterone Cortisol interaction; see Figure 1).

The second hypothesis, that a dose–response relationship existed between cheating and reductions in physiological and psychological distress, was also supported. The more one cheated, the greater the hormonal and emotional rewards of cheating, as indicated by reductions in cortisol and negative affect, respectively. To the best of our knowledge, neither effect—neither the joint influence of testosterone and cortisol on cheating nor the role of cheating in reducing cortisol and negative affect—have been reported previously.

These findings contribute to a growing body of evidence that has been helping to sort out inconsistencies plaguing the literature on testosterone and social-approach behaviors. That said, our primary finding—that testosterone levels predict unethical behavior only under high cortisol levels—contrasts with the majority of dual-hormone evidence, which shows high cortisol levels blocking the association between testosterone and approach behaviors. At this early stage in the research program, it is impossible to puzzle out with any confidence a solution explaining this difference. With this caveat in place, however, one possible answer may be found in the evidence supporting our second hypothesis, namely, that cheating reduces stress and negative affect. If this effect replicates, then it seems plausible that those individuals most likely to cheat are motivated by a desire to reduce an aversive emotional state, which in this case is marked by an elevated cortisol concentration. Even if this dose–response relationship is replicated, questions remain unanswered as to why other experimental designs produced the more typical high-testosterone, low-cortisol interaction, whereas ours didn’t.

The findings from the main study provided preliminary insight into the physiological and affective consequences produced by unethical behavior. Whereas past research has focused largely on how unethical behavior triggers negative affect such as guilt and shame (Tracy & Robins, 2006), our research built on findings into the physiological and affective consequences produced by unethical behavior. Whereas past research has focused largely on how unethical behavior triggers negative affect such as guilt and shame (Tracy & Robins, 2006), our research built on findings.

Table 2

<table>
<thead>
<tr>
<th>Predictor variables</th>
<th>Step 1</th>
<th>Step 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Actual performance</td>
<td>−.11*</td>
<td>−.14**</td>
</tr>
<tr>
<td>Time of day</td>
<td>2.81</td>
<td>.33</td>
</tr>
<tr>
<td>Age</td>
<td>−.06</td>
<td>−.10</td>
</tr>
<tr>
<td>Trait anxiety</td>
<td>−.19</td>
<td>−.40</td>
</tr>
<tr>
<td>State anxiety at baseline</td>
<td>.21</td>
<td>.33</td>
</tr>
<tr>
<td>Preperformance testosterone</td>
<td>−.06</td>
<td>−.10</td>
</tr>
<tr>
<td>Preperformance cortisol</td>
<td>.25</td>
<td>.25</td>
</tr>
<tr>
<td>Interaction: Testosterone × Cortisol</td>
<td>.25</td>
<td>.25</td>
</tr>
<tr>
<td>N</td>
<td>110</td>
<td>110</td>
</tr>
<tr>
<td>Wald $\chi^2$</td>
<td>9.82</td>
<td>29.54</td>
</tr>
<tr>
<td>Prob $&lt; \chi^2$</td>
<td>.08</td>
<td>&gt;.001</td>
</tr>
</tbody>
</table>

Note. Prob = probability.

*p < .05. **p < .01. ***p < .001.

Figure 2. Effects of preperformance testosterone levels and preperformance cortisol levels on frequency of cheating, main study.
In part because of the novelty of these findings, limitations exist which prohibit a clear and unambiguous interpretation of the results. One of the most significant of these limitations precluded us knowing exactly what motivational or emotional state preperformance cortisol or testosterone levels were measuring. Participants were unaware at the time the first saliva sample was taken that they were about to engage in a task that provided the opportunity to cheat; thus, it is unlikely that pretask cortisol or testosterone levels reflected participants’ stress about getting caught cheating. It is possible, however, that fear of performing poorly on the task, and/or of appearing incompetent in front of the experimenters may have contributed to elevated cortisol and testosterone levels. Future research might address this by comparing hormone levels prior to engaging in an ability-based task, such as the one used in the current studies, with a task that also provides the opportunity to cheat but is not perceived by participants to be linked to one’s ability (e.g., die-rolling task under the cup, Fischbacher & Föllmi-Heusi, 2013; Shalvi, Handgraaf, & De Dreu, 2011).

In closing, we believe that our findings offer exciting, albeit preliminary evidence fundamentally challenging existing models of ethical decision making by incorporating the role of the endocrine system. People are motivated to strike a balance between maximizing self-interest by taking undeserved money and maintaining a positive self-concept for a financial gain, potentially due to the experimenters may have contributed to elevated cortisol and testosterone levels. Future research might address this by comparing hormone levels prior to engaging in an ability-based task, such as the one used in the current studies, with a task that also provides the opportunity to cheat but is not perceived by participants to be linked to one’s ability (e.g., die-rolling task under the cup, Fischbacher & Föllmi-Heusi, 2013; Shalvi, Handgraaf, & De Dreu, 2011).

References


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