Social Neuroscience

ISSN: 1747-0919 (Print) 1747-0927 (Online) Journal homepage: http://www.tandfonline.com/loi/psns20

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To cite this article: Keith M. Welker, Ashlin R. K. Roy, Shawn Geniole, Shinobu Kitayama & Justin M. Carré (2017): Taking risks for personal gain: An investigation of self-construal and testosterone responses to competition, Social Neuroscience, DOI: 10.1080/17470919.2017.1407822

To link to this article: https://doi.org/10.1080/17470919.2017.1407822

Accepted author version posted online: 20 Nov 2017.
Published online: 24 Nov 2017.

SOCIAL NEUROSCIENCE, 2017
https://doi.org/10.1080/17470919.2017.1407822

Routledge
Taylor & Francis Group

ARTICLE

Taking risks for personal gain: An investigation of self-construal and testosterone responses to competition

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ABSTRACT

Recent research on testosterone and risk-taking behavior is beginning to focus on the role of context-dependent changes in testosterone. Extending this research, our study investigated the association between testosterone reactivity to competitive outcomes and risk-taking in the context of a video game based competition. The study also examined whether self-construal moderated this relationship. Results indicated that a rise in testosterone during competition did not predict subsequent risk-taking behavior. However, a rise in testosterone during competition predicted subsequent risk-taking behaviors within winners with independent self-construals. Nevertheless, results did not reveal an association between basal testosterone and risk-taking, nor did competitive outcomes modulate a differential testosterone response. Overall, we treat these findings as preliminary, as there were multiple analyses conducted and effect sizes were relatively small. We discuss these results in the context of recent animal findings that testosterone facilitates success at future competitions after winning a competition, as well as recent research suggesting self-construal moderates associations between testosterone and aggression.

ARTICLE HISTORY
Received 21 March 2017
Revised 20 October 2017
Published online 27 November 2017

KEYWORDS
Testosterone; competition; risk-taking; self-construal

Emerging research suggests that testosterone responses to competition may lead to elevated risk-taking behavior, including reactive aggression (e.g., Apicella et al., 2014; Carré, Campbell, Lozoya, Goetz, & Welker, 2013). Moreover, previous research suggests that this effect may be more pronounced within men with more independent self-construals, or an individualistic view or concept of the self (Welker, Norman, Goetz, Kitayama, & Carré, 2017). In general, aggression may be seen as a form of risk-taking behavior, as it carries the risk of a potential loss of status, trust, or reputation, with the possibility of incarceration, retaliation, or harm to the self and others. Given the pattern of previous findings, we might expect that the testosterone responses to competition might predict impulsive, but non-aggressive forms of risk-taking behavior within men that have more independent self-construals. Thus, in the present work, we examined whether testosterone responses to interpersonal competition (i.e., competition amongst individuals rather than between groups) predicted men's risk-taking behavior, and whether these effects were moderated by self-construal.

Risk-taking and testosterone

Risk-taking behaviors have a variety of definitions depending on the focus and discipline, but broadly defined, they invite opportunity to obtain a form of reward at the potential cost of danger, harm, or loss of resources (See Leigh, 1999; Lejuez et al., 2002; for reviews). The implications of risk-taking permeate many areas of human functioning, well-being, and behavior, including psychopathology (e.g., Reddy et al., 2014), behavioral economics (e.g., Ioannidou, Ongena, & Peydró, 2015), sexual behaviors (e.g., Hoyle, Fejfar, & Miller, 2000), and substance use (e.g., Verdejo-García, Lawrence, & Clark, 2008), highlighting the potential costs, possible rewards, and the pervasiveness of risky decision making. Although risk-taking is relatively common, it is not always easy to predict when people will make risky-decisions. Psychologists have sought to explain risk-taking behavior through many individual differences and contexts, including reward-sensitivity (e.g., Steinberg, 2007), impulse control (Baumann & Odum, 2012; Stanford, Greve, Boudreauz, Mathias, & Brumbelow, 1996), sensation-seeking (e.g., Greene, Krcmar, Walters, Rubin, & Hale, 2000), and social status (Wilson & Daly, 1985, 1997), among others.

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Supplemental data can be accessed here.

One potential biomarker of risk-taking propensity is testosterone (Apicella et al., 2008; Coates & Gurnell, 2017; Coates & Herbert, 2008; Sapienza, Zingales, & Maestripieri, 2009; Schipper, 2012; Stanton, Liening, & Schultheiss, 2011; Vermeersch, T'sjoen, Kaufman, & Vincke, 2008). A product of the hypothalamic-pituitary-gonadal (HPG) axis, testosterone is a steroid hormone implicated in the formation of the genitalia and central nervous system in perinatal development (Arnold & Breedlove, 1985; Arnold & Gorski, 1984; Chura et al., 2010). Testosterone plays a critical role modulating male laryngeal growth, muscle growth, body hair, and bone density in puberty (Molina, 2013). In addition, testosterone is thought to influence, and be influenced by, social behaviors (Mazur & Booth, 1998; Wingfield, Hegner, Dufty, & Ball, 1990).

Despite growing interest in this topic, research supporting an association between basal testosterone and risk-taking behavior in humans has been relatively mixed. Some studies find that higher endogenous levels of testosterone in adulthood are associated with increased risk-taking behaviors (Apicella et al., 2008; Coates & Herbert, 2008; Vermeersch et al., 2008). However, other researchers have reported elevated risk-taking in those with only very high or low testosterone (Stanton et al., 2011), whereas others have reported a positive association for women, but no association within men (Sapienza et al., 2009). Further, others have found that testosterone is linked to risk-proclivity for gains but not losses (Schipper, 2012) or when basal cortisol is low but not high (Mehta, van Son et al., 2015).

However, instead of focusing on basal or trait-like neuroendocrine function, more state-based testosterone dynamics might more robustly predict risk-taking behavior (e.g., Apicella et al., 2014). Testosterone often rises in response to several different types of social situations, including competition, where it often remains elevated in winners relative to losers (Archer, 2006; Geniole, Bird, Ruddick, & Carré, 2017). These changes in testosterone are hypothesized to fine-tune behavior according to changes in the social environment (see Carré & Olmstead, 2015, for a review). For instance, in response to competition, changes in testosterone reliably predict variability in aggressive behavior (e.g., Carré et al., 2013; Geniole et al., 2017).

Do testosterone responses to competition predict other types of behaviors, namely risk-taking? A recent study has examined the role of testosterone responses to monetary wins and losses, finding that increases in men's testosterone concentrations during interpersonal competition predicted financial risk-taking (Apicella et al., 2014). Specifically, participants won or lost money in a chance-based competition ("rock, paper, scissors"). The researchers reported that men who increased in testosterone during the competition took more risks in a subsequent computerized financial decision-making task, and this effect did not differ between winners and losers (Apicella, personal communication, November 16, 2016).

Self-construal moderations of the associations between testosterone and risk-taking

Despite competitive statuses and contexts, very little is known about the psychological correlates and mechanisms of testosterone on risk-taking behavior. What motivates individuals with elevated testosterone responses or high basal testosterone to take risks in competitive situations? The answer, in part, may lie in the traits and motivations of those who engage in risk-taking behavior or whose testosterone responses are coupled with elevated risk-taking.

Recent work has focused on how self-construal moderates the associations between testosterone and interpersonal aggression. Self-construal, or how individuals construe the self in relation to others, was initially coined to describe differences in self-concept between people in individualistic and collectivistic cultures (Markus & Kitayama, 1991). Furthermore, interdependent (i.e., collectivistic) people tend to see the self as interconnected with others, whereas more independent (i.e., individualistic) people tend to see the self as independent of others. One major limitation of existing neuroendocrine research is that it has neglected to account for cultural factors that influence the link between biological function and behavior. Recent preliminary research on self-construal and testosterone (Welker et al., 2017) has found that testosterone reactivity to competition positively predicts aggression among more independent but not interdependent individuals. On the other hand, basal testosterone was negatively associated with aggression among more interdependent but not independent individuals. Although it is unclear whether testosterone and self-construal would interact similarly to predict more collectivistic or intergroup forms of aggressive behavior, this research suggests that state-like, dynamic HPG axis reactivity to competition might modulate aggression in the independent self, but more stable, resting, and trait-like levels of testosterone might modulate aggression in the interdependent self.

A critical extension of this work is to investigate if self-construal and testosterone dynamics also co-regulate other behaviors often linked to status, impulsivity, or poor self-regulation, in addition to aggression, such as risk-taking. Although research suggests self-construal

might not modulate how testosterone responds to competition (Welker et al., 2017), it may moderate the extent to which testosterone responses to competition influence risk-taking behavior. Considering work on testosterone and power-motive (e.g., Schultheiss, Campbell, & McClelland, 1999; Schultheiss & Rhode, 2002), we previously speculated that testosterone changes in more independent people may modulate status-seeking behaviors through personalized means (e.g., using force or obtaining economic resources) whereas testosterone changes in more interdependent people would prompt more socially oriented status seeking strategies (e.g., prosocial behavior, generosity; Welker et al., 2017).

Other traits may also moderate whether testosterone predicts social behaviors related to status and competition. For example, when trait dominance is high, testosterone predicts increased dominance in men's mating behaviors (Slatcher, Mehta, & Josephs, 2011) and exogenous testosterone administrations promote aggressive behavior in men (Carré, et al., 2017) and alter competitive decision making in women (Mehta, Welker, Zilioli, & Carré, 2015). Additionally, testosterone administration in more impulsive men are more likely to increase aggressive behavior (Carré et al., 2017). Testosterone responses to competition are also associated with aggression in men with low anxiety (Norman, Moreau, Welker, & Carré, 2015). Coupled with recent work examining self-construal and testosterone (Welker et al., 2017), this emerging work suggests that testosterone predicts status-relevant social behavior in those with more independent self-construals, low anxiety, low control over impulses, and high dominance. However, this body of work is rather preliminary, and it is currently unknown whether the same traits moderate associations between testosterone dynamics and risk-taking behavior, specifically.

Overview of the current research

In the present study, we extended research on the neuroendocrinology of risk-taking for personal financial gain, competition, and self-construal in several important ways. First, using a relatively large sample (N = 165), we investigate whether changes in testosterone concentrations during competition predict subsequent risk-taking behavior (see Apicella et al., 2014), and whether competition outcome moderates the relationship between testosterone responses to competition and risk-taking. Also, we built upon recent evidence (see Welker et al., 2017) by examining the extent to which individual differences in trait self-construal moderate the relationship between testosterone (baseline, and reactivity to competition) and risk-taking.

We tested these possibilities in a relatively large sample of men who were assigned to win or lose a competitive game. Because we have previously used this dataset to replicate the effect of basal testosterone and cortisol on risk-taking (Mehta, van Son et al., 2015, Study 2), we do not focus on this association here, unless otherwise specified. Instead, we examine three key aims: First, we investigated the association between testosterone reactivity to wins and losses and risk-taking (Aim 1). Then, we evaluated the role of self-construal in moderating the effect of testosterone reactivity on risk-taking (Aim 2). Furthermore, in an effort to conceptually replicate previous work (Welker et al., 2017), we examined whether self-construal moderated the effects of competitive outcomes on testosterone reactivity (Aim 3). Finally, we aimed to explore whether several other previously identified moderators (dominance, impulse control, and anxiety) also influence the extent to which testosterone reactivity predicts risk-taking.

Methods

Participants and design

Participants were 165 male university psychology students (M_{age} = 20.64, SD = 3.00) that were randomly assigned to a win (47.8%) or lose (52.2%) a competitive game. The sample was rather diverse (38.2% Caucasian, 19.4% Black, 18.1% Asian, 4.8% Latin America, .6% Native American, and 18.8% Other). Participants were recruited through an online psychology subject pool and all participants were compensated by receiving partial course credit and being entered in a raffle for a 150 dollar gift card. Using a two-tailed alpha of .05, this sample size provides substantial power for detecting large effect sizes ($|r|$ = .50, power > .99), and medium effect sizes ($|r|$ = .30, power = .98), and low power for detecting small effect sizes ($|r|$ = .10, power = .25).[1]

Results from this dataset have previously examined the roles of basal testosterone and cortisol (Mehta, van Son et al., 2015) and facial structure and status (Welker, Goetz, & Carré, 2015) predicting risk-taking behavior.

[1]Alternatively, one could conduct power analyses with effect size for the T reactivity x self-construal interactions reported by Welker and colleagues (2017). Using a generic effect size of r and a two-tailed alpha of .05, we have 49% power (power = .49) to assess effects of the magnitude of the T-reactivity x Self-construal effect size reported by the integrated data analysis of Welker and colleagues (2017, partial r = .15). We also provide power analyses for our 3-way interaction in our linear multiple regression models for this effect size. For this effect size in a 7-predictor moderated regression model (f^2 = .023), our sample size achieved 49% statistical power (power = .49).

Thus, we did not examine or present research involving the research questions examined in those papers.

Materials and procedure

Before the session, participants were asked to not exercise on that day, eat one hour before the study, or drink anything other than water one hour before the study. On the way to the laboratory, participants were asked to rinse their mouths with water from a drinking fountain to prevent food particles from contaminating the samples. In the lab, participants first completed the consent form and a battery of pretest personality and demographic questionnaires. Then, participants played a competitive video game with a rigged outcome of victory or defeat, with saliva samples taken pre and post-game. After this, participants completed a post-task questionnaire and then completed a risk-taking task. A full list of all self-report measures in the full dataset this paper originates from is presented in the supplemental materials.

Pre-experimental questionnaire

First, participants completed a demographic questionnaire assessing their age, gender, and race. Then, participants completed a battery of personality measures including a self-report measure of self-construal. Participants also completed measures of anxiety, dominance, and impulse-control. The self-construal scale (Singelis, 1994) is a 30-item measure consisting of seven-point Likert-type items measuring the extent to which participants hold interdependent and independent construals (1 = Strongly Disagree, 7 = Strongly Agree). Similar to previous work (e.g., Aaker & Williams, 1998; Zhang, Feick, & Price, 2006), we reverse-scored interdependence scales (Cronbach's α = .80) compared to independence scores (Cronbach's α = .78), averaging them to create one measure of self-construal where more independent scores had higher values and more interdependent scores had lower values.[2]

Saliva samples

Saliva samples were obtained from participants immediately before and approximately 5–10 minutes after playing the XBOX 360 Kinect volleyball game. Samples were taken between 11am and 5pm to minimize diurnal variation in testosterone. To provide saliva, participants passively drooled through a straw into a polystyrene tube. Saliva samples were frozen at −20°C until shipped to Nipissing University (North Bay, Ontario), where they were frozen at −60°C until assayed. At Nipissing University, saliva samples were assayed for testosterone in duplicate using immunoassay kits from DRG International. The intra-assay and inter-assay coefficients of variation were below 10%.

Competitive outcome manipulation

Similar to Carré and colleagues (2013), participants played an XBOX 360 Kinect game of volleyball set to either the highest difficulty (loss) or the lowest difficulty (win), unbeknownst to the players. In this game, the XBOX 360 Kinect device could sense the motions of participants' movements, allowing them to jump, serve, hit, and spike the ball throughout the match. In the easiest condition, participants achieved a series of victories, self-reporting an average of 83.77% of rounds won (SD = 21.89%), and all participants in the win condition won at least one round. On the other hand, no participants in the loss condition won any of the rounds, according to our research assistants (with one exception mentioned in our preliminary analyses), although a minor amount of participants self-reported winning some rounds (M = 4.32% of rounds won, SD = 15.03%). Nevertheless, the difference in self-reported victories between these conditions was substantial ($t(154) = -26.58$, $p < .001$). Participants played the video game for 15–20 minutes before being stopped by the researcher to complete the post-task saliva sample, post-experimental questionnaire, and the Balloon Analog Risk Task (described below).

Collective and individual competition manipulations

Participants in the team condition were paired with a male confederate, who appeared to be another participant scheduled for the study. The confederate and participant were told by the experimenter that they were part of a team and the goal of this task is to work together to collectively win the video game. When playing as a team, the Kinect sensor read both of the participants' movements as they stood next to each other and played the game on the same television screen. Participants in the individual condition played the same game – but without a partner. This experimental manipulation (team vs. individual) was not associated with testosterone (concentrations and changes),

[2]Other researchers have calculated this score by subtracting the sum of the interdependence scale from the independent scale (e.g., Kitayama et al., 2014). Although we did not happen to use that approach in this paper, since the subscales have equal items, this approach is a linear transformation of reverse-scoring the interdependent items and computing an average. Choosing the alternate approach does not change any inferential statistics and conclusions in this paper.

risk-taking, or any of the individual differences measures in this study (|*r*|s ≤ .11, *p*s ≥ .166). Unless otherwise noted, the team vs. individual manipulation did not moderate any of our presented results (*p*s ≥ .144).

Balloon risk analog task

Participants then completed a widely-used measure of risk taking, the Balloon Analogue Risk Task (BART; Lejuez et al., 2002). The BART has been used by a wide variety of researchers to predict how performance on the task is linked to dispositional anxiety (Maner et al., 2007), smoking (Lejuez et al., 2003), and risk-taking behaviors in adolescents (Lejuez et al., 2007). In this version of the task, participants accumulated money points by pumping up 30 virtual balloons. Participants were informed that there were 30 balloons in total, and that each balloon pump earned them $.05 of token "money points," and for every $.10 earned, participants earned a raffle ticket for a 150 dollar gift card. Each balloon had a maximum threshold of pumps it could reach before it exploded, ranging between 1 to 30 pumps. If a balloon exploded, all points were lost from that specific balloon. Participants also had an option to save the points from a balloon, provided that the balloon has not yet exploded, and move on to pumping the next balloon in the sequence. Altogether, when performing this task, participants must make a decision to engage in risky behavior with each button press, as the balloon has a chance to explode with each press. Consistent with previous work (e.g., Maner et al., 2007), the average number of pumps (M = 9.80, SD = 3.24, ranging from 1.65 to 19.27) from unexploded balloons served as participants' index of risky behavior.

Other potential moderators

Dominance

To measure dominance, participants completed the 10-item trait dominance scale from the international personality item pool (IPIP; Goldberg et al., 2006), which is part of a publicly available stand-in version of the Gough California Psychological Inventory (Gough & Bradley, 1996). Sample items for the dominance scale (Cronbach's α = .86) included "I try to surpass others' accomplishments" and "I try to outdo others." This scale used a 7-point Likert scale (1 = Disagree Strongly, 7 = Agree Strongly).

Impulse control

To assess impulse control, we had participants complete the 10-item impulse-control scales provided by the IPIP (Goldberg et al., 2006). Sample items for the impulse control scale (Cronbach's α = .85) included "I am able to control my cravings" and "I do things I later regret" (reversed). Similar to the dominance scale, this scale also used a 7-point Likert scale (1 = Disagree Strongly, 7 = Agree Strongly).

Anxiety

As a measure of anxiety, participants completed the Spielberger Trait Anxiety Scale (STAI; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983). This scale asked participants to rate how often they generally feel 20 different statements on a 4-point scale (1 = almost never, 4 = almost always). Example items included " I worry too much over something that really doesn't matter" and "I feel nervous and restless" (Cronbach's α = .90).

Statistical analyses

Results were predominantly analyzed using moderated multiple regression analysis. As many of our aims were interactive hypotheses, by nature, our models regressed outcomes (risk-taking and testosterone responses) on mean-centered predictors and their cross-product(s). Simple slopes were assessed using PROCESS (Hayes, 2013), an SPSS plug-in that computes simple slopes for a variety of configurations of interactive and mediational regression models. Testosterone changes were computed by regressing post-competition concentrations on pre-competition conditions and saving the unstandardized residuals (e.g., Carré et al., 2013). To reduce the influence of outliers, all outliers were Winsorized to ±3 SDs. Plots of the interaction terms with confidence bands were generated in R software (R Development Core Team, 2009) using the *visreg* package (Breheny & Burchett, 2012).

Results

Preliminary analyses

One participant did not play the video game competition due to arthritis, another four participants failed to win any of the rounds in the win condition, and another participant discovered a glitch that allowed him to win all of the rounds played in the defeat condition. These participants (N = 6) were removed from the analyses leaving the analyzed sample size at 159 participants. Intercorrelations and descriptive statistics for all study variables are presented in Table 1. As reported by Mehta, Welker and colleagues (2015) using this same

Table 1. Correlations and descriptive statistics for study variables.

		1	2	3	4	5	6	7	M	SD
1.	ln(T1)	–							4.57	.40
2.	ln(T2)	.79***	–						4.51	.42
3.	T1 (pg/mL)	.97***	.78***	–					103.81	40.62
4.	T2 (pg/mL)	.78***	.94***	.82***	–				98.31	40.20
5.	TR	−.01	.55***	.00	.58**	–			−.12	21.71
6.	Risk-taking	.04	.06	.02	.02	.07	–		9.80	3.24
7.	SCNST	.08	.07	.04	.01	−.05	.01	–	4.18	.43

SCNST = Self Construal, TR = Testosterone Reactivity. The mean of testosterone residuals is nonzero because of Winsorized values, T1 and T2 represent testosterone measured at times 1 and 2, respectively.
†$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$.

dataset, basal testosterone was not significantly associated with risk-taking behavior ($r = .04$, $p = .662$).[3]

Aim 1: evaluate the role of testosterone reactivity to wins/losses on risk-taking

To evaluate Aim 1, we regressed risk-taking behavior on competitive outcome, testosterone reactivity, and their interaction. The results of this regression are presented in Table 2, including confidence intervals and effect sizes. We report slopes and inferential statistics in the main text for concision. Although there were no significant main effects of outcome and testosterone reactivity (*p*s ≥.212), there was a significant outcome X testosterone reactivity interaction ($B = .03$, $t(152) = 2.30$, 95% CI [.00, .05], $p = .023$, partial $r = .18$, See Figure 1). This moderation was characterized by a significant positive association between testosterone reactivity and risk-taking in winners ($B = .04$, $t(152) = 2.42$, 95% CI [.01, .08], $p = .017$, partial $r = .19$), but not losers ($B = -.01$, $t(152) = -.73$, 95% CI [-.04, .02], $p = .468$, partial r = -.06).

Aim 2. evaluate self-construal moderating the effects of t-reactivity on risk-taking

We then ran another moderated regression analysis to determine whether self-construal moderated the association between testosterone reactivity and risk-taking established in Aim 1. This model is summarized in Table 3, which includes confidence intervals and effect sizes. Self-construal significantly moderated the testosterone reactivity X outcome interaction found in Aim 1 (3-way interaction: $B = .07$, $t(148) = 2.27$, 95% CI [.01, .14], $p = .025$, partial $r = .18$). The pattern of this interaction is displayed in Figure 2. In particular, the conditional testosterone reactivity X outcome interaction reported in Aim 1 was significant when participants were more independent ($B = .06$, $t(148) = 3.27$, 95% CI [.03, .10], $p = .001$, partial $r = .26$) rather than interdependent ($B = .00$, $t(148) = .03$, 95% CI [-.04, .04], $p = .976$, partial $r = .00$). Specifically, in independent men (Self-construal +1 SD), simple slopes analysis revealed that testosterone reactivity was positively associated with risk-taking in winners ($B = .08$, $t(148) = 2.82$, 95% CI [.03, .14], $p = .006$, partial $r = .23$), but in a marginally significant, negative direction in losers ($B = -.05$, $t(148) = -1.76$, 95% CI [-.10, .01], $p = .080$, partial $r = .14$). The simple slopes in interdependent (Self-construal −1 SD) participants were non-significant (*p*s ≥ .967).

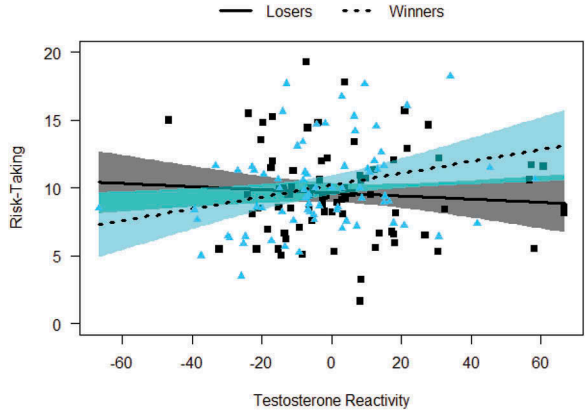

Figure 1. Risk-taking behavior as a function of outcome and testosterone reactivity.

Note: Slopes are plotted separately for winners and losers.

Table 2. Moderated regression analysis for risk-taking behavior, competitive outcome, and testosterone reactivity.

	B	*SE*	*t*(152)	*p*	95% CI LB	95% CI UB	Partial *r*
OC	.28	.26	1.08	.282	−.23	.79	.09
TR	.02	.01	1.25	.212	−.01	.04	.10
OC X TR	**.03**	**.01**	**2.30**	**.023**	**.00**	**.05**	**.18**

TR = Testosterone Reactivity, OC = Outcome. Model $R^2 = .04$.

[3]Team condition significantly moderated a 3-way outcome X basal testosterone X self-construal interaction (i.e., a 4-way interaction). Although our sample is very underpowered to examine 4-way interactions and there are difficulties interpreting 4-way interactions, we nevertheless explored this interaction in our supplemental materials for interested readers.

Table 3. Moderated regression analysis for risk-taking behavior as a function of self-construal, competitive outcome, and testosterone reactivity.

Model/Variable	*B*	*SE*	*t*	*p*	95% CI LB	95% CI UB	Partial *r*
Self-Construal Moderation (R^2 = .08, df = 148)							
OC	.28	.26	1.09	.278	−.23	.79	.09
TR	.01	.01	.59	.558	−.02	.03	.05
SCNST	.39	.62	.63	.528	−.83	1.61	.05
OC X TR	**.03**	**.01**	**2.61**	**.010**	**.01**	**.06**	**.21**
TR X SCNST	.02	.03	.54	.59	−.05	.08	.04
OC X SCNST	.41	.62	.67	.51	−.81	1.63	.05
OC X SCNST X TR	**.07**	**.03**	**2.27**	**.025**	**.01**	**.14**	**.18**

SCNST = Self Construal, TR = Testosterone Reactivity, OC = Outcome.

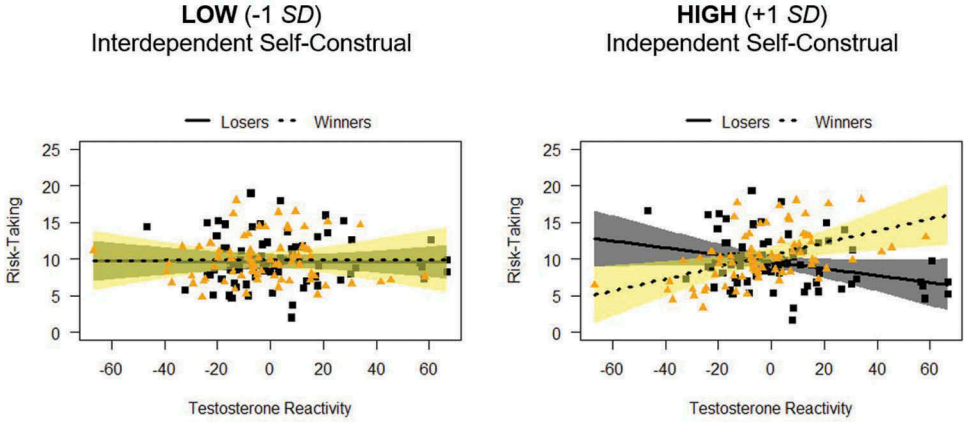

Figure 2. Risk-taking behavior as a function of testosterone reactivity, competitive outcome, and self-construal.

Aim 3. evaluate testosterone reactivity to competition and moderation by self-construal

Next, we evaluated the effect of winning vs. losing on testosterone responses and then ran another series of moderated regression analyses to see if self-construal moderated the effects of competitive outcomes on testosterone reactivity. Although nonsignificant ($t(154) = 1.77$, 95% CI [-.70, 12.95], $p = .078$, $d = .29$), losers showed slightly greater testosterone reactivity residuals ($M = 2.82$, $SD = 22.49$) than winners ($M = -3.30$, $SD = 20.51$). This finding is in contrast with previous research suggesting a small, yet differential effect of winning a competition increasing testosterone relatively to losing a competition (Geniole et al., 2017). Following this, we also examined whether self-construal moderated testosterone responses to competitive outcomes, using a moderated regression analysis with a two-way interaction (outcome X self-construal) and main effects predicting testosterone residuals. Self-Construal did not significantly moderate the effect of competition outcomes on testosterone reactivity ($B = 2.53$, $t(152) = .62$, 95% CI [−5.51, 10.56], $p = .536$, partial $r = .05$).[4]

Analyses with dominance, impulse control, and anxiety as moderators

We examined whether three other individual differences (anxiety, dominance, and impulse control) might

[4]Because we recorded the amount of money points participants earned in the BART, our analyses invited assessing whether the interaction effects used in Aims 1 and 2 replicated when predicting money points earned on the BART. Money points earned were strongly related to risk-taking behavior ($r = .74$, $p < .001$), although this relationship was curvilinear with some heteroscedasticity (See Supplemental Materials). However, when we ran the models associated with Aims 1 and 2, there was no significant competition outcome X testosterone reactivity interaction predicting money points ($B = .01$, $t(152) = 1.66$, $p = .100$, Aim 1 Model) or a significant three-way outcome X testosterone reactivity X self-construal interaction ($B = .02$, $t(148) = 1.26$, $p = .211$, Aim 2 Model). Although nonsignificant in the Aim 1 Model, the testosterone reactivity X outcome interaction was significant in the Aim 2 model, with a trend hinting at a positive direction between testosterone reactivity and points earned in winners ($B = 1.04$, $t(148) = 1.69$, $p = .093$), but not losers ($B = -.96$, $t(148) = -1.28$, $p = .202$). Altogether, these interaction effects on money points earned were not particularly robust compared to the standard BART measure of risk-taking.

moderate the influence of testosterone reactivity and competitive outcomes on risk-taking. These three models are summarized in Table 4. As is shown, the outcome X testosterone reactivity moderation effect was robust across all individual difference moderation models (*p*s ≤ .043, partial *r*s ≥ .17). However, the individual differences did not significantly moderate the outcome X testosterone reactivity effect in any models. Despite this lack of a significant interaction, the pattern of simple slopes suggested that the Competitive Outcome X Testosterone Reactivity interaction might occur in men with high levels of impulse control and low levels of trait anxiety. We explore these provisional simple slopes analyses below. However, we caution readers that although our sample is relatively large, it is likely underpowered for testing 3-way interactions, which tend to be fairly small in effect size (Aguinis, Beaty, Boik, & Pierce, 2005). Due to the more exploratory nature of these analyses, it is necessary to use some caution and seek to replicate these findings in studies with larger sample sizes. Specifically, these results hint that the association between winners' – but not losers' – testosterone reactivity and risk-taking was specific to men with high impulse control and low trait anxiety. These results are comparable with previous research suggesting testosterone reactivity to competition predicts aggression in low-anxiety men (Norman et al., 2015). However, these results, if more robust, would be inconsistent with recent reports that exogenous testosterone administration increases aggressive behavior in more impulsive men (Carré et al., 2017).

Impulse control

In our model with impulse control, the 3-way impulse control X outcome X testosterone reactivity interaction was nonsignificant (95% CI [-.01, .04], p = .224, partial r = .10), which failed to provide evidence that impulse control altered the presence of the outcome X testosterone reactivity. Despite this, there was a significant conditional outcome X testosterone for people with high levels of impulse control (B = .05, t(148) = 2.46, 95% CI [.01, .10], p = .015, partial r = .20) but not low levels of impulse control (B = .02, t(148) = 1.07, 95% CI [-.02, .05], p = .286, partial r = .09). Specifically, simple slopes revealed testosterone was reactivity was positively associated with risk-taking behavior in high impulse control men who won (B = .10, t(148) = 2.59, 95% CI [.02, .17], p = .010, partial r = .21), but was not associated with risk-taking behavior in high-impulse control men who lost (B = -.01, t(148) = -.39, 95% CI [-.05, .03], p = .700, partial r = -.03). The simple slopes of this interaction are presented in Figure 3 (top portion).

Table 4. Moderated regression analysis for risk-taking behavior as a function of individual differences, competitive outcome, and testosterone reactivity.

					95% CI		
Model/Variable	*B*	*SE*	*t*	*p*	LB	UB	Partial *r*
Impulse control Moderation (R^2 = .09, df = 148)							
OC	.37	.26	1.43	.156	−.14	.89	.12
TR	.02	.01	1.63	.105	−.00	.05	.13
IC	.47	.25	1.85	.067	−.03	.96	.15
OC X TR	**.04**	**.01**	**2.73**	**.007**	**.01**	**.06**	**.22**
TR X IC	.02	.01	1.55	.124	−.01	.05	.13
OC X IC	.38	.25	1.51	.134	−.12	.88	.12
OC X IC X TR	.02	.01	1.22	.224	−.01	.04	.10
Anxiety Moderation (R^2 = .06, df = 148)							
OC	.23	.26	.88	.381	−.29	.75	.07
TR	.02	.01	1.46	.146	−.01	.04	.12
ANX	−.13	.55	−.24	.810	−1.21	.95	−.02
OC X TR	**.03**	**.01**	**2.04**	**.043**	**.00**	**.05**	**.17**
TR X ANX	−.01	.03	−.19	.850	−.06	.05	−.02
OC X ANX	−.17	.55	−.31	.757	−1.25	.91	−.02
OC X ANX X TR	−.04	.03	−1.40	.163	−.10	.02	−.11
Dominance Moderation (R^2 = .06, df = 148)							
OC	.30	.26	1.16	.248	−.21	.82	.09
TR	.01	.01	1.08	.282	−.01	.04	.09
DOM	−.28	.24	−1.14	.255	−.76	.20	−.09
OC X TR	**.03**	**.01**	**2.52**	**.013**	**.01**	**.06**	**.20**
TR X DOM	−.01	.01	−1.00	.320	−.04	.01	−.08
OC X DOM	.15	.24	.61	.544	−.33	.63	.05
OC X DOM X TR	−.00	.01	−.02	.986	−.02	.02	−.00

IC = Impulse control, ANX = Trait Anxiety, DOM = Dominance, TR = Testosterone Reactivity, OC = Outcome.

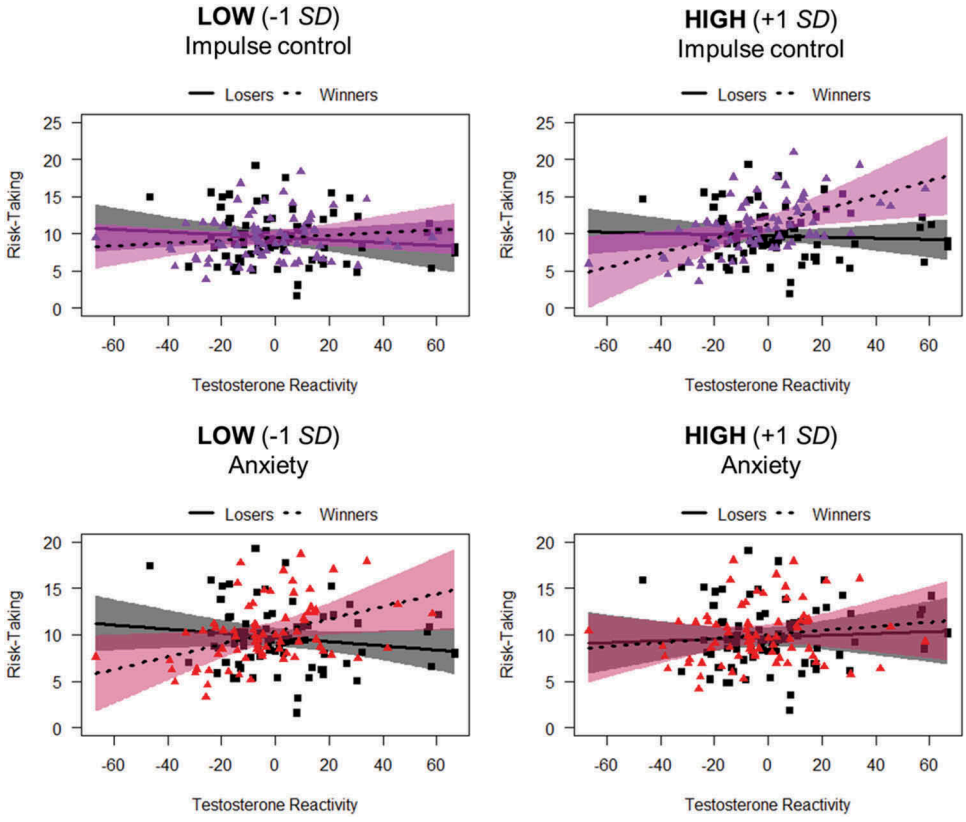

Figure 3. Risk-taking Behavior, Testosterone Reactivity, and Competitive Outcome: moderation by Impulse Control and Anxiety.

Trait anxiety

Although there was no significant 3-way anxiety X outcome X testosterone reactivity interaction (p = .163, partial r = .11, 95% CI [-.10, .02]), the conditional interactions suggested a pattern consistent with a three-way interaction. However, the sample to test this interaction may have been under-powered. Specifically, there was a significant conditional outcome X testosterone reactivity interaction in men with lower anxiety (B = .05, t (148) = 2.47, 95% CI [.01, .08], p = .015, partial r = .20) compared to men with higher anxiety (B = .01, t (148) = .32, 95% CI [-.03, .04], p = .748, partial r = .03). In low anxiety men, winners' testosterone reactivity was positively associated with risk-taking (B = 1.04, t (148) = 2.18, 95% CI [.01, .13], p = .031, partial r = .18), but losers' testosterone reactivity was not (B = -.02, t (148) = −1.17, 95% CI [-.06, .02], p = .245, partial r = -.10; See Figure 3, bottom portion).

Dominance

In our model examining dominance as a moderator, the 3-way dominance X outcome X testosterone reactivity interaction was nonsignificant (p = .986, 95% CI [-.02, .02], partial r = .00). There were no significant conditional outcome X testosterone reactivity interactions in men with either high dominance (p = .094, 95% CI [-.01, .07], partial r = .14) or low dominance (p = .087, 95% CI [.00, .07], partial r = .14), which follows the pattern that the outcome x testosterone interaction was not moderated by dominance.

Effects on testosterone reactivity

The association between basal testosterone and risk-taking was also not moderated by impulse control (B = -.14, t(153) = -.25, 95% CI [−1.31, 1.02], p = .804, partial r = -.02), self-construal (B = −2.34, t(153) = −1.49, 95% CI [−5.44, .77], p = .139, partial r = -.12), anxiety (B = .96, t(153) = .73, 95% CI [−1.63, 3.56], p = .465, partial r = .06), or competition outcome (B = .28, t (153) = .43, 95% CI [−1.01, 1.56], p = .668, partial r = .03). However, trait dominance did significantly moderate the association between basal testosterone and risk taking (B = −1.64, t(153) = −2.56, 95% CI [−2.91, -.38], p = .011, partial r = -.20). Specifically, basal testosterone was positively associated with risk-taking when dominance was low (B = 1.83, t(153) = 2.08, 95% CI [.10, 3.56], p = .039, partial r = .17), but negatively (albeit marginally) when dominance was high (B = −1.79, t (153) = −1.75, 95% CI [−3.81, .23], p = .082, partial r = .14).

Similar to self-construal, none of the individual differences significantly moderated the effect of competition outcomes on testosterone reactivity (*p*s ≥ .249, partial *r*s ≤ .09). It might be of interest to some researchers that although the interaction between outcome and trait anxiety predicting testosterone reactivity was nonsignificant (p = .249, 95% CI [−2.88, 11.02], partial r = .09), the simple slopes suggested that those with lower levels of anxiety experienced greater declines in testosterone among winners compared to losers (B = −4.88, $t(152)$ = −2.01, 95% CI [−9.68, -.09], p = .046, partial r = .16). The simple slope of outcome was not significant in those with higher trait anxiety (B = -.91, $t(152)$ = -.38, 95% CI [−5.69, 3.87], p = .707, partial r = .03).[5]

Discussion

Overall, these results suggest that men's competition-based testosterone reactivity predicts risk-taking behavior oriented toward personal financial gain in winners. However, in contrast to previous work, there was no association between basal testosterone and risk-taking, and competitive outcomes did not modulate a T response consistent with previous literature. However, previous work on testosterone reactivity and risk-taking has found that young men's testosterone concentrations after interacting with attractive females predict risky behavior (Ronay & von Hippel, 2010). The current research expands upon this work by showing that testosterone reactivity specific to winning rather than losing a competition uniquely predicts risk-taking. One reason for this effect might lie in the "winner effect," or the increased effort (and often success) in winning future competitions or fights after experiencing a victory (Hsu, Earley, & Wolf, 2006). Recent experimental animal research suggests that testosterone responses to victory indeed facilitate this winner effect across several species (Fuxjager, Montgomery, & Marler, 2011; Oyegbile & Marler, 2005). In the context of our study, testosterone responses to victory may have facilitated increasing efforts to win money on the BART through taking greater risks, although our data suggests that these responses did not robustly predict actual money points earned. Future research is needed to experimentally test this possibility, perhaps by pharmacologically manipulating testosterone concentrations.

Another explanation for this interactive effect could involve the traits both associated with a proclivity toward testosterone responses and risk. Testosterone responses to rewarding experiences such as winning have been thought to reflect a hedonic drive or pleasure (see Welker, Gruber, & Mehta, 2015, for a review), and recent work suggests that those who increase in testosterone in response to winning a competition often report enjoying the experience (Mehta, Snyder, Knight, & Lassetter, 2015). Generally, those that both are driven toward reward and experience reward tend to show elevated risk-taking behavior (e.g., Devlin, Johnson, & Gruber, 2015; Isen & Patrick, 1983; Tixier, Hallowell, Albert, van Boven, & Kleiner, 2014; Welker, Gruber et al., 2015; for a review). Animal research suggests testosterone modulates the regions of brain linked to reward such as the mesolimbic dopaminergic system, which includes the ventral tegmental area and nucleus accumbens (Aubele & Kritzer, 2011; Bell & Sisk, 2013; DiMeo & Wood, 2006; Fuxjager et al., 2010; Hernandez et al., 1994; Packard, Cornell, & Alexander, 1997; Packard, Schroeder, & Alexander, 1998). Testosterone administration in humans increases ventral striatal responses to financial reward cues (Hermans et al., 2010; Op de Macks et al., 2011). This heightened reward function by testosterone might also be elevated in those who are more independent, or perhaps independent people find risk-taking more rewarding. For instance, research suggests that priming independent self-construals can increase impulsive consumption (Zhang & Shrum, 2009), suggesting that the elevated reward motivation associated with testosterone increases may be stronger for individuals with more independent self-construals. On the other hand, the costs of risk-taking or other impulsive behaviors such as aggression might be lower for independents than interdependents.

This interactive effect might also be explained by the role of testosterone in how people maintain or protect

[5]We previously examined dual effects of basal cortisol and testosterone on risk-taking within this data (Mehta, van Son et al., 2015 Study 2), as well as the effects of facial width-to-height ratio (fWHR) and status (Welker, Goetz et al., 2015). For robustness, we also examined the analyses for Aims 1 and 2 (Presented in Tables 2 and 3) controlling for basal testosterone, basal cortisol, and a basal testosterone X cortisol interaction term, as well as fWHR, status, and a fWHR status interaction term. With these covariates, the testosterone reactivity x outcome interaction presented in Tables 2 and 3 remained significant in both models (*p*s < .006). Within the analyses in Table 3, the three-way testosterone reactivity X self-construal X outcome interaction became nonsignificant (p = .262), but the general pattern of slopes held. Moreover, the outcome x testosterone reactivity conditional interaction was significant when people were more independent (p = .008) but not more interdependent (p = .266). Although the three-way interaction was attenuated with these covariates, it is important to note that the sample size of these models dropped (N = 144) in these follow-up analyses due to missing data in the covariates, contributing to decreased statistical power.

social status. Work surrounding the "challenge hypothesis" suggests that nonhuman animals' rises in testosterone facilitate preparing for status contests, which can include both contests to gain status and to maintain or protect status (Wingfield, 1985; Wingfield et al., 1990). In the case of winners in our study, these testosterone rises may serve to maintain status in a future contest such as the risk-taking task in this paper. In those with independent self-construals, the effect may have been more pronounced because risk-taking is a preferred strategy among more independent individuals (e.g., Bao, Zhou, & Su, 2003; Weber & Hsee, 1998). With more interdependence, impulsive behaviors such as risk-taking or aggression may be seen as a failure to regulate emotions and behavior (Cross & Madson, 1997) or challenge the group's interests (Tse, 1996) and may thus jeopardize, rather than promote social standing.

Unlike our study, previous research investigating testosterone changes and risk-taking (Apicella et al., 2014) did not find any interaction between competitive outcomes and testosterone reactivity (C. Apicella, personal communication, November 16, 2016). It is possible that this interaction may have needed a larger sample size to have adequate statistical power or that our study failed to find an effect within losers when it should have (i.e., type II error). Future work is needed to replicate this interaction between testosterone changes and competition outcomes.

Additionally, these results, in combination with other recent findings (Welker et al., 2017) suggest that self-construal may modulate how or whether testosterone predicts behavior. Specifically, Welker and colleagues (2017) reported that testosterone responses to competition predicted aggressive behavior when men had more independent rather than interdependent self-construals. Taking these findings in stride with the current study, testosterone reactivity may generally be more associated with impulsive or risky behaviors in those with a more independent self-construal. More broadly, replicating and extending this work could suggest that the predictions of prevailing neuroendocrine theories of competitive behavior and dominance (Mazur & Booth, 1998; Van Anders, Goldey, & Kuo, 2011) may primarily apply to the independent self.

The current study did not find, however, that testosterone responses to competitive outcomes were moderated by self-construal. Indeed, our study failed to find the typical rise in testosterone in winners and decline in losers reported in previous literature (see Archer, 2006; Geniole et al., 2017; for meta-analyses). However, laboratory-based competitions often show very small "winner-loser" effects in testosterone compared to field-based competitions (Geniole et al., 2017). Because of this, it is perhaps unsurprising that we did not find a significant moderation of this effect by self-construal.

An additional limitation of our study is that it examined risk-taking within an individualistic context (taking risks to win money for oneself). Much of the psychological literature on risk-taking behaviors involves measures of individualistic risk-taking (e.g., Apicella et al., 2008; Lejuez et al., 2002; Stanton et al., 2011), highlighting the need to investigate more collectivistic forms of this behavior, such as taking risks to win money for one's friends, family, or organizations. Complementing our findings with an individual-level risk-taking task, testosterone may modulate more collectivistic risk-taking behaviors in interdependent individuals. However, our findings and those of previous studies on testosterone and risk-taking have failed to converge on a replicable relationship between testosterone and risk-taking, showing somewhat mixed results (Sapienza et al., 2009; Schipper, 2012), main effects of basal testosterone (Apicella et al., 2008; Coates & Herbert, 2008; Vermeersch et al., 2008), nonlinear associations (Stanton et al., 2011), and associations with testosterone reactivity that are not dependent on competitive outcomes (Apicella et al., 2014). Including our study, research has not converged on a consistent relationship between testosterone and risk-taking. Broadly, our findings and previous investigations of testosterone and risk-taking need to be replicated with a variety of risk-taking measures.

Our study did not find substantive evidence that anxiety, dominance, or impulse control modulate how testosterone predicts risk-taking. Although these effects may be type II errors, the pattern of findings does not converge with previous research suggesting that testosterone may promote status/dominance related behavior (in the form of aggression) when impulse control is low and dominance is high (Carré et al., 2017). Furthermore, the finding that basal testosterone was positively associated with risk-taking in low dominance individuals contrasts with other work suggesting testosterone promotes status-oriented behaviors when dominance is high (e.g., Mehta, Welker et al., 2015; Slatcher et al., 2011). Although these findings cast doubt on whether dominance is a key moderator of how testosterone promotes social behavior, dominance may be a moderator of the effects of testosterone on aggression, but not risk-taking.

Future work is needed to improve upon the limitations of this study. One limitation is that the sample was exclusively men. Recent meta-analytic work indicates that there are no sex differences in testosterone

responses to competition (Geniole et al., 2017), suggesting that women's testosterone responses to winning a competition may also predict risk-taking. However, a recent concern raised is that commonly used enzyme-linked immunosorbent assays tend to produce inaccurate estimates of women's testosterone (Taieb et al., 2003; Welker et al., 2016). Future work with female samples using highly accurate measures of testosterone (e.g., mass spectrometry) will help extend this research in women. Additionally, although our study used a relatively large sample, this sample was likely underpowered to detect most interaction effects, such as those investigated in our supplemental analyses. Larger sample sizes are needed to better test more nuanced interactions.

Additionally, our dataset has been used previously to address research questions related to risk-taking behavior (Mehta, van Son et al., 2015, Study 2; Welker, Goetz et al., 2015). Although examining self-construal, testosterone, and risk-taking was the central focus of this paper, we also conducted many secondary analyses for interested readers. These additional analyses inflate the familywise error rate, increasing the likelihood of type I errors or false positive effects. It is important to note that our reported results would not be statistically significant when correcting for these multiple analyses, pointing to the need for researchers to replicate our findings. Indeed, other novel published social neuroendocrine effects, such as the effect of postures on testosterone and risk-taking (Carney, Cuddy, & Yap, 2010) have failed to replicate in larger samples (Smith & Apicella, 2017). Despite having a relatively large sample size, our data were also underpowered for detecting interaction effects of the magnitude published in previous research (Welker et al., 2017). Although more studies are needed to extensively evaluate the magnitude of a testosterone x self-construal interaction, future studies in this area of research will likely benefit from adopting larger sample sizes.

Conclusion

Altogether, this research presents emerging evidence that testosterone responses to competition modulate risky behavior in winners. Beyond competition, testosterone can increase in response to a wide variety of social contexts such as interactions with sexually-attractive people (e.g., Roney, Lukaszewski, & Simmons, 2007; Roney, Mahler, & Maestripieri, 2003) or aggressive provocation (e.g., Carré, Baird-Rowe, & Hariri, 2014). Testosterone responses in each of these contexts might facilitate or reflect increased risky behaviors, such as sexual risk-taking when in a close interaction with someone sexually attractive (e.g., unprotected sex, flirting) or antisocial risk-taking when aggressively provoked (e.g., violence, criminal acts). Research will benefit from moving toward a broad approach to understanding how testosterone responses to a variety of social situations may predict or increase a wide range of risk-taking behavior. Nevertheless, due to the multiple analyses, relatively small effect sizes, and lack of a replication study, we treat these data as preliminary and recommend that others replicate these findings.

Acknowledgments

We thank Shyneth Galicia, Jordan Liphardt, Elliana Lozoya, Nicholas Jones, and Brian Tyminsky for their assistance with data collection, as well as Benjamin Moreau for help with salivary assays. Funding of this research was supported by faculty start-up funds at Wayne State University and Nipissing University, and a Thomas C. Rumble Graduate Fellowship at Wayne State University. JMC is funded by a Natural Sciences and Engineering Research Council of Canada (NSERC) Discovery Grant and by a Northern Ontario Heritage Fund Corporation (NOHFC) grant.

Contributions

KMW designed the study, collected the data, performed the analyses, and drafted and revised the paper. AR, SG, and SK edited and revised the paper. JMC oversaw the hormone assays and edited and revised the paper.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

This work was supported by the Natural Sciences and Engineering Research Council of Canada [1502]; Northern Ontario Heritage Fund Corporation; Wayne State University.

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