The Causal Effect of Testosterone on Men's Competitive Behavior is Moderated by Basal Cortisol and Cues to an Opponent's Status: Evidence for a Context-Dependent Dual Hormone Hypothesis

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Abstract

Testosterone has been theorized to direct status-seeking behaviors, including competitive behavior. However, most human studies to date have adopted correlational designs, and findings across studies are inconsistent. This experiment (n = 115) pharmacologically manipulated men's testosterone levels prior to a mixed-gender math competition and examined basal cortisol (a hormone implicated in stress and social avoidance) and context cues related to an opponent's perceived status (an opponent's gender or a win/loss in a prior competition) as factors that may moderate testosterone's impact on competitive behavior. We test and find support for the hypothesis that testosterone given to low-cortisol men evokes status-seeking behavior, whereas testosterone given to high-cortisol men evokes status-loss avoidance. In the initial rounds of competition, testosterone's influence on competitive decisions depended on basal cortisol and opponent gender. After providing opponent-specific win-lose feedback, testosterone's influence on decisions to re-enter competitions depended on basal cortisol and this objective cue to status, not gender. Compared to placebo, men given exogenous testosterone who were low in basal cortisol showed an increased tendency to compete against male and high-status opponents relative to female and low-status opponents (status-seeking). Men given exogenous testosterone who were high in basal cortisol showed the opposite pattern - an increased tendency to compete against female and low-status opponents relative to male and high-status opponents (status-loss avoidance). These results provide support for a *context-dependent dual hormone hypothesis*: Testosterone flexibly directs men's competitive behavior contingent on basal cortisol levels and cues that signal an opponent's status.

Key words: testosterone administration, cortisol, social status, competition, opponent gender

Introduction

Competitions determine access to valuable resources that are fundamental components of social mobility and societal life: Securing jobs, promotions, and financial compensation are often contingent on an individual's willingness to enter and ultimately succeed in competitions. Competitive behavior can also be destructive, foster violence and aggression, and lead to toxic social environments (Carré & Olmstead, 2015; Kohn, 1992; Wilson & Daly, 1985). Testosterone is a steroid sex hormone that is theorized to drive status-seeking behavior (Mazur & Booth, 1998), including aggressive, dominant, and competitive behaviors (Archer, 2006). However, most human studies to date have adopted correlational designs, and findings across studies are inconsistent. The primary aim of the present research is to identify dispositional and contextual factors that may account for heterogeneity in testosterone's association with status-seeking behavior. A secondary aim is to enable causal inference about testosterone's role in social behavior.

In service of these aims, we pharmacologically manipulated men's testosterone levels prior to a mixed-gender math competition and examined individual differences in endogenous basal cortisol (a hormone linked to stress and social avoidance) and cues to an opponent's social status (opponent gender, and a win/loss in a prior competition) as factors that may moderate testosterone's effect on competitive behavior. We use this design to test a *context-dependent dual hormone hypothesis*: That testosterone treatment given to men low in basal cortisol will evoke status-seeking motivation, resulting in a preference to compete against high-status relative to low-status opponents; by contrast, testosterone treatment given to men high in basal cortisol is expected to evoke status-loss avoidance motivation, resulting in a preference to compete against low-status relative to high-status opponents.

Testosterone, Cortisol, and Social Behavior

Testosterone is a steroid sex hormone produced and released from Leydig cells in the testes following activation of the hypothalamic-pituitary-gonadal (HPG) axis; in women, testosterone is produced in the ovaries and adrenal cortices. A broad literature has focused on testosterone's role in directing social behavior, extending particularly from theoretical frameworks focused on status seeking and dominance (Archer, 2006; Mazur & Booth, 1998; Wingfield et al., 1990). Empirical evidence indicates that testosterone is associated with a suite of psychological (e.g., implicit power motives; Stanton & Schultheiss, 2009), physiological (e.g., reduced cardiovascular indices of fear; Hermans et al., 2006; Van Honk et al., 2001), and morphological characteristics (e.g., facial cues that signal dominance; Hodges-Simeon et al., 2016; Swaddle & Reierson, 2002; Welling et al., 2016; *cf.* Kordsmeyer et al., 2019) that support the pursuit and maintenance of status and dominance within social hierarchies. Competitive behavior – the act of challenging an opponent over a limited resource (e.g., money) or for the purpose of besting a specific individual (e.g., a rivalry; Deutsch, 1949; Mead, 1937) – is a direct means of seeking status. Specifically, competing against an individual presents an opportunity to boost or affirm the winner's rank relative to the loser; this rank comparison defines a hierarchy and is an explicit indicator of social status.

Two theoretical frameworks generate predictions for testosterone's effects on competitive behavior. First, according to the challenge hypothesis, status-relevant conflicts increase testosterone levels in males and these fluctuations in testosterone in turn drive status-seeking behavior, such as competitive or aggressive behaviors (Archer, 2006; Wingfield et al., 1990). Testosterone has been found to rise in anticipation of and during competitions and fluctuate dependent on competitive outcomes (Casto & Edwards, 2016; Cheng et al., 2018; Geniole et al.,

2017; van der Meij et al., 2011). Testosterone responses to competitions, in turn, have been associated with increases in competitive behavior particularly in males (Carré & McCormick, 2008; Casto et al., 2020; Losecaat Vermeer et al., 2020; Mehta & Josephs, 2006). However, other studies indicate that higher testosterone relates to avoiding competitions in certain situations, presumably to prevent loss of status under conditions of status threat (Mehta et al., 2008; Mehta, Snyder, et al., 2015; Mehta, van Son, et al., 2015) and, in two experiments, testosterone treatment in men did not increase competitive behavior (Nadler et al., 2021). The challenge hypothesis is thus partly supported by human research primarily in correlational studies, but some inconsistencies remain for testosterone's links to competitive behavior.

The second theoretical framework, the dual-hormone hypothesis (Mehta & Josephs, 2010), provides a possible explanation for these inconsistencies: Testosterone's link to status-seeking behavior may depend on basal cortisol levels, a glucocorticoid hormone produced and released by the hypothalamic-pituitary-adrenal (HPA) axis in response to physical and psychological stress (Dickerson & Kemeny, 2004)[1]. Basal cortisol is an individual difference factor that is related to exposure or the propensity to respond to recent, chronic, or ongoing stress (McEwen, 2019). According to the dual-hormone hypothesis, testosterone's influence on status-seeking behavior is posited to be more robust when basal cortisol levels are low; when basal cortisol levels are high, the effect of high testosterone on status-seeking behavior is expected to be inhibited (Knight, Sarkar, et al., 2020; Mehta & Prasad, 2015; Sarkar et al., 2019). Consistent with this hypothesis, higher basal testosterone was positively related to behaviors such as

[1]Our primary analyses focused on basal cortisol as a dispositional factor that was expected to modulate testosterone's effects on competitive behavior. Another theoretical framework, which has received less attention in previous work than the challenge hypothesis or dual-hormone hypothesis, suggests that testosterone's associations with status-relevant behaviors may be stronger among individuals high in self-reported trait dominance. We examined this possibility in secondary analyses. See the Methods section for further background.

dominant leadership behavior, risk-taking, overbidding in a competitive auction, and higher social status when basal cortisol levels were low but not when basal cortisol levels were high (Edwards & Casto, 2013; Mehta, Welker, et al., 2015; Study 1, Mehta & Josephs, 2010; Sherman et al., 2016; Van Den Bos et al., 2013)

The dual-hormone hypothesis is informed by the interplay between the psychological motives associated with testosterone and cortisol. High testosterone is theorized to increase the desire for status (Mazur & Booth, 1998). Higher basal cortisol is associated with stress, anxiety, threat vigilance, and social avoidance, whereas low basal cortisol is linked to decreased stress and social approach (Bertsch et al., 2011; L. L. Brown et al., 1996; Enter et al., 2019; McEwen, 2019; Montoya et al., 2012; Pfattheicher, 2016; Roelofs et al., 2005; Van Honk et al., 1998). Because status-seeking behaviors are approach-oriented in nature, coupling high motivation for social status (high testosterone) and a predisposition toward social approach (low cortisol) may enhance status-seeking behaviors such as dominance and competitive behavior. By contrast, high social avoidance (high cortisol) may inhibit the effect of a high motivation for status (high testosterone) on the expression of status-seeking behaviors.

The dual-hormone hypothesis is also informed by research on the physiological interplay between the HPA and HPG axes (Viau, 2002), which may underlie the psychological mechanisms for status-seeking behavior. For example, cortisol suppresses the activity of the HPG axis, antagonizes the effect of testosterone on target tissues, and downregulates androgen receptors (Burnstein et al., 1995; Chen et al., 1997; Johnson et al., 1992; Mehta & Josephs, 2010; Smith et al., 1985; Tilbrook, 2000; Viau, 2002). The inhibitory effects of cortisol on the HPG axis are particularly evident when cortisol levels are elevated for prolonged periods (i.e., basal cortisol) and are mediated by genomic mechanisms (i.e., hormones binding to and activating

receptors, transcription, and protein synthesis), which occur over relatively long time scales (Tilbrook et al., 2000). Collectively, this evidence suggests that high basal cortisol concentrations may inhibit the effect of testosterone on status-seeking behavior through multiple physiological pathways, and these pathways involve relatively slow genomic mechanisms of action. These physiological pathways may drive an interplay between status and approach-avoidance motivational systems that give rise to status-seeking behaviors

Despite emerging evidence supporting the dual-hormone hypothesis, some studies have revealed different patterns of results. For example, some studies have found higher basal testosterone to be positively related to aggressive behavior, cheating behavior, and psychopathic traits among individuals with *high* basal cortisol (Denson, Mehta, et al., 2013; Lee et al., 2015; Roy et al., 2019; Welker et al., 2014). These findings were taken as evidence against the standard predictions of the dual-hormone hypothesis because these behaviors were considered status-seeking behaviors. High testosterone coupled with high cortisol levels is also an endocrine pattern associated with socially threatening situations (Knight & Mehta, 2017; Marceau et al., 2015; Scheepers & Knight, 2020; Turan et al., 2015). Overall, basal testosterone's relationship to social behaviors implicated in the pursuit of status do appear to depend on basal cortisol in several correlational studies, but with some variability in the exact pattern of results (Dekkers et al., 2019).

Context-Dependent Dual Hormone Hypothesis

We introduce a new theoretical framework – the context-dependent dual-hormone hypothesis – that may account for some of the discrepancies in testosterone and cortisol's links to status-relevant behavior. This framework extends prior theorizing in three related ways. First, and most importantly, the model makes a distinction between two types of status motives –

status-seeking and status-loss avoidance. Second, the model makes predictions about dual-hormone profiles that map onto these two motives. Third, according to the model, these two motives should have context-dependent effects on behavior. We introduce the model in Figure 1 and describe it in detail below.

According to this context-dependent dual-hormone hypothesis, higher testosterone should increase a general motivation for status, which can manifest as status-seeking or status-loss avoidance. Crucially, which type of status motive is dominant within an individual should depend on basal cortisol. Among low-cortisol individuals (low stress and high approach motivation), high testosterone is expected to promote status-seeking. But among high-cortisol individuals (high stress and high avoidance motivation), high testosterone is expected to induce status-loss avoidance. These two status motives should have divergent effects on competitive behavior that depend on an opponent's perceived status. The status-seeking motive in high testosterone-low cortisol individuals is expected to evoke competitive behavior against opponents perceived to be of high status as a means to earn higher rank in the social hierarchy, but less competitive behavior against low-status opponents because these competitions do not provide a status-gain opportunity. In contrast, the status-loss avoidance motive in high testosterone-high cortisol individuals is expected to evoke a fear of losing competitions, and hence, a preference to avoid competing against high-status opponents and, instead, compete against low-status, less skilled opponents.

These hypothesized interactions between biological and contextual factors extend separate areas of research that have studied hierarchy and competition from different perspectives. Research in behavioral endocrinology has focused primarily on the roles of these

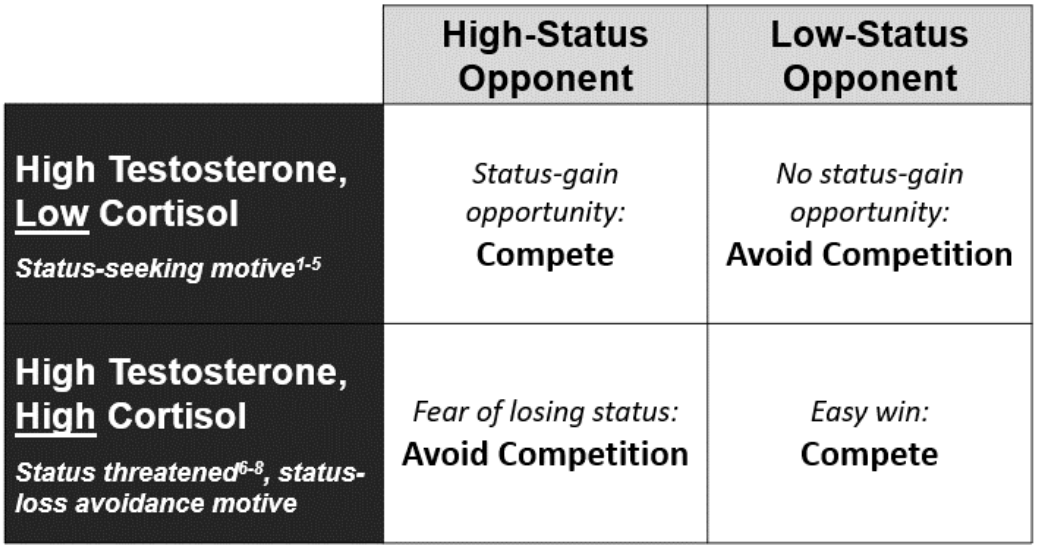

Figure 1. Theoretical model of a context-dependent dual-hormone hypothesis for testosterone's effects on competitive behavior. References in figure: 1) Mehta & Josephs, 2010; 2) Mehta & Prasad, 2015; 3) Sarkar et al., 2019; 4) Knight et al., 2020; 5) Dekkers et al., 2019; 6) Knight & Mehta, 2017; 7) Marceau et al., 2015; 8) Turan et al., 2015. See Knight et al. (2020) for a broader review.

hormones in hierarchy-relevant behavior without considering how the social context may alter hormone-behavior associations (Dekkers et al., 2019), whereas research in social psychology and related fields has examined the impact of hierarchy-relevant contextual factors on behavior with little attention to biological moderators (Buser, 2016; Fast & Chen, 2009; N. L. Mead & Maner, 2012). Thus, a gap remains for understanding whether contextual factors like an opponent's perceived status alter biological determinants of competitive social behavior as only limited work has examined these factors together in competitive settings.

Consistent with this biology × context interactionist framework, one correlational study found that men with high basal testosterone and low basal cortisol levels who had experienced a competitive defeat tended to compete again against the same opponent – that is, against a higher-status opponent. But this tendency to compete against the same opponent was not seen for high-testosterone, low-cortisol individuals who had experienced a victory (Study 2; Mehta & Josephs, 2010). In the same study, men with high basal testosterone and high basal cortisol displayed the

opposite pattern, a tendency to avoid competitions against high-status but not low-status opponents. Collectively, these results suggest that testosterone's association with competitive behavior depends on cortisol and on an opponent's social status: High basal testosterone coupled with low basal cortisol is associated with increased competitive behavior against high-status but not low-status opponents (status-seeking), whereas high basal testosterone coupled with high basal cortisol is associated with avoiding competitions against high-status opponents and increased competitive behavior against low-status opponents (status-loss avoidance).

Present Research

An important limitation of previous research is its correlational design with regard to the direct and moderated effects of testosterone. Thus, it remains unknown whether testosterone has a direct causal effect on men's decisions to enter competitions in line with the challenge hypothesis and/or whether testosterone's causal influence on men's competitive behavior depends on an opponent's perceived status and cortisol levels. Understanding testosterone's causal impact on competitive behavior within the context-dependent dual-hormone hypothesis framework is a crucial step in developing comprehensive theory on the pursuit of social status.

To test hypotheses about the nature of testosterone's causal role in competitions, the present experiment administered exogenous testosterone or placebo to men prior to a competitive decision-making task. The ideal design for testing the modulatory effects of cortisol would involve simultaneous manipulation of the HPG axis (testosterone) and HPA axis (cortisol). However, such dual-systems pharmacology protocols are not readily available because the validity of a joint manipulation of both testosterone and cortisol levels has not been established. Instead, we adopted a mixed experimental design, where we pharmacologically manipulated testosterone levels and examined the moderating effects of endogenous basal cortisol levels and

perceived opponent status. This design represents a novel extension of previous correlational work on hormones and competitive behavior[2].

Prior correlational research has also tended to measure competitive behavior with a single decision to compete or not (Carré & McCormick, 2008; Mehta et al., 2008; Mehta & Josephs, 2010), preventing assessment of the relative propensity to compete against low- and high-status opponents. The present experiment improved on this prior work by using a competition task with multiple decisions to enable within-person comparisons of competitive behavior. Such an approach also increases statistical power compared to paradigms with a single decision.

Much of the previous work examined associations between endogenous hormones and competitive behavior after an explicit social status manipulation (a previous win/lose experience) in men competing against other men. However, individuals often make decisions to enter competitions lacking explicit information relevant to an opponent's perceived status. Real-world settings like academia and other workplaces are also increasingly diverse in terms of gender (Cheryan et al., 2017; Joshi et al., 2015) while notably still lacking equality in terms of power, prestige, and financial compensation (A. J. Brown & Goh, 2016; Gruber et al., 2020; Skitka et al., 2020). Absent explicit evidence of an opponent's status, gender may be used as a cue to an

[2]In line with previous theory and research on the dual-hormone hypothesis, our primary analyses examined basal cortisol as a dispositional factor that was expected to moderate exogenous testosterone's behavioral effects. Indeed, the physiological evidence guiding the dual-hormone hypothesis suggests that chronically elevated cortisol (basal cortisol) robustly affects HPG axis functioning, rather than acute changes in cortisol (Tilbrook, 2000). In line with this physiological evidence, previous research on the dual-hormone hypothesis has primarily examined basal hormone concentrations as predictors of status-seeking behaviors (Dekkers et al., 2019; Knight, Sarkar, et al., 2020). Finally, the previous correlational study that tested the context-dependent dual-hormone hypothesis found that basal cortisol moderated basal testosterone's association with competitive behavior, as opposed to state measures of cortisol taken during the study (Mehta & Josephs, 2010). Acute cortisol changes seem to be a more critical modulator of testosterone-behavior associations after an acute stressor (Prasad et al., 2017, 2019), but the present experiment did not include an acute stressor prior to the competition task. Overall, prior research indicates that basal cortisol is expected to moderate exogenous testosterone's impact on competitive behavior in the present experiment, rather than state measures of cortisol. Nevertheless, because little is known about state cortisol within the dual-hormone hypothesis literature, in the supplemental material we report exploratory analyses with state measures of cortisol and have made the raw data open for further exploration.

opponent's perceived social status based on culturally-determined stereotypes (Datta Gupta et al., 2013; Ellemers, 2018; Fiske et al., 2002; Gneezy et al., 2003; Niederle et al., 2013). Gender stereotypes may be especially relevant in a math-based competition, a domain in which women are stereotyped to perform poorly (Cheryan et al., 2017; Ellemers, 2018; Josephs et al., 2003; Spencer et al., 1999) despite evidence that women can out-perform men on math tasks in laboratory settings (Niederle & Vesterlund, 2011). Based on these gender stereotypes, men who are motivated to seek challenging opponents as a means to gain social status (i.e., men with high testosterone and low cortisol) should prefer competing against male opponents relative to female opponents in a math-based competition; conversely, men threatened by the prospect of losing competitions (men with high testosterone and high cortisol) should actively avoid competition against male opponents and pursue competition against female opponents instead.

Stereotypes are particularly likely to guide person perception and behavior in the absence of objective, individuating information based on social experience (Fiske & Neuberg, 1990). This research suggests that a man competing with an unknown woman in a math competition may initially perceive her to be a low-status opponent based on gender stereotypes (Fiske et al., 2002; Spencer et al., 1999). But if additional information based on social experience suggests that she is a high-status competitor (she wins in a prior math competition), this new information may override gender stereotypes when evaluating her social status. Consistent with this general notion, Wozniak, Harbaugh, and Mayr (2014) found that effects of participant's gender and position in the menstrual cycle in choices to compete in math tasks disappeared once valid performance information was provided. Based on these results, we expected that gender may moderate effects of testosterone and cortisol on competitive behavior only in the absence of objective information of an opponent's status (win/lose performance feedback). Testing these

opponent-status hypotheses in a math-based competition extends prior work on testosterone and cortisol's interactions with opponent status, which has focused on objective cues to opponent status but has neglected subjective cues to status based on stereotypes.

In sum, the challenge hypothesis predicts that testosterone should cause increased competitive behavior overall (Archer, 2006; Wingfield et al., 1990). However, initial correlational evidence indicates that testosterone and cortisol interact with perceived opponent status to predict status-seeking behavior (Mehta & Josephs, 2010). Hence, exogenous testosterone given to men with low basal cortisol may increase willingness to compete against seemingly high-status opponents, predicated on gender stereotypes or feedback that indicates an opponent's relative status. Conversely, exogenous testosterone given to men with high basal cortisol may result in avoiding competition against high-status opponents and instead choosing to compete against "easy-to-beat" low-status targets. The current work examined these hypotheses by measuring basal cortisol levels and pharmacologically manipulating testosterone prior to a mixed-gender competition in which men made decisions to enter competitions before and after receiving accurate win/lose feedback.

Methods

Participants

As part of a broader experiment on exogenous testosterone (Knight et al., 2017), men (n = 120) between the ages of 18-40 (M = 21.5 years, SD = 3.5 years) were recruited via flyers on and near campus and by contacting email lists (see Figure S1 for full experimental timeline). We maximized the diversity of the sample within the constraints of the local population by recruiting students and community members, on and off campus (28% people of color; see Supplementary Materials, Table S1 for diversity evident in socioeconomic indicators). All participants were

prescreened for physical and mental health conditions via a telephone interview prior to the laboratory day (see Supplementary Materials for full list). Upon verifying that participants met the requirements to participate, a laboratory session was scheduled, and they were instructed to abstain from eating, drinking, smoking, or brushing their teeth at least two hours prior to the experimental session. The protocol was approved by the University of Oregon's Institutional Review Board.

Protocol

Participants arrived at the laboratory between 9:00 AM and 11:00 AM. Informed consent was obtained during a 15- to 20-minute resting period to allow participants to acclimate to the laboratory setting, after which participants provided a baseline saliva sample in order to measure pre-treatment, basal cortisol values. This approach to basal cortisol measurement is consistent with previous research on the dual-hormone hypothesis, which also measured baseline hormone levels after a similar acclimation period (Mehta & Josephs, 2010). Participants then applied topical testosterone gel or placebo to their shoulders and upper arms under the supervision of an experimenter. Three hours after gel application, participants provided a second saliva sample and then immediately began the competition task. Approximately fifteen minutes after completing the competition task (and immediately after another, unrelated decision-making task), participants provided another saliva sample. Participants received payment at the end of the experimental session (approximately 2 hours after the end of the competition task) for their time in the laboratory. Participants were also paid based on their performance in the competition task and one other decision-making task.

Exogenous testosterone and blinding

We chose to manipulate testosterone levels in a placebo-controlled fashion to derive causal inference of testosterone's role in competitive behavior. This experimental approach extends previous correlational work that measured endogenous hormone levels only (Apicella et al., 2011; Dekkers et al., 2019; Eisenegger et al., 2017; Mehta & Josephs, 2010). Topical testosterone gel (AbbeVie, Inc., Chicago, IL) was portioned into 150-mg doses and placed in blunted-tip syringes with no indication of the contents. The placebo consisted of a gel produced to exactly match the vehicle of the testosterone gel and was placed in syringes in an equivalent volume to the testosterone samples. Half of the participants were told which treatment they were given (single blind), in order to emulate real-world environments in which testosterone is prescribed. The other half of participants were only told they had an equal chance of receiving testosterone or placebo (double blind). This information was conveyed through a letter in a sealed envelope that had been prepared by members of our laboratory who were not involved in data collection. The experimenters never knew which treatment or blinding condition a participant was assigned. The blinding manipulation was included to facilitate measurement and control of potential expectancy effects related to testosterone and social behavior (Eisenegger et al., 2010). All behavioral analyses control for the blinding condition; follow-up analyses reported in the supplemental materials explored blinding condition as a moderator to ensure any patterns observed in the main analyses replicated across single- and double-blinded participants.

Pharmacokinetics

In prior testosterone administration research, testosterone levels reached peak levels 3 hours after application of topical gel (Eisenegger et al., 2013) and physiological differences due

to testosterone were evident within 3-6 hours after sublingual intake[3] (Radke et al., 2015; Tuiten et al., 2000). Thus, the protocol was designed such that the competition task began approximately three hours after gel application (Mean = 2.92, SE = 0.03 hours).

Salivary Hormone Measurement

Participants were instructed to drool approximately 2 mL of saliva into polypropylene tubes, which were immediately frozen in a -20 °C freezer, prior to transportation to a -80°C freezer for longer-term storage. All samples were assayed for testosterone and cortisol in duplicate consistent with standard, published procedures (Schultheiss & Stanton, 2009) using commercial enzyme immunoassay kits (DRG International, Germany).

Due to the large dose of exogenous testosterone, 17% of the samples from the testosterone treatment group were above the kit's maximum testosterone concentration (no samples in the placebo group were above threshold). Prior research has shown that topical testosterone heightens blood-based testosterone concentrations to a high-normal level despite more extreme values evident in saliva (Krebs et al., 2019; Puiu et al., 2019; Schönfelder et al., 2016). Supraphysiological salivary hormone concentrations after topical treatment may result from absorption of the hormone into subcutaneous tissue and transport to the salivary glands via the lymphatic system (Du et al., 2013; Krebs et al., 2019). Due to these concerns with salivary concentrations after testosterone administration, testosterone concentrations in the present experiment are used only to ensure testosterone treatment increased testosterone levels and are not used to predict behavior. Samples with concentrations above the kit's threshold were

[3] This delay of several hours between testosterone treatment and behavioral testing is consistent with relatively slow, genomic mechanisms for hormonal effects on behavior. Later research published after these data were collected suggests that topical testosterone treatment increases testosterone and produces measurable physiological differences within an hour post-gel administration (Bird et al., 2016; Carré et al., 2017). Hormonal influences on behavior over this shorter time period may be occurring through more rapid, non-genomic mechanisms (Makara & Haller, 2001; Moore & Evans, 1999).

replaced with the kit's maximum (5250 pg/mL) as a conservative approximation of the sample's testosterone concentration. For cortisol, the average intra-assay coefficient of variation (CV) was 4.68%; the inter-assay CV was 14.8%. For testosterone (ignoring samples above kit threshold), the average intra-assay CV was 6.55% and the inter-assay CV was 16.1%. Testosterone and cortisol concentrations were square-root transformed to correct positively skewed distributions (see Figure S2 in Supplemental Materials for distributions of hormone concentrations).

Basal Cortisol Measurement

Our primary analyses focus on pre-treatment basal cortisol in line with previous research on the dual hormone hypothesis, which has focused almost exclusively on basal hormone levels (Dekkers et al., 2019; Mehta & Josephs, 2010). We considered the first saliva sample of the experiment a basal measurement because it occurred after an acclimation period but prior to testosterone or placebo administration, prior to laboratory task instructions, and prior to any laboratory tasks. The second cortisol measurement in the present experiment, collected prior to the competition task, could not be considered a basal measure as it occurred approximately three hours post-administration of testosterone treatment and because testosterone treatment can influence activity across the HPA axis (Rubinow et al., 2005; Viau, 2002). This approach to basal cortisol measurement follows directly from previous research. For example, the previous correlational study upon which we are building also measured basal cortisol with an initial sample that was taken after an acclimation period but before behavioral task instructions (Mehta & Josephs, 2010). Our approach to basal cortisol measurement is consistent with genomic mechanisms of action, whereby pre-treatment basal cortisol is expected to moderate the effects of exogenous testosterone treatment on competitive behavior measured several hours later.

Competition Task

The competition task was designed to measure competitive decisions in mixed-gender math competitions before and after receiving win/lose feedback (Figure 2; Mayr et al., 2012). Performance metrics were also measured in the task to determine whether effects on competitive behavior were or were not explained by performance.

The initial phase consisted of forty-eight rounds of a math task in which participants were given opportunities to compete against other players for points in a winner-take-all payment scheme or to play for a piece rate instead. In each round, subjects were tasked with deciding if

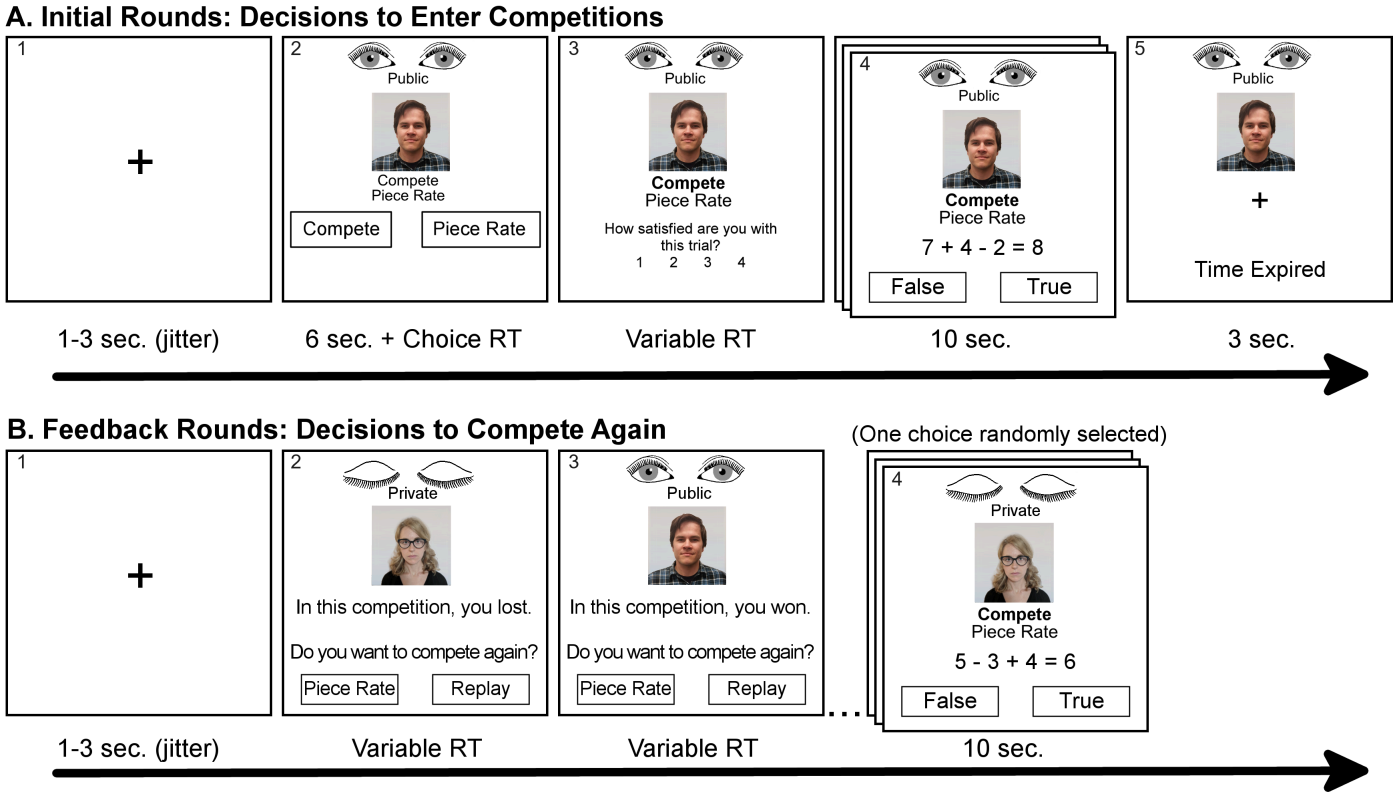

Figure 2. The competition task. **A.** In the early rounds of the competition task, participants made decisions to enter competitions against 16 opponents (n = 8 female) or play for a piece rate instead. Participants also played in 16 mandatory compete and 16 mandatory piece rate rounds against the same opponents (48 rounds in total). The actual competition consisted of answering as many True/False equations as possible within 10 seconds. Scores in a given competition trial were compared to the opponent's actual score on the same set of True/False equations. **B.** After completing all 48 rounds, participants were provided feedback from the 16 mandatory compete rounds and asked whether they wanted to compete against that opponent again ("Replay") or play for a piece rate instead. One of these decisions was randomly selected to be played. Opponent images in this figure were not part of the stimuli but are representative of the types of photos in the opponent pool. The numbers appearing in the upper left corner of each frame are included to be able to describe the task and were not part of the task.

simple math equations containing addition and subtraction were true or false. Participants had ten seconds in each round to answer as many equations as possible (panel 4 in Figure 2A and 2B). One point was awarded for every correct answer and one point was deducted for every incorrect answer. Each round presented one of sixteen possible opponents (n = 8 female) who were individuals who had completed the task previously and had their actual scores saved from the same series of equations.

During "compete" rounds, a winner was determined by comparing the number of points earned by the participant to the number of points earned previously by the opponent. For these rounds, each point was worth $4 but only if the participant won the round; if they scored fewer points than their opponents, they earned nothing ($0). In the case of ties, the amount of time required to respond to the round's equations was used to determine a winner[4]. During "piece rate" rounds, participants attempted to answer as many math equations as possible, but their score was not compared to that of the opponent. In piece rate rounds, every point the participant earned was worth $2 regardless of how the opponent performed in that round. In rounds in which participants earned negative points (more incorrect than correct responses), participants' earnings were set to $0.

In one-third of the rounds, participants chose whether to compete against the current opponent or play for a piece rate (i.e., "choice rounds"; panel 2 in Figure 2A). Decisions to play a competition in these choice rounds were coded as competitive behavior. In the remaining rounds, participants were forced to compete or play for a piece rate ("mandatory rounds"). In

[4] For example, assume the participant and opponent each scored 5 points, but the participant earned those points in 8.9 seconds compared to the opponent, who required 9.2 seconds. In this case, the participant (8.9 < 9.2) won that round.

summary, there were 48 rounds divided into three categories – 16 choice, 16 mandatory compete, 16 mandatory piece rate – and each opponent appeared once in each category.

After the initial 48 rounds of the task (Figure 2A), participants completed the "Feedback rounds," in which feedback was provided on whether the participant had won or lost in the sixteen mandatory compete rounds (Figure 2B). Immediately after providing feedback on the outcome of a prior competition, participants were asked whether they preferred to compete against that opponent again in a follow-up round or play a piece rate round instead (panels 2 and 3, Figure 2B). After all of these decisions were made, one of these post-feedback decisions was randomly chosen and participants played a round based on that choice (i.e., as a compete or piece-rate round; panel 4, Figure 2B). Participants' performance-based payout consisted of winnings based on one randomly chosen choice round, one randomly chosen mandatory competition round, one randomly chosen mandatory piece-rate round, and the randomly chosen post-feedback round.

These instructions were explained to the participant approximately an hour prior to the start of the competition task. The experimenter guided the participant through an interactive demonstration of the task that ended with a practice trial of the math task. Participants also had to successfully complete a short, verbal quiz focused on key features of the task (see Supplementary Materials).

Competition Score, Satisfaction, and Outcome

Prior to the start of each trial's math task (and after participants made decisions in choice rounds), participants were asked how satisfied they were with the present trial on a scale from 1 (not at all satisfied) to 4 (very satisfied; panel 3, Figure 2A). We also recorded the number of points earned in each trial and the participants' rate of winning rounds. These additional

measures were included to explore the extent to which they did or did not explain hormonal effects on decisions to compete.

Subjective Ratings of the Opponents

To examine whether gender was used as a cue to perceived opponent status in our competition task, participants rated the opponents' images on a set of nine variables on a Likert scale from 1 (strongly disagree) to 7 (strongly agree). Here we report the extent to which the rater felt the opponent was "good at simple math tasks." (see supplement for analyses of the remaining variables). Participants rated the photos at the end of the laboratory session, several hours after feedback had been provided during the competition task.

Because these ratings are confounded with the feedback provided in the competition task, a small sample (n = 16) of men who did not participate in the competition task also rated each opponent on the "good…at math" item. These follow-up raters consisted of a convenience sample of undergraduate research assistants and graduate students not affiliated with this experiment.

Other Aspects of the Competition Task

The task also contained a social-evaluative manipulation (Figure 2). In half of all trials, participants were instructed that the experimenter could see the participants' decisions and competitive performances displayed on a screen in another room (Public condition); the public trials contained an outline of open eyes and the word "Public" written at the top of the screen throughout those trials. In the remaining trials, participants' decisions and performance were not visible to the experimenter (Private condition); the private trials contained an outline of closed eyes and the word "Private" written at the top of the screen. Because few studies have examined a social-evaluative manipulation as a moderator of testosterone's influence on behavior

(Losecaat Vermeer et al., 2020; Wu et al., 2020), on an exploratory basis we tested the hypothesis that social evaluation might enhance testosterone's effects on behavior by raising the status implications of the competition in trials when there was an audience. Further background and results for this manipulation are discussed in the Supplementary Materials. All behavioral analyses control for the social-evaluative manipulation.

Analyses

Endocrine Levels

Group differences in testosterone and cortisol levels were examined at baseline and in terms of overall exposure across the duration of this experiment (AUC_G). General linear models (GLM) were produced with hormone concentration (baseline or AUC_G) regressed on testosterone treatment group to test for group differences. Coefficients representing the difference between the testosterone treatment and placebo groups are reported with 95% CIs, F-tests, and p-values.

Primary Behavioral Analyses

Primary analyses were focused on participants' decisions to compete or play for a piece rate in the choice rounds of the early phase and in the feedback rounds as a function of the main effects and interactions of testosterone treatment, basal cortisol, and opponent status. In each set of models, the effects of testosterone treatment group and opponent gender in a given trial were analyzed as categorical variables (Testosterone Treatment: testosterone = 1, placebo = 0; Opponent Gender: female opponent = 1, male opponent = 0); basal cortisol was included as a continuous variable.

Binomial logistic multilevel models were constructed in R (3.5.1; R-Team, 2018) using the 'glmer' function from the *lme4* package (Bates et al., 2015). Decisions were binary coded as

Compete = 1 and Piece rate = 0. For behavioral analyses, we report odds ratios (ORs) with 95% confidence intervals (95%CIs).

For decisions to enter competitions, our model to test the interaction among testosterone treatment, cortisol, and opponent gender consisted of the following variables across two levels for round *i* within participant *j*:

Level 1:

$$logit(Compete_{ij}) = \beta_0 + \beta_1 Gender_i + \beta_2 Private_i + r_{ij}$$

Level 2:

$$\beta_0 = \gamma_{00} + \gamma_{01} Testosterone_j + \gamma_{02} Cortisol_j + \gamma_{03} Testosterone_j \times Cortisol_j + \gamma_{04} Blinding_j + e_{0j}$$

$$\beta_1 = \gamma_{10} + \gamma_{11} Testosterone_j + \gamma_{12} Cortisol_j + \gamma_{13} Testosterone_j \times Cortisol_j + \gamma_{14} Blinding_j + e_{1j}$$

$$\beta_2 = \gamma_{20} + e_{2j}$$

We also examined a model with just the main effects (i.e., removing all interactions of testosterone, cortisol, and gender) as well as a model with just two-way interactions with testosterone treatment (i.e., separate models for testosterone treatment and cortisol, testosterone treatment and gender).

For our primary analyses of decisions to compete again in the feedback rounds, the models contained an additional term representing the prior competition outcome from the mandatory compete round against a given opponent (Won = 1, Lost = 0). Thus, our principal model to test the interaction among testosterone treatment, cortisol, and prior outcome consisted of the following variables across 2 levels:

Level 1:

$$logit(CompeteAgain_{ij}) = \beta_0 + \beta_1 PriorOutcome_i + \beta_2 Gender_i + \beta_3 Private_i + r_{ij}$$

Level 2:

$$\beta_0 = \gamma_{00} + \gamma_{01} Testosterone_j + \gamma_{02} Cortisol_j + \gamma_{03} Testosterone_j \times Cortisol_j + \gamma_{04} Blinding_j + e_{0j}$$

$$\beta_1 = \gamma_{10} + \gamma_{11}Testosterone_j + \gamma_{12}Cortisol_j + \gamma_{13}Testosterone_j \times Cortisol_j + \gamma_{14}Blinding_j + e_{1j}$$
$$\beta_2 = \gamma_{20} + e_{2j}$$
$$\beta_3 = \gamma_{30} + e_{3j}$$

Similar to the early phase models, we examined a model with just the main effects (i.e., testosterone treatment, cortisol, opponent gender, and prior outcome) as well as models with just two-way interactions with testosterone treatment (i.e., testosterone treatment and cortisol, testosterone treatment and prior outcome). We also explored the four-way interaction between testosterone treatment, cortisol, opponent gender, and prior competitive outcome (i.e., this was not considered a primary analysis).

In cases where the initial and feedback phase models could not be satisfactorily fit (i.e., due to a singular fit), the complexity of the model was reduced by sequential removal of the random term for social-evaluative observation, gender, and/or prior outcome and re-run (Nakagawa et al., 2017).

Significant interactions were broken down in two ways. First, a simple slopes approach was used to examine within-person comparisons of high and low status opponents based on opponent gender in the initial phase or prior outcome in the feedback phase among individuals in the testosterone treatment and placebo groups with high (+1 SD) or low (-1SD) cortisol levels (Hughes, 2020; Preacher et al., 2006). Second, we calculated empirical Bayes estimates of the slopes of opponent gender and prior outcome. To estimate these slopes from the initial phase, decisions to compete were regressed on opponent gender with a random slope and intercept for opponent gender per participant, controlling for the social evaluation condition. In the feedback phase, decisions to compete were regressed on prior outcome with random slope and intercept for prior outcome per participant, controlling for opponent gender and social evaluation

condition. For each of these sets of slopes, a positive slope indicates a greater propensity to compete against female opponents or prior losers relative to male opponents or prior winners (respectively), and a negative slope value indicates a greater propensity to compete against male opponents or prior winners relative to female opponents or prior losers. In separate models, these slopes were then regressed on the two-way interaction between testosterone treatment condition and basal cortisol.

Follow-up Analyses with Covariates. We followed up our initial analyses with separate models that controlled for time of day of the laboratory task and time since awakening prior to the first salivary sample. Both of these variables may index diurnal aspects of endocrine functioning. We also included participants' overall skill in the task – indexed by each participant's mean points earned on the mandatory piece rate rounds – as a covariate in follow-up analyses.

Secondary Analyses

Behavioral analyses with trait dominance. Some prior work suggests that trait dominance – defined as the tendency to rely on force, fear, and intimidation to take or defend higher status positions (Cheng et al., 2013) – may accentuate testosterone's association with status-relevant behavior[5]. Within this theoretical framework, self-reported trait dominance is considered an explicit component of dominance, whereas testosterone is considered an implicit component of dominance that operates outside conscious awareness (Knight, Sarkar, et al., 2020). Because implicit and explicit forms of a given construct can interactively determine behavior (Slatcher et al., 2011), high levels of testosterone in an individual with high trait

[5]Trait dominance × testosterone interactions have been studied using different definitions of dominance – a possible "jingle fallacy" (Block, 1995) – and therefore different scales to measure trait dominance. In the supplementary material, we discuss how these issues may be contributing to heterogeneous results across studies.

dominance may synergistically heighten concern for status and increase status-relevant behaviors.

However, the behavioral effects within the testosterone × trait dominance literature are somewhat nuanced. For example, exogenous testosterone's effects on competitive motivation were exaggerated among women who were high in trait dominance (Mehta, van Son, et al., 2015) and among men high in trait dominance who were assigned to a low status position (Losecaat Vermeer et al., 2020). However, this latter effect was not observed in a later portion of the same contest and, in another experiment focused on men's physical persistence in a competition, trait dominance did not moderate the effects of exogenous testosterone (Kutlikova et al., 2021). Among other status-relevant behaviors and contexts, trait dominance enhanced endogenous testosterone's association with men's mating behavior (Slatcher et al., 2011) and with men's aggressive behavior (albeit only after a victory experience; Carré et al., 2009); trait dominance also enhanced exogenous testosterone's causal effect on men's aggressive behavior (Carré et al., 2017). Another experiment found that a personality risk factor that included dominance and other related traits significantly accentuated the effects of exogenous testosterone on men's aggressive behavior; the moderating effect of trait dominance alone was not significant but was similar in magnitude and direction as the personality risk factor (Geniole et al., 2019). In the same sample of men reported here, trait dominance amplified the effects of testosterone on cortisol and negative affect responses to social-evaluative stress (Knight et al., 2017). However, other work found that trait dominance did not significantly moderate the association between testosterone and men's risk-taking behavior, with a directional pattern that was unexpected (Welker et al., 2019).

One previous study also investigated trait dominance in the context of the dual-hormone hypothesis. Trait dominance did not significantly moderate the interactive association of testosterone and cortisol with aggressive behavior in another study, although this report suggests that the high-testosterone, low-cortisol association with aggressive behavior may be more evident in men with higher trait dominance (Pfattheicher, 2017). It therefore remains unknown whether testosterone and cortisol interactions with trait dominance will extend to men's competitive decisions.

Because of this small but growing literature, we conducted secondary analyses to explore the moderating effect of trait dominance on testosterone and cortisol's associations with men's decisions to compete. We indexed trait dominance via the dominance subscale of the Dominance and Prestige scale. The dominance subscale consists of 8 items related to dominance (e.g., "I try to control others rather than permit them to control me.") on a scale from 1 (not at all) to 7 (very much). Dominance items (Cronbach's $\alpha = .68$) were averaged and standardized. To limit the number of tests conducted, we tested testosterone treatment × trait dominance, testosterone treatment × basal cortisol × trait dominance, and testosterone treatment × opponent status cue × trait dominance effects on decisions to enter competitions in each phase of the competition task.

Subjective ratings of the opponents. We examined perceptions of how "good…at math" each of the opponents were via an MLM as an implicit index of perceived opponent status in the competition. Opponent gender was entered as a dummy code in each model (1 = female opponent, 0 = male opponent), with a random intercept and a random slope of opponent gender for each participant and a random intercept for each opponent. We next examined the effect of gender on "good at…math" ratings while controlling for whether the participant won or loss to that opponent in the mandatory compete rounds. In a separate model, we examined the effects of

gender on "good at…math" ratings among the follow-up sample. Finally, we examined the similarity of the ratings across the two datasets by pooling data and producing a model that included the effects of opponent gender, source of ratings (participants = -0.5, follow-up raters = 0.5), and the interaction between gender and rating source.

Exploratory Analyses

Dual-hormone effects with other cortisol measures. Because the dual-hormone hypothesis focuses on basal cortisol, less is known about state cortisol measures as moderators of testosterone's behavioral effects. The few studies that examined state measures of cortisol within the dual-hormone literature suggest that acute cortisol fluctuations may be a relevant moderator for testosterone's behavioral effects when an acute stressor is included prior to the measurement of the behavioral outcome measure (Prasad et al., 2017; 2019; Knight et al., 2020). However, the present experiment did not include an acute stressor prior to the competition task. Thus, we did not expect state measures of cortisol to moderate the impact of exogenous testosterone on competitive behavior.

Nevertheless, given that few studies within the dual-hormone literature have examined state cortisol measures, we conducted exploratory analyses with such measures to guide ongoing theory development. Specifically, we explored cortisol fluctuations around the competition task using a difference score from pre- to post-competition cortisol and diurnal cortisol dynamics with area-under-the-curve with respect to ground (AUC_G) using the variable time durations between samples (Pruessner et al., 2003). AUC_G with variable time durations provides a relatively precise index of total cortisol exposure during the period before and concurrent to the competition task that other approaches (e.g., a single measure or averaging across several measures) cannot readily provide (Pruessner et al., 2003). For behavioral analyses, transformed

cortisol values were standardized. Readers interested in other cortisol measures not included in this report are invited to explore the open dataset.

Competition score, satisfaction, and outcome. On an exploratory basis, we investigated whether testosterone treatment, basal cortisol, and opponent gender predicted points earned in each round, self-reported satisfaction, and likelihood of winning competitions during the initial phase of the competition task. These analyses were intended to explore if testosterone, cortisol, and opponent status led to differential performance or satisfaction in this task. These measures are of interest for theory development to determine if testosterone, cortisol, and opponent status predicted differences in competitive behavior with or without subsequent differences in actual performance or satisfaction. Multilevel linear regression models were produced that examined points earned and satisfaction among all choice trials. A binary logistic multilevel model was used to investigate whether testosterone treatment and its interaction with basal cortisol and opponent gender predicted likelihood of winning. A second set of models were analyzed that included the participant's choice (piece rate = 0; compete = 1) as an additional moderator of testosterone treatment, basal cortisol, and opponent gender in order to explore if participants decisions were associated with score, satisfaction, or likelihood of winning.

False Discovery Rate (FDR) Correction

Our primary analyses build on previous research and the theoretical framework outlined in Figure 1 to test the context-dependent dual-hormone hypothesis. Specifically, the testosterone × cortisol × prior outcome (i.e., win/lose feedback) interaction has prior support in previous correlational research (Study 2, Mehta & Josephs, 2010), and we tested this interaction and extended it to opponent gender (i.e., testosterone × cortisol × opponent gender) as a second status cue based on stereotypes in both phases of the competition. As discussed in the introduction,

previous research points to additional hypotheses about the influence of testosterone or opponent status cues on competitive behavior. Therefore, we tested eight other effects (representing thirteen results; hence, sixteen results corrected in total) for which support was evident in the prior literature and applied Benjamini-Hochberg (1995) FDR correction. Specifically, primary and secondary analyses also tested the following effects in both stages of the competition (initial phase and feedback phase, where applicable): the main effects of testosterone (Mehta et al., 2017), opponent gender (Datta Gupta et al., 2013), and prior outcome (Buser, 2016); testosterone × cortisol (Mehta & Josephs, 2010); testosterone × prior outcome (Mehta et al., 2008); testosterone x opponent gender (Josephs et al., 2003); testosterone × trait dominance (Slatcher et al., 2011); and testosterone × trait dominance × prior outcome (Carré et al., 2009; Mehta, van Son, et al., 2015). We report adjusted p-values for these behavioral results and consider $q = .05$ as a cutoff for FDR corrected statistical significance.

Meta-Analyses

In order to examine the meta-effect evident across this experiment and one prior correlational study (Studuy 2, Mehta & Josephs, 2010), we examined a fixed-effects meta-analysis of the three-way interaction among testosterone, cortisol, and prior competition outcome (win vs. lose) on decisions to compete again. T-test scores from each study's three-way interaction model were transformed into correlation coefficients, then transformed further to Fisher's z scores (Dekkers et al., 2019). Degrees of freedom from this experiment's multilevel models were calculated using the between-within method, which has been shown to maintain appropriate Type I error rates, maintain power, and is robust to small numbers of clusters and variation in cluster size (Li & Redden, 2015). To examine the effects of testosterone, cortisol, and opponent status across both phases of the competition task, a second, random effects (RE)

meta-analysis was examined that included both of the primary three-way interactions from the initial and feedback phase and the prior correlational effect. This model included random effects per study in order to account for the non-independence of the two effects from the present experiment.

Justification for Sample Size and Maximizing Power

The sample size for this experiment was determined by power to detect between-group differences in a pharmacological treatment experiment and was as large as possible within the experiment's budget. The present experiment improves power compared to previous work on testosterone, cortisol, and opponent status (e.g., Mehta & Josephs, 2010) by 1) doubling the sample size; 2) using a within-subjects comparisons based on multiple trials for each opponent gender and for winning and losing opponents; and 3) administering exogenous testosterone treatment, which was expected to boost effect sizes relative to an observational approach based on endogenous testosterone. Power simulations conducted after data collection was completed indicate that, given our intended sample size (n = 120), a within-subjects approach, and assuming a moderate level of within-subject correlation, this experiment was 80% powered to detect a three-way interaction term equivalent to log(B) = 0.7, OR = 2.0, akin to a moderate effect size (Figure S2; see Supplemental Materials for details of simulation and a comparison of our within-subjects approach to simulations of one-shot study designs). The simulated, 80%-powered effect size was also weaker than an effect size reported in earlier correlational work (Mehta & Josephs, 2010; see Supplemental Materials).

Transparent Reporting

This report is part of a larger project examining exogenous testosterone's effects on social behavior and responses to social stress (e.g., a social stressor occurred after the

competition task and participants did not know the nature of the stressor task prior to the competition; Knight et al., 2017; Knight, McShane, et al., 2020). All data, code, and experimental materials are archived online at the project's Open Science Framework website (https://osf.io/hvumx/?view_only=eff1d4befafc47f6ab73d6a44c0173be).

Results

Data Attrition and Descriptive Results

Four participants' data were lost due to equipment or software malfunction during the competition task; one additional participant left the experiment prior to the competition task, leaving a total of n = 115 participants in the analyses. See Table 1 for descriptive statistics of the experiment's sample.

Endocrine Levels

Testosterone

Baseline differences in testosterone were not robust between treatment groups (B = 0.230, *95%CI* [-0.048, 0.508], $F(1,113) = 2.68$, $p = .104$). Analysis of testosterone AUC_G revealed substantial differences between treatment groups, indicating that the pharmacological manipulation increased testosterone levels (B = 5639.7, [4735.9, 6543.5], $F(1,111) = 3.50$, $p < .001$; see Knight et al., 2017 for group differences based on full time series of testosterone data).

Cortisol

Table 1: Descriptive statistics [mean (SD)] of study sample

	Testosterone	Placebo	All
Sample size	58	57	115
Testosterone (pg/mL)			
Baseline	136.3 (172.3)	112.5 (188.1)	124.5 (179.9)
Pre-Competition[1]	2768.5 (2130.7)	108.1 (95.3)	1449.9 (2014.8)
Post-Competition[1]	3229.0 (2167.1)	244.7 (426.6)	1750.0 (2164.2)
Cortisol (μg/dL)			
Baseline	0.367 (0.209)	0.310 (0.181)	0.339 (0.197)
Pre-Competition	0.213 (0.154)	0.217 (0.127)	0.215 (0.141)
Post-Competition	0.154 (0.089)	0.163 (0.090)	0.158 (0.090)
Decisions to Compete (% of choice trials in Initial Phase)	67.5 (25.6)	59.1 (30.1)	63.3 (28.1)
Male Opponents	60.3 (29.5)	54.4 (33.9)	57.4 (31.8)
Female Opponents	74.6 (26.4)	63.8 (31.7)	69.2 (29.5)
Decisions to Compete Again (% of choices in Feedback Phase)	48.6 (23.9)	47.7 (23.5)	48.2 (23.6)
Male Opponents[2]	49.1 (28.1)	43.9 (27.8)	46.6 (28.0)
Female Opponents[2]	54.1 (28.8)	57.5 (25.8)	55.8 (27.3)
Prior Winners[3]	47.0 (37.1)	42.4 (33.6)	44.7 (35.3)
Prior Losers[3]	60.9 (40.8)	64.1 (38.6)	62.4 (39.6)
Trial Score (all trials in Initial Phase)	2.59 (0.99)	2.52 (0.73)	2.55 (0.87)

Notes:

1. A portion of samples in the testosterone treatment group were above the maximum values of the kits used. For the purposes of calculating these means, the missing values were replaced with the kit's maximum levels.
2. Regardless of prior win/loss.
3. Regardless of opponent gender. "Winners" and "losers" in these rows refers to the opponent – e.g., "prior winners" are opponents that won a mandatory compete trial against the participant in the initial phase.

Robust differences between treatment groups were not evident in baseline cortisol (B = 0.051, [-0.010, 0.111], $F(1,112) = 2.76$, $p = .099$) or overall cortisol exposure as indexed by AUC_G (B = 5.26, [-4.70, 15.21], $F(1,111) = 0.725$, $p = .298$).

Initial Decisions to Enter Competitions

For our primary behavioral analyses, we examined decisions to enter competitions against a mixed-gender pool of opponents in the initial phase of the competition. All analytical

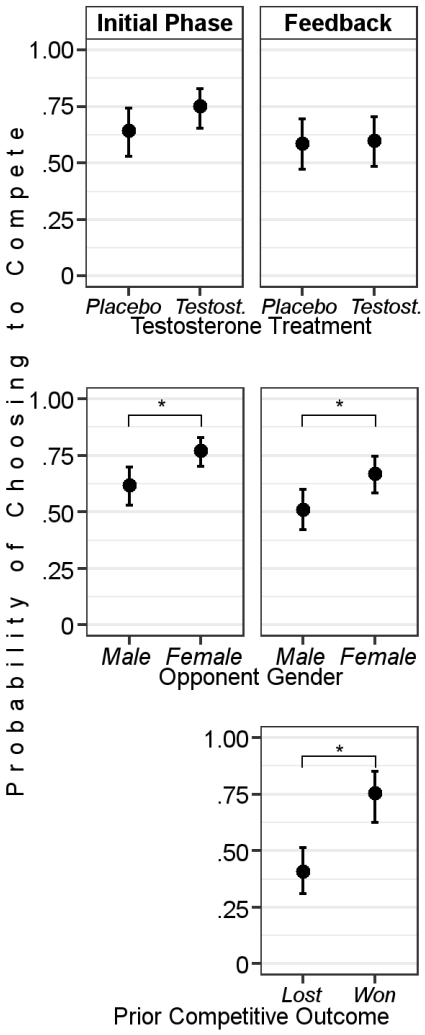

Figure 3. Estimated marginal means of main effects of key study variables on probability of choosing to compete. Charts in left column are from decisions to enter competitions in the Initial Phase; charts in right column are from Feedback Phase decisions to re-enter competitions. *indicates 95%CI of difference does not contain zero.

models[6] were satisfactorily fit with random terms for both within-participant variables in the initial phase (i.e., gender and social-evaluative observation).

Main Effect of Testosterone Treatment[7]

According to the challenge hypothesis, testosterone should directly increase decisions to compete. Controlling for all other study variables, exogenous testosterone did not substantially affect decisions to enter competitions compared to placebo, though the effect was in the direction of testosterone causing increased competitive behavior (*OR* = 1.67, [0.86, 3.27], $p = .132$, $p_{FDR} = 0.303$; Table S2; Figure 3, upper panel).

Main Effect of Opponent Gender (Perceived Opponent Status Cue)

In the same model, we tested whether opponent gender predicted decisions to compete. We found that participants were over twice as likely to compete against female opponents compared to male opponents (*OR* = 2.09, [1.56, 2.79], $p < .001$, $p_{FDR} < .001$; Table S2; Figure 3, middle panel), consistent with prior

[6] See supplement for model fit statistics for all models.
[7] See supplement for reporting of main effects of basal cortisol and experimental blinding manipulation, each of which was not a primary focus of this experiment and each of which demonstrated non-significant associations with decisions to compete in the initial and feedback round of the competition task.

research (Datta Gupta et al., 2013). This result supports the hypothesis that opponent gender was used as a cue to perceived opponent status that influenced decisions to compete in this task.

Testosterone × Opponent Gender

In a separate model, we examined the extent to which testosterone treatment interacted with the gender of an opponent to cause competitive behavior. Testosterone treatment and opponent gender did not robustly interact to predict competitive behavior (OR = 1.40, [0.81, 2.42], p = .227, p_{FDR} = .404; Table S2).

Dual-Hormone Hypothesis

Next, we tested the original, concise variant of the dual-hormone hypothesis that does not take opponent status cues into consideration. According to this hypothesis, testosterone treatment should increase competitive behavior, but only among men with low basal cortisol. The model's estimate of a two-way interaction between testosterone treatment and basal cortisol did not support this hypothesis (OR = 1.13, [0.54, 2.34], p = .753, p_{FDR} = .926; Table S2).

Context-Dependent Dual-Hormone Hypothesis

We next assessed our context-dependent dual-hormone hypothesis, which predicts that the effect of testosterone treatment on competitive decision should be moderated by basal cortisol and cues to an opponent's perceived status (i.e., opponent gender in the initial phase of our task). In support of this hypothesis, the testosterone treatment × basal cortisol × opponent gender interaction was found to predict decisions to enter competitions (OR = 2.54, [1.47, 4.37], p<.001, p_{FDR} = .003; Table S2; Figure 4).

To break down this three-way interaction, we examined the simple slopes associating opponent gender with likelihood to compete at high (+1SD) and low cortisol levels (-1SD) for each treatment group (Preacher et al., 2006). In the placebo group, men competed more against

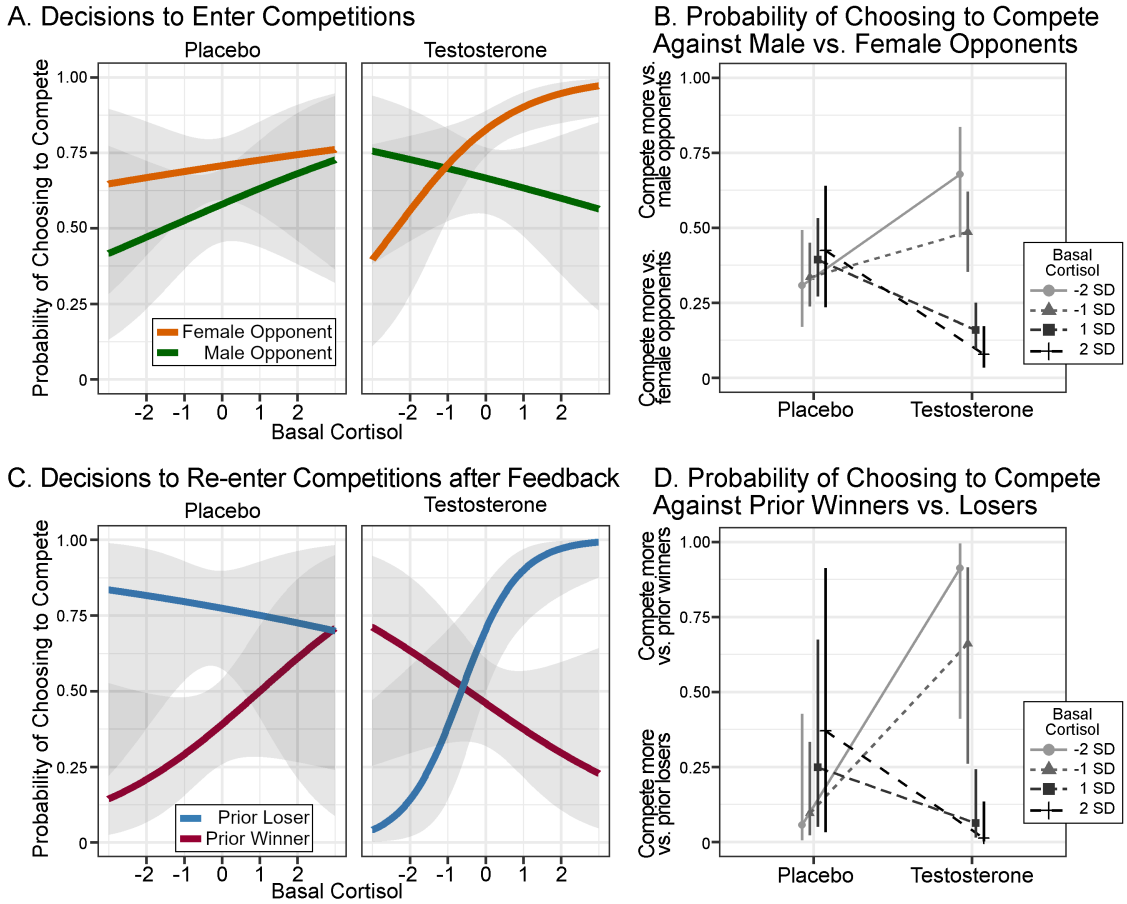

Figure 4. Estimated marginal probability of choosing to compete as a function of testosterone treatment, basal cortisol, and social contextual factors. All values on every panel are derived from the main analytical models (e.g., estimated marginal means or simple slopes). Error bands represent 95%CI of model estimates. Panels A and C represent the full range of basal cortisol values, whereas the simple slope analyses in text and panels B and D present results from ±1and 2 SD. For illustrative purposes and to better match the prior literature, the probabilities calculated from simple slope logits extracted from the three-way interaction were inverted (i.e., "1 – *p*(Compete)") in Panels B and D. **A.** Probability of choosing to enter competitions from the initial phase, conditional on the gender of an opponent. **B.** Probability of competing against female opponents versus male opponents for testosterone treatment and placebo groups at several basal cortisol levels. Positive values on this chart indicate that the probability to compete against male opponents is higher than competing against female opponents. **C.** Probability of choosing to re-enter competitions after feedback, conditional on prior competitive outcomes. **D.** Probability of competing after winning versus after losing for testosterone treatment and placebo groups at several basal cortisol levels. Positive values on this chart indicate that the probability of choosing to compete against a prior winner is higher than competing against a prior loser.

female opponents (perceived low status) than against male opponents (perceived high status), but this effect was roughly equivalent at high (*OR* = 1.54, [0.882, 2.70]) and low basal cortisol levels (*OR* = 1.99, [1.23, 3.22]; Figure 4B). In the testosterone group, men who had high basal cortisol levels were substantially more likely to compete against female opponents compared to male opponents (*OR* = 5.30, [3.01, 9.34]), whereas in men with low basal cortisol levels, the pattern of competing more against female than male opponents was inhibited, due primarily to an increased preference for competing against male opponents (*OR* = 1.06, [0.611, 1.84]; Figure 4B).

As another strategy to break down this three-way interaction, we examined slopes linking the probability of competing against female and male opponents. A positive slope indicates a greater propensity to compete against female relative to male opponents, and a negative slope value indicates a greater propensity to compete against male relative to female opponents. We regressed opponent gender slope scores on testosterone treatment condition, basal cortisol, and their interaction. The testosterone treatment × basal cortisol interaction was significant (B = 0.184, t(111) = 2.96, p = 0.004). We conducted simple slopes analyses by examining the effect of testosterone treatment (compared to placebo treatment) on the opponent gender slope one standard deviation above and below the basal cortisol mean. Similar to the pattern depicted in Figure 4 (Panel B), these analyses revealed that the effect of testosterone versus placebo on the opponent gender slope went in the opposite directions for those high and low in basal cortisol: There was a positive effect of testosterone compared to placebo on the opponent gender slope for high-cortisol individuals (B = 0.24, [0.07, 0.42]) and a directionally negative effect of testosterone compared to placebo on the opponent gender slope for low-cortisol individuals (B = -0.13, [-0.29, 0.04]). We interpret the somewhat stronger effect of testosterone versus placebo on the opponent gender slope in high-cortisol relative to low-cortisol individuals as noise that is

unlikely to be theoretically meaningful (for additional discussion regarding this interpretation, see Supplementary Material)[8].

Although overall men are more likely to compete against female opponents compared to male opponents (main effect of opponent gender), these results show that men who were administered testosterone and who had high basal cortisol displayed an increased tendency to compete against women relative to men (i.e., prefer competing against perceived low-status opponents over perceived high-status opponents based on stereotypes), while men given testosterone with low basal cortisol showed the opposite pattern. We also note that the critical three-way interaction between testosterone, basal cortisol, and perceived opponent status replicated across the two blinding conditions, indicating the robustness of this complex pattern of results (see full results in the Supplemental Material).

Other Covariates

In follow-up analyses, variables related to diurnal aspects of endocrine functioning (time of day and time since awakening) and underlying participant skill level (mean points earned in the mandatory piece rate trials) did not substantially alter the results (Table S3).

Decisions to Compete After Win-Loss Feedback

We next examined decisions to re-enter competitions against a mixed-gender pool of opponents in the Feedback rounds, in which participants made decisions after being provided explicit win-loss results from prior rounds in the competition task. The added complexity of these models required removal of the random terms for social-evaluative observation and

[8]We also conducted simple slopes analyses by examining the association of basal cortisol with the opponent gender slope in the testosterone treatment and placebo conditions. These simple slopes analyses indicated that basal cortisol was unrelated to this opponent gender slope in the placebo group (B = -0.02, [-0.11, 0.07]), whereas basal cortisol was positively related to the opponent gender slope in the testosterone group (B = 0.16, [0.08, 0.25]). This pattern is consistent with the simple slopes reported in the main text. Collectively, these results are consistent with the context-dependent dual-hormone hypothesis: Basal cortisol moderates the causal effect of testosterone on decisions to enter competitions against female relative to male opponents.

opponent gender to achieve satisfactory fit. We note that none of the primary behavioral effects or interpretations were meaningfully altered between the more complex and simpler models (Table S11).

Main Effect of Testosterone Treatment

Testosterone did not robustly predict increased decisions to compete after receiving feedback (OR = 1.04, [0.54, 2.02], p = .906, p_{FDR} = .952; Table S4; Figure 3, upper panel). This result does not provide support for the challenge hypothesis.

Main Effects of Opponent Gender and Win-Lose Feedback (Opponent Status Cues)

Participants were still more likely to compete against female compared to male opponents after receiving feedback (OR = 1.90, [1.45, 2.49], p < .001, p_{FDR} < .001; Table S4, Figure 3, middle panel). Participants were also more likely to compete against an opponent that they had previously beaten (controlling for opponent gender) compared to opponents that had previously beaten the participant (OR = 4.48, [1.91, 10.52], p = .001, p_{FDR} = .003; Table S4; Figure 3, lower panel). This result indicates that win-lose feedback was also a cue to opponent status that predicted decisions to compete again.

Testosterone × Opponent Status Cues

Neither of the testosterone treatment interactions with opponent status cues were significantly related to decisions to compete again (testosterone treatment × opponent gender: OR = 0.68, [0.40, 1.16], p = 0.156, p_{FDR} = 0.311; testosterone treatment × prior outcome: OR = 0.56, [0.10, 3.00], p = 0.498, p_{FDR} = 0.725; Table S4).

Dual-Hormone Hypothesis

As in the initial phase, strong support was not evident for a concise dual-hormone hypothesis that does not account for opponent status in the feedback rounds. The effect of

testosterone treatment on competitive behavior was not robustly moderated by basal cortisol (*OR* = 0.93, [0.46, 1.88], $p = .840$, $p_{FDR} = .952$; Table S4).

Context-Dependent Dual-Hormone Hypothesis

We next tested the context-dependent dual-hormone hypothesis in the feedback rounds. Building on previous correlational work (Mehta & Josephs, 2010, Study 2), we expected that testosterone, cortisol, and win-lose feedback (a cue to opponent status) would interact to predict decisions to re-enter competitions. A robust testosterone treatment × basal cortisol × prior outcome (win/lose) interaction was found to predict decisions to compete again (*OR* = 9.55, [1.75, 52.20], $p = .009$, $p_{FDR} = .030$; Figure 4; Table S4).

To break down this three-way interaction, we examined the simple slopes comparing likelihood of competing after wins and losses for each treatment group at high (+1SD) and low cortisol (-1SD). In the placebo group, men competed more against losers (objectively lower-status opponents) compared to winners (objectively higher-status opponents) regardless of whether the men had high (OR = 3.02, [0.48, 18.81]) or low cortisol values (OR = 9.49, [2.00, 44.92]). In the testosterone group, men who had high basal cortisol levels were substantially more likely to compete again against losers (lower-status opponents) compared to winners (higher-status opponents; *OR* = 14.90, [3.13, 70.85]), whereas men with low basal cortisol were *less* likely to compete against losers compared to winners (*OR* = 0.51, [0.093, 2.85]). This pattern of results – that is, testosterone and cortisol linked with competitive behavior conditional on an opponent's status – is consistent with previous correlational research on decisions to compete against the same opponent (Study 2, Mehta & Josephs, 2010)[9].

[9]This previous study experimentally manipulated opponent status as a between-subjects factor, and therefore, the three-way interaction between testosterone, cortisol, and opponent status was interpreted by examining hormone-behavior patterns separately in each opponent status condition. Nevertheless, to facilitate comparison with the

As with the initial phase, we used another strategy to break down this three-way interaction by extracting slopes linking probability of competing against low- and high-status opponents. A positive slope value indicates a greater propensity to re-compete against low-status (prior losers) relative to high-status opponents (prior winners), and a negative value indicates a greater propensity to re-compete against high-status relative to low-status opponents. We regressed testosterone treatment condition, basal cortisol, and their interaction on opponent status slope scores, which revealed a significant treatment × basal cortisol interaction (B = 1.41, t(109) = 2.45, p = .016). We conducted simple slopes analyses by examining the effect of testosterone treatment (compared to placebo treatment) on opponent status slope scores one standard deviation above and below the basal cortisol mean. Similar to the pattern shown in Figure 4D, these analyses revealed that testosterone's effect on the opponent status slope went in opposite directions for those low and high in basal cortisol: There was a negative effect of testosterone versus placebo on the opponent status slope for low-cortisol individuals (B = -1.84, [-3.41, -0.28]) and a directionally positive effect of testosterone versus placebo on the opponent status slope for high-cortisol individuals (B = 0.97, [-0.64, 2.58]). We interpret the somewhat stronger effect of testosterone on the opponent status slope in low-cortisol relative to high-cortisol individuals as noise that is unlikely to be theoretically meaningful (for additional discussion regarding this interpretation, see Supplementary Material)[10].

present experiment, we went back to this previous study and examined the directional patterns for competitive behavior towards high- and low-status opponents as a function of testosterone and cortisol levels. The directional patterns were consistent with the present experiment's findings. For example, high-testosterone low-cortisol individuals showed an increased propensity to compete against high-status opponents relative to low-status opponents, whereas high-testosterone high-cortisol individuals were more likely to avoid competitions against high-status relative to low-status opponents. Therefore, we conclude that the interaction between testosterone, cortisol, and opponent status was consistent across the two studies, and both studies are consistent with the context-dependent dual hormone hypothesis.

[10]We also conducted simple slopes analyses by examining the association of basal cortisol with opponent-status slope scores (i.e., the difference in competing against high- vs. low-status opponents) in the testosterone treatment

Overall, these analyses reveal that testosterone treatment coupled with low basal cortisol levels was associated with a relative preference to compete more against men and re-compete more against higher status opponents (prior winners). Testosterone treatment coupled with high basal cortisol levels was associated with a relative preference to compete more against women and re-compete more against lower status opponents (prior losers). We note, again, that this pattern replicated across both blinding conditions (see Supplemental Material).

Notably, the testosterone treatment × basal cortisol × opponent gender interaction was not robustly linked to decisions to compete again in the feedback rounds, controlling for competition outcome (*OR* = 0.93, [0.52, 1.65], $p = .796$, $p_{FDR} = .910$; Table S4). The exploratory four-way interaction among testosterone treatment, cortisol, opponent gender, and outcome was not robust (*OR* = 1.02, [0.28, 3.72], $p = .976$; Table S4). Thus, after receiving explicit opponent status information via relative performance feedback (win/loss feedback), men given testosterone who had high basal cortisol were more likely to compete against lower status opponents (prior losers), rather than compete discriminately against female opponents. Men given testosterone who had low basal cortisol showed the opposite pattern. An objective indicator of relative opponent status – that is, prior victory or defeat – seems to override gender as a status cue.

Other Covariates

Controlling for time of day, time since awakening, and participant skill level did not substantially alter the three-way interaction between testosterone treatment, cortisol, and prior competitive outcome on decisions to re-enter competitions after feedback (Table S5).

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and placebo conditions. Simple slopes analyses indicated that basal cortisol was not strongly related to the opponent status slope in the placebo group (B = -0.40, [-1.22, 0.42]), whereas basal cortisol was positively related to the opponent status slope in the testosterone group (B = 1.00, [0.23, 1.78]). This pattern is consistent with the simple slopes reported in the main text. Collectively, these results are consistent with the context-dependent dual-hormone hypothesis: Basal cortisol moderates the causal effect of testosterone on decisions to enter competitions against low-status relative to high-status opponents.

Secondary Analyses

Trait dominance

We did not find strong evidence that trait dominance moderated the effect of testosterone, either alone or in interaction with basal cortisol or opponent status cues, on men's decisions to compete (Table S9). See supplemental materials for additional discussion on this topic.

Subjective Ratings of Opponents

In line with prior work on gender stereotypes (Spencer et al., 1999), female opponents were rated lower than male opponents on "good at…math" by the participants who completed the competition task (B = -0.41, [-0.68, -0.15], p = .021). This effect of opponent gender was robust to controlling for previously winning or losing to a given opponent (B = -0.36, [-0.68, -0.05], p = .039). In the follow-up sample of men who did not participate in the competition task, an effect of gender on "good at…math" was again observed (B = -0.625, [-1.18, -0.07], p = .038). Supplementary analyses with other subjective ratings (e.g. intelligent, dominant) reveal that these opponent gender effects were specific to math-ability ratings (Table S10). These results are suggestive that female opponents were perceived as lower-status opponents compared to male opponents in the math-based competition.

Exploratory Analyses

Dual Hormone Effects with Other Cortisol Measures

We repeated analyses on decisions to enter and re-enter competitions using cortisol change from before to after the competition task and cortisol AUC_G, a measure of diurnal cortisol exposure across the experimental period that encompasses the basal measure taken prior to testosterone treatment (or placebo) and the two measures taken immediately before and after the competition task. Cortisol change scores from before to after the competition task did not

robustly moderate the interaction between testosterone treatment and opponent status cues on competitive behavior (Tables S6 and S7). These exploratory analyses did reveal an unexpected, weak two-way interaction between testosterone treatment and cortisol change scores in both phases of the competition task. However, the effect was even weaker and the estimates more variable in models that did not include the higher-level interactions with opponent status cues (see Supplementary Materials). Cortisol AUC_G showed similar though weaker moderation effects compared to our primary analyses that focused on basal cortisol. The weaker effects in these analyses compared to primary basal cortisol analyses are consistent with previous work on the dual-hormone hypothesis, which suggests that basal cortisol is a key moderator of testosterone's behavioral effects in the absence of acute stress, compared to state measures of cortisol (Knight, Sarkar, et al., 2020).

Competition Score, Satisfaction, and Outcome

We next explored if the three-way interaction between testosterone treatment, basal cortisol, and opponent gender was associated with points scored, likelihood of winning, or trial satisfaction. These analyses did not reveal robust effects of the three-way interaction on any of these measures in the choice trials (Table S8). These results indicate that although testosterone treatment interacted with basal cortisol and opponent gender to predict decisions to compete, this three-way interaction did not predict performance outcomes or trial-by-trial satisfaction.

Meta-analyses

We examined the meta-effect of the interaction of testosterone, cortisol, and prior competition outcome across this experiment and the previously published correlational study (Mehta & Josephs, 2010). In the fixed-effects model, a significant three-way interaction meta-effect was evident (Fisher's Z = 0.272, [0.119, 0.425], $Z = 3.48$, $p < 0.001$; Figure 5A). We also

examined a random-effects model with both of the three-way interactions from the initial and feedback rounds included (i.e., testosterone treatment × basal cortisol × opponent gender and testosterone treatment × basal cortisol × prior outcome, respectively). This model also provided support for a three-way interaction among testosterone, cortisol, and opponent status (Fisher's Z = 0.293, [0.175, 0.411], $Z = 4.86$, $p < .0001$; Figure 5B).

Discussion

This experiment examined the causal effects of testosterone on men's competitive behavior within a context-dependent dual-hormone framework by measuring basal cortisol levels

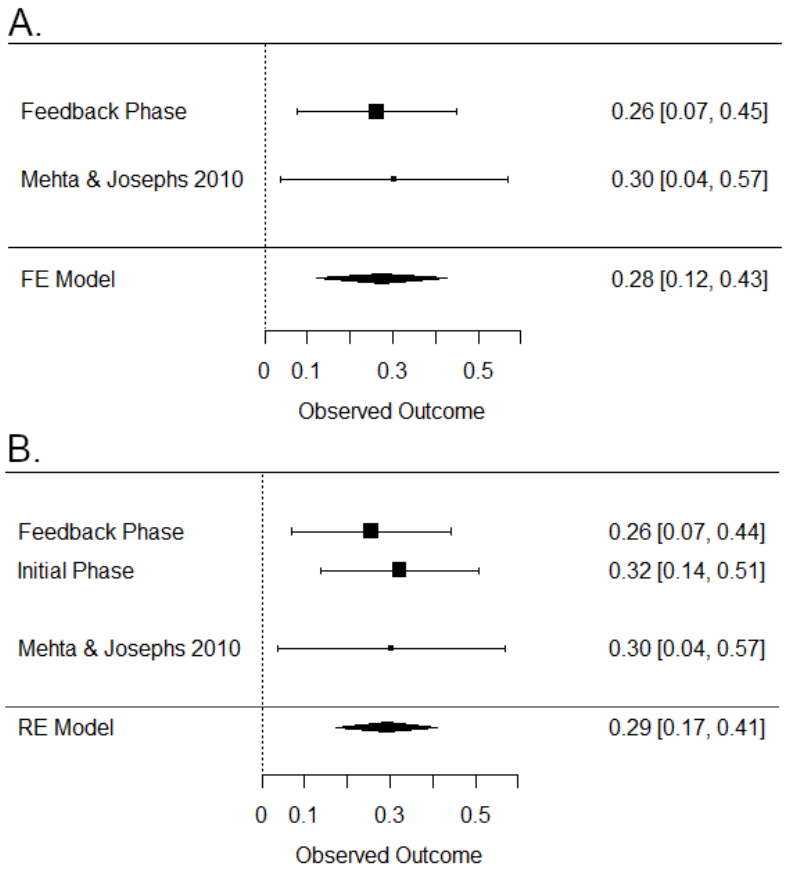

Figure 5. Meta-analyses of testosterone × cortisol × opponent status in two studies. These figures depict Fisher's *z* values. A) Fixed effects (FE) model with prior outcome as indicator of opponent status in the present experiment. B) Random effects (RE) model that includes both prior outcome and opponent gender as indices of perceived opponent status.

and pharmacologically manipulating testosterone prior to a math-based competition against male and female opponents. Prior research inconsistently linked testosterone and testosterone-cortisol interactions to competitive behavior (Dekkers et al., 2019; Nadler et al., 2021). Our results indicate that the context-dependence of testosterone-cortisol interactions is critical in explaining these inconsistencies.

Specifically, the causal effects of testosterone on competitive behavior depended on basal cortisol and opponent gender in the competition's initial rounds. After win-lose feedback was provided, testosterone's influence on decisions to re-enter competitions depended on basal cortisol and this objective opponent status cue, not gender. Compared to men given placebo, exogenous testosterone given to men low in basal cortisol promoted status-seeking behavior – competing more against male and prior-winning opponents compared to female and prior-losing opponents. Exogenous testosterone given to men high in basal cortisol induced status-loss avoidance behavior – competing more against female and prior-losing opponents compared to male and prior-winning opponents. In the present experiment, testosterone treatment's effect on competitive behavior was not robust; a robust effect of testosterone was evident only when examined within a *context-dependent* dual-hormone framework.

Our results extend existing theoretical frameworks (Archer, 2006; Wingfield et al., 1990) and correlational research (Henry et al., 2017; Mehta & Josephs, 2010) by providing experimental evidence that testosterone's influence on men's competitive behavior depends on basal cortisol levels and cues to an opponent's perceived status[11]. The present experiment used an innovative design that included pharmacological hormone administration, within-subject

[11] We did not find evidence in support of trait dominance accentuating the effects of testosterone on competitive behavior. In the supplemental materials, we discuss several possibilities that might explain this lack of support and future directions to continue to investigate the interaction between testosterone and trait dominance.

comparisons of competitive decisions toward high- and low-status opponents, multiple cues of an opponent's status, and a larger sample size compared to prior work. Further, supplemental (meta-)analyses (i) confirmed an internal replication of the context-dependent dual-hormone interaction across two opponent status cues and two blinding conditions, and (ii) provided robust evidence for this interaction across the present experiment and previous work.

Opponent gender was likely used as a status cue because the competition task was math-based and contained no explicit status indicators in the initial rounds. This interpretation is supported by subjective ratings and stereotypes of women as less skilled in math (Cheryan et al., 2017; Ellemers, 2018; Spencer et al., 1999). Moreover, objective opponent status information eliminated the testosterone, cortisol, opponent gender interaction, consistent with evidence that stereotypes influence person perception and behavior in the absence of objective, individuating information about a target (Fiske & Neuberg, 1990). The importance of gender as a status cue likely varies across competitions; other domain-specific cues may outweigh gender in certain contexts, such as cues to intelligence in a trivia contest (Talamas et al., 2016; see Supplemental Materials for further discussion).

Possible Mechanisms

Threat and reward may explain testosterone and cortisol's associations with status-seeking or status-loss-avoidance. Elevated testosterone and cortisol levels have been linked to psychological and neural markers of social threat (Denson, Ronay, et al., 2013; Enter et al., 2019; Goetz et al., 2014; Knight & Mehta, 2017; Mehta & Beer, 2010; Van Honk et al., 1998). High-testosterone, high-cortisol men may have been threatened by high-status opponents, resulting in avoiding status loss by eschewing high-status opponents and pursuing competition against lower-status opponents. Further, some previous research has linked high testosterone and

low cortisol to psychological and neural markers of reward (Duell et al., 2021; Hermans et al., 2010; Montoya et al., 2014; Op De Macks et al., 2011; Welker et al., 2015). A high-testosterone, low-cortisol individual facing a high-status opponent may have experienced reward anticipation due to the potential for gaining status, rather than a threat response.

These reward and threat mechanisms may be further informed by the biopsychosocial model of challenge and threat (Tomaka et al., 1993). Challenge and threat states – the perceived availability or lack of resources (respectively) to deal with the demands of a situation – are associated with approach and avoidant behavior, respectively (Blascovich, 2008). We speculate that high testosterone and low cortisol levels may activate a challenge state, prompting individuals to approach competitions against high-status opponents relative to low-status opponents. High testosterone and high cortisol levels may instead activate a threat state, prompting individuals to avoid high-status opponents and approach competitions against lower-status opponents. Future research can test these hypotheses by examining psychological and cardiovascular indices of challenge and threat states (Blascovich et al., 2003; Scheepers & Knight, 2020; Tomaka et al., 1993), in conjunction with dual hormone profiles.

An additional mechanism related to reward and threat may involve risk taking (Knutson et al., 2008). Two correlational studies found that testosterone levels were positively related to risk taking among men with low basal cortisol, but were unrelated or negatively related to risk taking among men with high basal cortisol (Mehta, Welker, et al., 2015; Ronay et al., 2018). Competing against a high-status opponent may be considered risky because the chances of losing are greater relative to competing against a low-status opponent.

These proximate mechanisms may inform a broader evolutionary framework that explains the impact of stress on the hormonal reproductive axis. When stress is low, it may be

evolutionarily adaptive for elevated testosterone to promote status-seeking behavior focused on challenging high-status individuals. But when stress is high, status-seeking may be inhibited because such behaviors are metabolically costly and potentially dangerous (Buchanan et al., 2003; Haselton & Buss, 2000; Maner et al., 2012), such that testosterone may instead promote status-loss avoidance behavior.

Theoretical Implications

The Social Neuroendocrinology of Status

Several studies have provided support for the dual-hormone hypothesis on attainment of social status. Specifically, higher basal testosterone is related to higher status among individuals with lower levels of basal cortisol, whereas higher basal testosterone is either unrelated or negatively related to status among individuals with higher levels of basal cortisol (Casto et al., 2019; Dekkers et al., 2019; Edwards & Casto, 2013; Ponzi et al., 2016; Sherman et al., 2016; *cf.* Mazur et al., 2015). This conditional theorizing aligns with non-human primate work, in which a search for "the" gonadal profile of status was argued to be unproductive given the strong influences of individual differences and the social context on social status outcomes (Sapolsky, 1991). The present results suggest a behavioral mechanism that may explain the testosterone-cortisol interaction as a predictor of social status. High testosterone-low cortisol individuals may attain higher status because competing against high-status opponents provides these individuals with increased opportunities to rise in the social hierarchy. Conversely, high testosterone-high cortisol individuals may fail to attain high status because they avoid competitions against high-status competitors.

Social Psychology of Hierarchies and Competition

Prior social psychological research has focused primarily on how hierarchies influence

psychology and behavior, including competitive and aggressive behavior, but has largely ignored biological factors (Buser, 2016; Fast & Chen, 2009; Hays & Bendersky, 2015; Keltner et al., 2003; Mead & Maner, 2012; van Kleef & Cheng, 2020). The present experiment points to the value of studying hormones in behavioral studies of hierarchy and competition.

Specifically, the present results suggest that social status does not indiscriminately increase or decrease competitive behavior; rather, strategic preferences to compete depend on interactions among testosterone and cortisol. Basal testosterone and cortisol levels can be considered biological individual differences (Liening et al., 2010; Mehta et al., 2008; Sellers et al., 2007) that weakly correlate with self-report measures (Grebe et al., 2019; Sundin et al., 2021) and operate largely outside of conscious awareness (Akinola et al., 2016; Josephs et al., 2006; Schultheiss et al., 2005; Terburg et al., 2012). Hence, hormones may be critical to advance theories of hierarchy and competition given the unique role of hormones in influencing behavior beyond standard self-report measures.

Mixed-gender Hierarchies

This experiment fills an important empirical gap by studying men engaging in a math-based competition against male and female opponents. The current findings may have implications for real world hierarchies and the representation and advancement of women within them. Women are under-represented in many hierarchies, including in science, technology, engineering, and math (STEM) fields, and are less likely to advance to high-status positions, in part due to hyper-competitive and other caustic behaviors directed toward them (Berdahl, 2007; Cheryan et al., 2009, 2017; Dasgupta & Stout, 2014; Flory et al., 2015; Glick & Fiske, 2001; Gruber et al., 2020; London et al., 2012; Vandello et al., 2008; Welde & Laursen, 2011). Our results imply that high-testosterone, high-cortisol men may engage in maladaptive competitive

behaviors (e.g., aggression, derogation, harassment) directed towards women and low-status individuals and deferential behaviors towards men and high-status individuals. This line of research at the intersection of biological and social factors that guide status-relevant behavior may help improve gender representation and working conditions in mixed-gender environments.

Limitations and Future Directions

We studied male participants due to restrictions on the use of exogenous testosterone in the experiment's location. Future work should test the generalizability of the present results to female participants (see Henry et al., 2017 for related work in females).

We measured basal cortisol levels because the dual-hormone hypothesis specifically focuses on basal cortisol as a dispositional measure (Dekkers et al., 2019). Future work may consider several options to improve measurement of trait-like basal cortisol, including collecting multiple samples prior to testosterone administration, examining diurnal endocrine rhythms over multiple days, or assaying hormones from hair (Grotzinger et al., 2018; Ronay et al., 2018).

The present experiment manipulated testosterone levels because of the foundational theory on testosterone and competitive behavior (Archer, 2006; Wingfield et al., 1990). An important next step is to conduct dual-systems pharmacology experiments in which the HPG and HPA axes are manipulated simultaneously. Such dual-systems protocols are not readily available and therefore will require rigorous pharmacokinetics testing before theory-driven psychological studies are conducted and interpreted[12]. A complementary approach is to manipulate testosterone levels indirectly via contextual manipulations rather than pharmacology (Kordsmeyer & Penke, 2019; Roney et al., 2007). Pharmacological and contextual approaches have strengths and

[12] It is not clear that manipulating both hormones simultaneously is feasible. Simultaneous manipulation could result in strong, negative feedback that reduces associations between biology and behavior or otherwise produces unexpected results (Rubinow et al., 2005). This experimental work also would need to aim to manipulate longer-term, basal HPA axis functioning to accommodate the focus on basal cortisol in the dual-hormone hypothesis.

limitations; we encourage adoption of both approaches or hybrid designs to build cumulative knowledge about testosterone-cortisol interactions on social behavior.

Cortisol levels can also be contextually manipulated via laboratory stressors (Dickerson & Kemeny, 2004), and cortisol reactions in the context of acute stress have been shown to moderate testosterone's association with status-relevant behavior (Prasad et al., 2017, 2019; see also Nitschke & Bartz, 2020)[13]. Despite the focus of prior dual-hormone studies and the present experiment on basal cortisol and its slower, genomic mechanisms (Dekkers et al., 2019; Tilbrook, 2000), acute cortisol reactions to stress may impact testosterone's association with behavior via faster, non-genomic mechanisms (Makara & Haller, 2001; Moore & Evans, 1999). Some evidence even suggests opposing effects of basal and state cortisol on behavior (reviewed in Montoya et al., 2012) . Although 'trait versus state' (i.e., basal versus acute cortisol) hormone distinctions are foundational to endocrine research, the psychosocial implications of these distinctions need further examination.

We chose to examine testosterone and cortisol because they have been widely studied across species in the context of hierarchy and competition. Yet, the HPG and HPA axes are complex and highly integrated (Rubinow et al., 2005; Viau, 2002). Thus, it will be important for future work to study other factors within these hormone axes, such as hypothalamic and pituitary hormones that regulate the release of cortisol and testosterone, other gonadal hormones like estradiol (Blake et al., 2017; Stanton & Schultheiss, 2007; Tackett et al., 2015), and androgen and glucocorticoid receptors. The HPA and HPG axes are also linked with the autonomic nervous system. For example, the sympathetic nervous system physiologically mediates co-

[13] Although we did not find evidence that cortisol levels rose during the competition task in the present experiment, future work may consider other competitive tasks to reduce the possibility of math eliciting a cortisol response.

activation of cortisol and testosterone responses in the context of threat by lowering HPG axis sensitivity to glucocorticoids (Chichinadze & Chichinadze, 2008; Wingfield & Sapolsky, 2003). Thus, we recommend that future studies build on this prior work by examining potential integration with the autonomic nervous system.

Conclusion

According to prevailing theories, increased levels of testosterone should directly increase competitive behavior (Archer, 2006; Wingfield et al., 1990). The results of our experiment indicate that a more nuanced perspective on the role of this hormone in regulating competitive behavior is needed. Using a mixed-gender math-based competition, we found that testosterone's influence on men's competitive decisions depends on basal cortisol and cues to an opponent's perceived status. Testosterone treatment in low-cortisol men evoked status-seeking behavior: An increased preference to compete against male opponents and previous winners (cues to high perceived status) compared to female opponents and previous losers (cues to low perceived status). Testosterone treatment in high-cortisol men evoked status-loss avoidance behavior: An increased preference to compete against female opponents and previous losers compared to male opponents and previous winners. Key next steps include identifying mechanisms and testing for similar interactions between biology and social context in real-world, mixed-gender hierarchies.

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Supplemental Materials

The Causal Effect of Testosterone on Men's Competitive Behavior is Moderated by Basal Cortisol and Cues to an Opponent's Status: Evidence for a Context-Dependent Dual Hormone Hypothesis

Supplementary Methods

Participants

Exclusion Criteria.

We briefly reported screening and recruitment procedures in the main document. Here we provide a full list of exclusion conditions in the screening process. After being read the list, the participants self-reported if any of the conditions were true in a pre-experiment phone call:

- Student-athlete or other professional for whom steroid hormone use is prohibited.
- Mental illness, including recurrent major depression, antisocial personality disorder, Schizophrenia, bipolar disorder, Tourette's syndrome, conduct disorder, serious emotional disturbance, intermittent explosive disorder
- Alcohol or drug dependency, including opiates, LSD, methamphetamine, cocaine, solvents, cannabis, or barbiturates
- A major neurologic condition such as recent head injury with loss of consciousness, tumor, stroke, or other brain lesions.
- History of autonomic failure
- History of clinically significant liver, heart, lung, obstructive respiratory, kidney, cerebrovascular disease, or metabolic syndrome
- Current periodontitis
- Diabetes
- Irregular sleep/wake rhythm (e.g., regular nightshifts or cross timeline travel)
- Any hormone disorders
- Any immune disorders
- Medical conditions affecting testosterone concentrations (such as hypogonadism or prostate cancer), taking psychotropic medications (such as SSRIs), or receiving medical treatment for conditions affecting cerebral metabolism and blood flow (such as hypertension)
- Receiving psychiatric treatment
- Receiving endocrine treatment, such as hormone replacement therapy
- Regularly using corticosteroids, like hydrocortisone
- Regularly using anabolic steroids

Participants who acknowledged that any of these situations, conditions, or disorders were true were excluded from recruitment for participation in the study.

Participant Diversity

We aimed to maximize the diversity of our sample by recruiting participants on campus and within the community. Participants self-reported race/ethnicity information, their current student status and educational attainment, and several indicators of objective socioeconomic status (i.e., parents' and own education level and the estimated annual income of the participant, his parents, and his family). Over 28% of our sample identified as non-white, which generally reflects the diversity of the community in which the experiment was run (26.7% non-white; (United States Census Bureau, 2010). A majority of the participants reported being a current student (94%) but the range of degrees attained varied, with 73% reporting have not received a

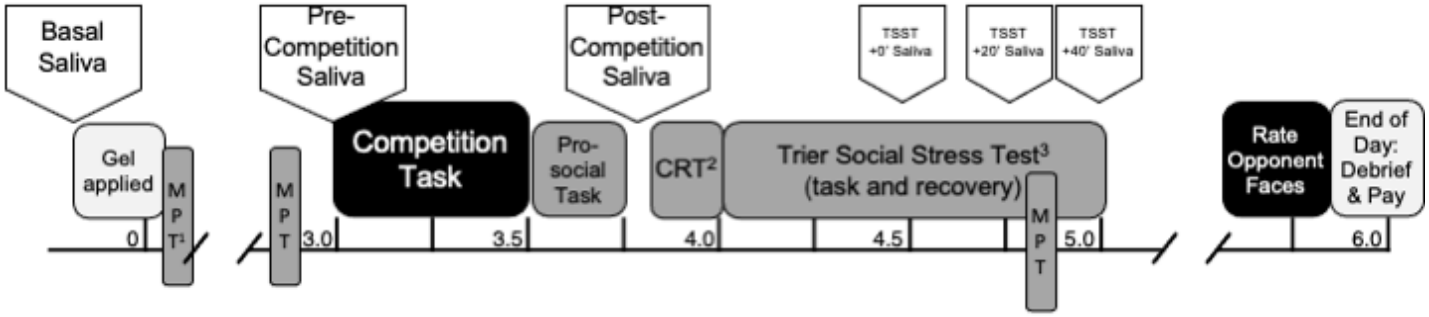

Figure S1. Timeline of experimental protocol. Time is listed in hours from gel application and are approximations. Black boxes with white font are the portions of the protocol during which data were collected for this report. Saliva samples (downward-pointing pentagons) analyzed in the present report were collected at three time points; three additional timepoints (smaller pentagons) associated with a stress task are reported elsewhere (the post-competition saliva sample was labeled as "Pre-stress" in one prior report; Knight et al., 2017). The pro-social task consisted of a dictator game decision-making task (unpublished). MPT = motivated persistence task, in which participants are asked to squeeze a hand-grip device for as long as they can. Instructions for each task were given immediately prior to the start of each task, with the exception of the competition and pro-social tasks, which were described approximately an hour prior to the start of the competition task (not pictured). No instructions or detailed descriptions of task were given prior to the collection of basal saliva. Prior reports (denoted with superscripts): 1) Knight, 2017; 2) Knight, McShane, et al., 2020; 3) Knight et al. 2017

post-secondary degree. Our sample also self-reported relatively diverse socioeconomic demographics, including a wide array of parental educational attainment (e.g., 49% of mothers obtained less than a college degree) and annual incomes (e.g., 33% of participants' family annual incomes reported below $50,000; Table S1).

Protocol

Timeline

We have included a timeline of the full-day experimental protocol (Figure S1).

Competition Task Tutorial

As part of the competition task, participants were guided through a tutorial of the task and completed an in-person, multiple-choice, verbal quiz based on the information covered in the tutorial. If a participant gave an incorrect answer, the experimenter provided feedback and described why his answer was incorrect. The quiz consisted of the following questions:

1. In COMPETE rounds, how much money can you earn?
    a. $4 per point
    b. $4 per point, but only if you win that round
    c. $4 total
    d. $4 total, but only if you win that round

2. In PIECE RATE rounds, how much money can you earn per point?
    a. $2 per point, but only if you win that round
    b. $2 per point
    c. $4 total
    d. $2 total

3. Describe a mandatory compete round
    a. A round where the computer chooses for you to compete
    b. A round where you have chosen to compete
    c. A piece rate round
    d. Any round where you competed

4. What happens after the feedback portion where you make choices to either compete again or play for a piece rate?
    a. The experiment ends.
    b. You then play out all of your choices.
    c. You then play out one randomly drawn choice.

5. How will you be paid based on your decisions in this task at the end of the day?
    a. I will not be paid based on my decisions in this task.
    b. I will be paid based upon my total earnings in this task.
    c. I will be paid one round from each condition selected at random, plus the round after the feedback portion.

Participants did not finish the tutorial until all questions had been successfully answered.

Subjective Ratings of Opponents.

To investigate gender differences in subjective ratings of opponents, we submitted participants' ratings and the follow-up sample's ratings to separate multilevel models for each rating. In order to account for variance due to rater and target and avoid problems inherent to arbitrarily aggregating across raters or targets (Judd et al., 2012), we included a random intercept and slope for gender for each participant and a random intercept for each opponent. Thus, for opponent *i* and participant *j*, our models consisted of the following:

Level 1:

$$Rating_{ij} = \beta_0 + \beta_1 Gender_i + r_{ij}$$

Level 2:

$$\beta_0 = \gamma_{00} + e_{0i} + e_{0j}$$

$$\beta_1 = \gamma_{10} + e_{1j}$$

In a follow-up analyses, we examined the effect of opponent gender controlling for the prior competition outcome (win = 1, loss = 0) and, combining data across both sets of raters, we included a term to denote which sample a rater was from (testosterone administration participant versus follow-up male sample) and the cross-level interaction between sample and gender.

Power Simulations

We examined the power to detect a range of possible logit effect sizes associated with the three-way interaction between testosterone treatment, cortisol, and opponent status (defined by opponent gender or prior win/lose feedback) given a sample size of n = 120 split evenly between treatment groups and assuming weak effects (logit(p) = 0.2) for all lower-order main effects, interactions, and covariates. Cortisol was simulated as a normally distributed variable across the sample. Random intercepts and slopes (i.e., for the effects of opponent status and public/private trials) per participant were included with assumed covariance of 0 and variance of 0.5 for each random variable. The model was simulated 1000 times at each logit value between logit(p) = 0.2 and logit(p) = 1.0 in increments of 0.1. Each model contained k = 16 simulated trials (50% lower status opponent) for each participant. Results from these simulations indicate that the experiment was 80% powered to detect a three-way interaction of logit(p) = 0.7 (OR = 2.0; Figure S2). When Fisher's Z estimates of this effect and a prior correlational study (Mehta & Josephs, 2010) were calculated, the effect size that the experiment was 80% powered to detect (Fisher's Z = 0.250) was determined to be smaller than the prior correlational results (Fisher's Z = 0.303).

For comparison to a one-shot competition task, in which a participant makes a single decision to compete or not, we ran a simulation that mirrored our principal simulations. We examined a simulated sample of n = 120 participants split evenly between testosterone treatment and placebo. Cortisol values were randomly generated from a standard normal distribution. Half of the participants were assumed to be exposed to a high-status opponent and half to a low-status

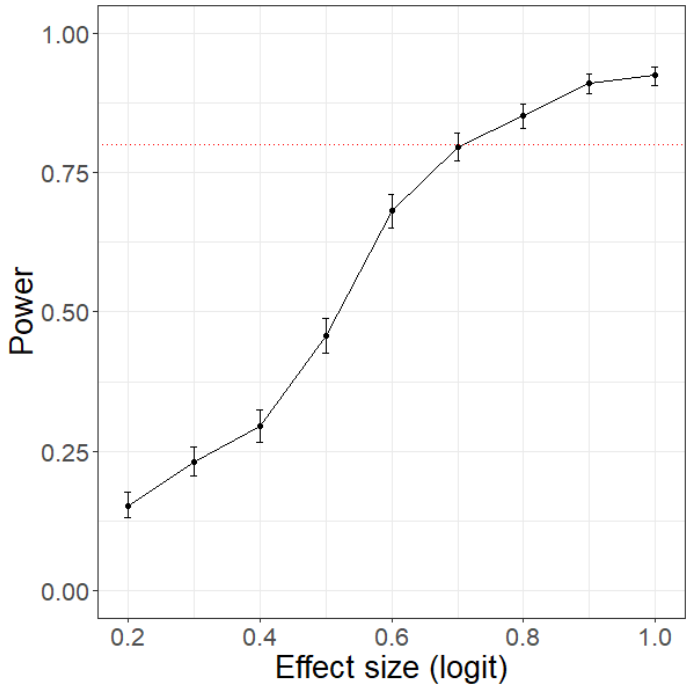

Figure S2. Power simulation results. Red dotted line indicates 80% power. Error bars represent 95% confidence intervals of the power to detect a given effect size. See OSF page for code to run this simulation.

opponent. We used the results from the simulation above (i.e., logit(p) = 0.7) as coefficient for the three-way interaction between testosterone treatment, cortisol, and opponent status within a (single-level) binomial linear regression model. The results from this comparison simulation indicate that a one-shot study design would have had 10.6% power to detect the effect that our principal simulations indicate that we were 80% powered to detect in our within-subjects approach. Hence, although evidence indicates that higher-order interactions are generally underpowered in the social psychological literature (Blake & Gangestad, 2020), the within-subjects approach helps maintain high power.

Supplementary Results

Hormone Concentration Distributions

We measured testosterone and cortisol in saliva collected at baseline (i.e., basal measures), immediately before, and approximately 15 minutes after the competition task. Distributions of raw testosterone and cortisol concentrations for each of these samples are illustrated in Figure S3.

Main Effects Not Reported in the Main Document

Basal Cortisol

Basal cortisol was not robustly associated with decisions to compete in the initial phase (*OR* = 1.22, [0.86, 1.73], *p* = .273) or in the feedback phase of the competition task (*OR* = 1.29, [0.93, 1.79], *p* = .124).

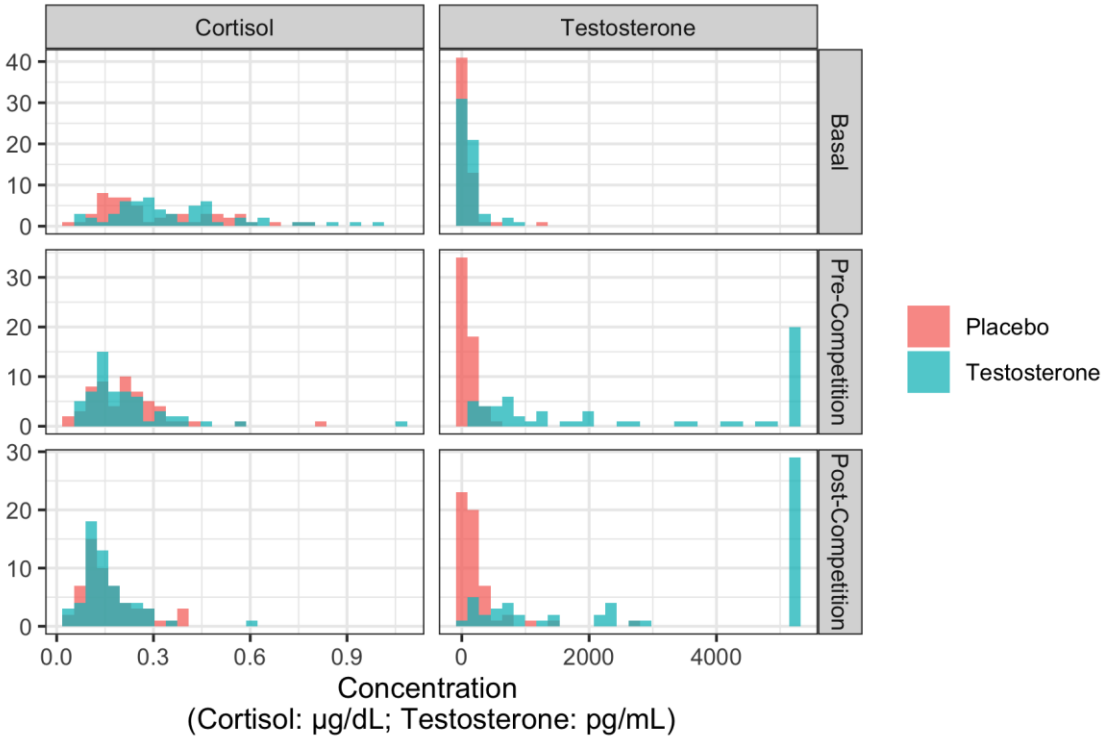

Figure S3. Distribution of hormone concentrations at three time points.

Blinding Manipulation

We examined the direct effect the experimental blinding manipulation – that is, a participant being told whether they had received testosterone or placebo or not – had on decisions to compete. The experimental blinding manipulation did not affect decisions to compete in the initial phase (OR = 1.07, [0.55, 2.07], p = .841) or in the feedback phase of the competition task (*OR* = 0.82, [0.42, 1.61], *p* = .572).

Social-Evaluative Manipulation

As discussed in the main document, the experimental task attempted to manipulate social-evaluation by including public and private conditions within the competitive task (Cottrell et al., 1968; Grush, 1978). We explored whether a social-evaluative condition might enhance testosterone's effects on behavior based on prior theorizing that testosterone directs motivations and behavior meant to seek or maintain social status. Specifically, having an audience was predicted to boost the stakes of the competition for men given testosterone treatment in terms of the competition's effect on social status. We therefore included this manipulation (i.e., the words "Public" or "Private" with open or closed eyes on the screen; Haley & Fessler, 2005) as an exploration of the extent to which having an evaluative audience might alter testosterone treatment's effects on decisions to enter competitions and decisions to re-enter competitions after feedback.

The social-evaluative manipulation did not moderate testosterone treatment's direct effects on decisions to enter competitions (T × Social Evaluation: *OR* = 0.76, 95%CI[0.47, 1.23], *p* = 0.262), did not moderate the interactive effects of testosterone treatment and basal cortisol (T × Cortisol × Social Evaluation: *OR* = 0.79, 95%CI[0.47, 1.32], *p* = 0.369) or opponent gender (T × Opponent Gender × Social Evaluation: *OR* = 0.68, 95%CI[0.27, 1.70], *p* = 0.410), and did not moderate the three-way interaction between testosterone treatment, basal cortisol, and opponent gender (T × Cortisol × Opponent Gender × Social Evaluation: *OR* = 1.50, 95%CI[0.56, 4.01], *p* = 0.422).

Similarly weak effects were found in the feedback portion of the experiment, in which the social-evaluative manipulation did not moderate testosterone's direct effects on decisions to re-enter competitions (T × Social Evaluation: *OR* = 0.87, 95%CI[0.50, 1.50], *p* = 0.610), did not moderate the interactive effects of testosterone treatment and basal cortisol (T × Cortisol × Social Evaluation: *OR* = 0.72, 95%CI[0.40, 1.27], *p* = 0.254) or prior competitive outcome (T × Prior Outcome × Social Evaluation: *OR* = 2.29, 95%CI[0.72, 7.32], *p* = 0.161), and did not moderate the three-way interaction between testosterone treatment, basal cortisol, and prior competitive outcome (T × Cortisol × Prior Outcome × Social Evaluation: *OR* = 0.67, 95%CI[0.18, 2.50], *p* = 0.550).

These null effects of social evaluations on testosterone's effects on behavior may be interpreted in several ways; we provide a non-exhaustive list here to help inform future research on the topic. First and most parsimoniously, perhaps testosterone's effects on competitive behavior are not altered by cues that indicate social evaluation, in line with some recent work on competitive motivation (Losecaat Vermeer et al., 2020) but in contrast to other recent work on charitable donations (Wu et al., 2020). Second, the manipulation itself may have failed to elicit the desired feelings of social evaluation. That is, there may be effects of social-evaluative versus non-social evaluative competitions, but our manipulation failed to evoke the desired valence or magnitude of an effect. Finally, the social-evaluative cues may have altered behavior in some

complex, interactive way that our study was underpowered to test. Future research might consider examining more explicit social-evaluative conditions (i.e., having rounds of competitions with an experimenter or other participants in the room versus playing competitions alone) and running larger studies to better estimate the effects of social-evaluative contexts on testosterone's link with social behaviors.

Dual-hormone Effects with Other Cortisol Measures

Exploratory analyses revealed an unexpected, weak testosterone treatment × cortisol change interaction on decisions to compete (Initial phase: *OR* = 2.22, [1.07, 4.58], *p* = .032; Feedback phase: *OR* = 2.28, [0.92, 5.67], *p* = .077; Tables S6 and S7). When the models were re-run without moderation by opponent status cues as an exploratory follow-up, the testosterone treatment × cortisol change interaction effects were somewhat weaker and non-significant (Initial phase: *OR* = 1.95, [0.99, 3.84], *p* = .055; Feedback phase: *OR* = 1.80, [0.90, 3.61], *p* = .095). The pattern of this interaction indicates that men given testosterone treatment with relatively high cortisol change (i.e. less of a circadian decline or a slight increase in cortisol) were more likely to compete, whereas men given testosterone with relatively low cortisol change (circadian decline in cortisol) were less likely to compete.

This weak interaction effect may be interpreted in several ways; we provide a non-exhaustive list of challenges with interpretation to help inform future research. First, this interaction may be a false positive; after all, it was unexpected, weak, and not robust in all analyses. Second, if this interaction is real, the causal direction is unclear. That is, it is not clear if testosterone interacted with cortisol change to influence competitive behavior, or perhaps testosterone interacted with competitive behavior to influence changes in cortisol in response to the competitive task. Third, cortisol decreased, on average, from before to after the competitive task consistent with circadian decline (Table S1). Thus, it remains unclear whether these cortisol changes were influenced by the competitive task at all or whether they were influenced primarily by circadian rhythms. Further complicating the interpretation is the unrelated decision-making task that occurred immediately after the competition task and immediately before the final cortisol measure. Future work interested in examining testosterone treatment's interaction with cortisol change dynamics as predictors of competitive decision-making will require study designs that measure or manipulate acute cortisol change prior to a competitive decision-making task (Prasad et al., 2017, 2019).

Replication of Testosterone Treatment, Basal Cortisol, and Opponent Status Interactions Across Blinding Conditions

In the main document, we reported analyses controlling for blinding condition. Here we examine the four-way interactions among testosterone treatment condition, basal cortisol, opponent status (opponent gender or prior outcome), and blinding condition. This four-way interaction was not robust in the initial phase (*OR* = 0.97, [0.32, 2.95], *p* = 0.954) or in the feedback phase (*OR* = 0.78, [0.02, 25.81], *p* = 0.891). Further, when point estimates of the three-way interactions were investigated after splitting the sample by blinding condition, the pattern of effects was evident across each condition (initial phase, double blind: *OR* = 2.84, [1.23, 6.56], *p* = 0.015; initial phase, single blind: *OR* = 2.70, [1.25, 5.83], *p* = 0.011; feedback phase, double blind: *OR* = 9.77, [1.07, 89.33], *p* = 0.044; feedback phase, single blind: *OR* = 11.43, [0.68,

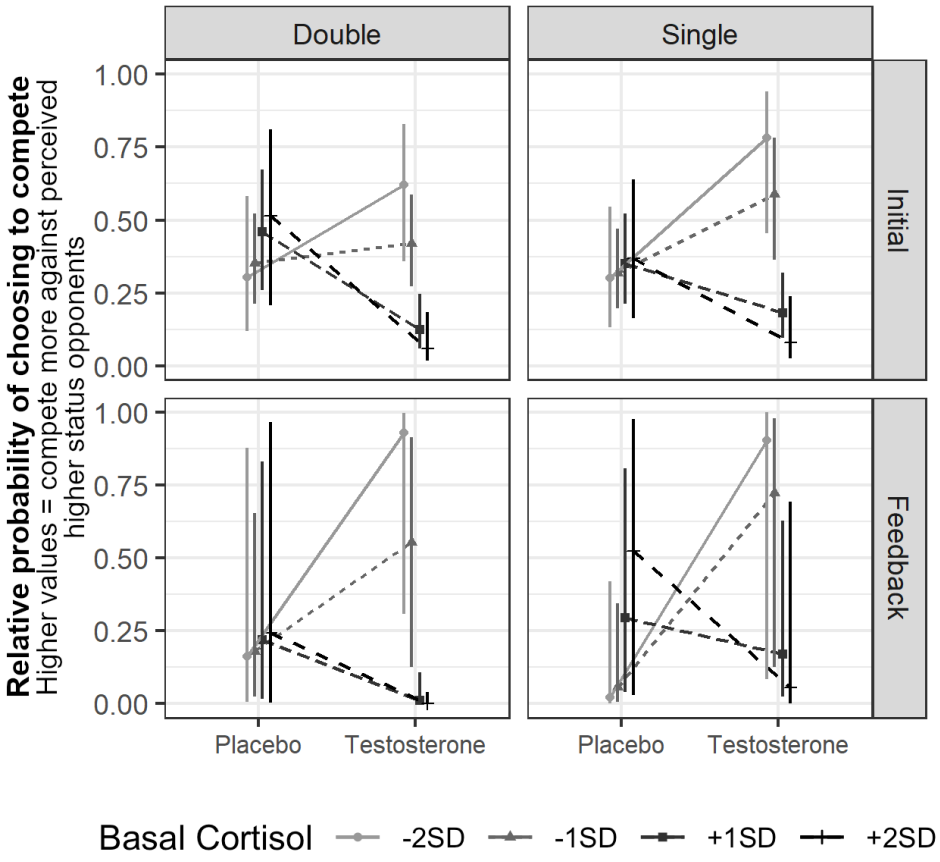

Figure S4. The simple slopes comparing probabilities of competing against high and low status opponents plotted for the testosterone and placebo groups at +/-1 and 2 SD. Higher values indicate a higher probability of competing against male opponents compared to female opponents in the initial phase and a higher probability of competing against prior winners compared to prior losers in the feedback phase. Probabilities were calculated from simple slope logits extracted from the three-way interactions. For illustrative purposes and to better match the prior literature, the probabilities were inverted (i.e., "1 – *p*(Compete)"). A probability of 0.50 (marked by the dotted line) indicates no preference for competing against higher or lower status opponents. Error bars represent 95% confidence intervals.

191.9], $p = 0.091$; Figure S4). These analyses demonstrate that the context-dependent dual-hormone hypothesis replicated internally across two experimental blinding conditions, indicating the robustness of this complex pattern of results.

Trait Dominance

We explored trait dominance as a moderator of testosterone's effects on decisions to compete in both phases of the competition task. None of the analyses provided strong evidence that trait dominance moderated the effect of testosterone on men's decisions to compete, either alone or in interaction with basal cortisol or opponent status cues (Table S9).

Subjective Ratings of the Opponent

The main document reports the effects of opponent gender on ratings of whether an opponent was "good at…math." Here we report eight other variables on which the participants rated opponents: Attractive, dominant, intelligent, mature, warm, "I feel close to this person," respect, and "I performed better than this person in the competition." Among these rating categories, only "I performed better" was associated with a robust effect of gender – participants rated female opponents as higher on this variable (B = 0.35, [0.14, 0.56], p = .013), indicative of having performed better than female opponents on average (Table S10B). The remaining variables were estimated with relatively high variance among participants, suggesting that participants did not readily agree on gender stereotypes among the remaining variables.

Model Fit Statistics

We report model fit statistics (AIC and BIC) for each of the principal models (Table S12). In each phase of the task, the context-dependent dual-hormone hypothesis model (that is, the model that contained the testosterone treatment by basal cortisol by opponent status interaction term) was considered the best fitting according to having the lowest AIC score. BIC penalizes model complexity more than AIC and so higher-order interaction models had consistently higher BIC scores than lower-order models. Of the three-way interactions modeled in the feedback phase (that is, testosterone treatment × basal cortisol × opponent gender or prior win/lose), the interaction with prior win/lose was found to be better fitting (lower BIC score) than the opponent gender model, suggesting prior win/lose is the more parsimonious result of the two.

Supplementary Discussion

The results of this experiment support the context-dependent dual-hormone hypothesis in showing that the causal effects of testosterone on competitive decisions depend on basal cortisol levels and two opponent status cues (opponent gender and win/lose feedback). Future research will be helpful for confirming the nature of the three-way interactions. The patterns reveal an internal replication of the context-dependent dual-hormone interaction as demonstrated by the meta-analysis, the global patterns shown in Figure 4, and the point estimates in the simple slopes analyses.

There was also a pattern in the simple slopes analyses suggesting that the influence of testosterone treatment (versus placebo) on decisions to compete against female relative to male opponents was somewhat stronger for the high-cortisol side of the basal cortisol distribution, whereas the influence of testosterone treatment (versus placebo) on the propensity to re-compete against low-status opponents (prior losers) relative to high-status opponents (prior winners) was somewhat stronger for the low-cortisol side of the distribution. We interpret these slight differences as driven by noise that is unlikely to be theoretically meaningful. We draw this conclusion for the following reasons. First, the overall patterns of the three-way interactions were clearly very similar as revealed in Figure 4. Second, our best guess for any particular effect is the point estimate, and the point estimates in the simple slopes analyses indicate that the three-

way interactions were driven by both the low- and high-cortisol sides of the basal cortisol distribution. Third, which side of the basal cortisol distribution showed a somewhat stronger effect of testosterone was different in the two three-way interactions, indicating that the patterns were unstable and did not replicate. Fourth, there is no strong theoretical reason to expect divergent patterns in the two three-way interactions, whereas there is reason to expect slight differences in patterns due to normal statistical variation. We welcome additional studies that further investigate these three-way interactions.

Cues to Perceived Opponent Status in Other Domains

Some evidence suggests humans infer status by a universal set of gender-differentiated characteristics (Buss et al., 2020; Durkee et al., 2020), but the exact cues that signal perceived status in a competitive setting likely depend on the nature of the competition. A competition based on a domain in which women are stereotyped to be more skilled than men – for example, based on word puzzles or other verbal task instead of math (Dreber et al., 2014; Hausmann et al., 2009; Josephs et al., 2003; Niederle & Vesterlund, 2011; Wozniak et al., 2014) or based on a jewelry-making task instead of physical strength in a hunter-gatherer population (Apicella & Dreber, 2015) may cause men with high testosterone and high cortisol levels to pursue male opponents as easy targets and avoid female opponents. Given our 1) theorizing on testosterone-cortisol profiles producing status-seeking versus status-loss avoidance motivation, 2) our focus on context-dependence, and 3) the results indicating the impermanence of subjective status cues, we believe it is likely that testosterone and cortisol will flexibly direct decisions to compete based on domain-specific stereotypes, rather than generally targeting women. Future research that includes multiple competitive domains will be able to test this possibility directly.

Trait Dominance

Exogenous testosterone's effects on decisions to compete did not appear to be moderated by trait dominance in the present experiment. An interaction between testosterone and trait dominance was explored because of prior work suggesting that testosterone's effects on status-relevant behavior (Carré et al., 2009, 2017; Losecaat Vermeer et al., 2020; Mehta et al., 2015; Slatcher et al., 2011) and on responses to status-relevant stress (Knight et al., 2017) are heightened among individuals who are high in trait dominance. However, as discussed in the main document (Methods section), other findings within this domain are nuanced. For instance, trait dominance interactions with exogenous testosterone may be specific to certain contextual aspects of a competitive setting (Losecaat Vermeer et al., 2020), may operate as part of a broader set of risk factors (Geniole et al., 2019), or may not be evident (Kutlikova et al., 2021; Welker et al., 2019). In at least two instances, an overall interaction term between testosterone and trait dominance (and cortisol) was not-significant but follow-up analyses suggested that an effect of testosterone (and cortisol) was more evident among men who are higher in trait dominance (Losecaat Vermeer et al., 2020; Pfattheicher, 2017).

Several factors may help explain the nuanced results in the literature and the lack of robust effects in the present experiment. First, the focus on testosterone and trait dominance is relatively less developed than other theoretical frameworks such as the challenge hypothesis or the dual-hormone hypothesis. As such, the known findings on testosterone's interactions with

trait dominance may be part of an initial exploratory phase for the field before more and larger studies can provide a better understanding of the theoretical framework. The relatively small size of the literature also leaves open the possibility that the initial effects reported may be prone to publication bias.

Second, the definition of trait dominance, and in turn, the scale used to measure trait dominance varies across studies. This possible "jingle fallacy" – in which separate psychological constructs are given the same name despite inherent differences (Block, 1995) – could lead to inconsistent findings in the field. In the present experiment, we used a trait dominance scale based on a definition of dominance as the use of force, fear, and intimidation to earn status (Cheng et al., 2013; e.g. "I am willing to use aggressive tactics to get my way."). Prior research indicates that trait dominance measured with this scale moderated the effect of exogenous testosterone on aggressive behavior (Carré et al., 2017; Geniole et al., 2019) and emotional states related to aggression such as hostility (Knight et al., 2017), but not other types of status-relevant behaviors such as competitive persistence (Kutlikova et al., 2021). Other researchers have employed trait dominance scales that focus on assertiveness and a desire for positions of authority and status, rather than force, fear tactics, and intimidation (e.g., the PRF dominance scale, "I would like to be an executive with power over others"; Jackson, 1984). Trait dominance measures with these scales did show some evidence of strengthening testosterone's effects on competitive behavior (Losecaat Vermeer et al., 2020; Mehta et al., 2015; Slatcher et al., 2011). Based on this pattern of results, trait dominance measured with scales that focus dominance as the use of force, fear, and intimidation to gain high rank may be more likely to heighten the effects of testosterone in studies that measure these types of anti-social dominant behaviors (e.g. aggressive behavior). But trait dominance measured with scales that focus on a desire to attain high-status positions may accentuate testosterone's effect on behaviors such as competitive decision-making (Mehta et al., 2015). However, no work to our knowledge has rigorously examined testosterone's interactions with various measures of trait dominance across different types of status-relevant behaviors.

Third, the psychopharmacogenetic approach used by Geniole and colleagues (2019) also found that trait dominance worked within a broader personality risk factor that accentuated the effects of testosterone on aggressive behavior. As discussed in the main document, this same experiment did not find a significant moderating effect of trait dominance on its own, although the effects were of a similar magnitude and direction as the broader personality risk factor. Combined with our speculation above about measurement of trait dominance, these results suggest that a broader approach that combines dominance-relevant traits may be necessary to find stable moderating influences of explicit, self-reported personality constructs on testosterone's association with behavior. By examining only one scale that may be part of a broader personality risk factor, results may be prone to instability.

Fourth, in this same psychopharmacogenetic work (Geniole et al., 2019), testosterone and trait dominance's effects on aggressive behavior depended on a gene polymorphism that alters efficacy of the androgen receptor [the cytosine-adenine-guanine (CAG) repeat in exon 1 of the androgen receptor]. Fewer CAG repeats, reflective of more effective androgen receptors (Chamberlain et al., 1994), heightened the interactive effects of testosterone treatment and trait

dominance on aggressive behavior. Hence, trait dominance's interactions with testosterone may further depend on factors that were not measured in the present experiment.

This non-exhaustive and non-exclusive set of possibilities suggests that more work is necessary. Work focused on these issues may need to administer several forms of explicit trait dominance, other related personality measures, consider other relevant moderators (e.g. androgen receptor gene) within varying behavioral assays to improve our understanding of trait dominance and its interactions with endocrine systems. Studying multiple forms of trait dominance across multiple behavioral assays will also help determine the specificity (or malleability) of the putative interactions between trait dominance and testosterone. Research could also attempt to experimentally heighten or reduce trait dominance levels (Roberts et al., 2017) in order to better understand causal, mechanistic pathways linking testosterone, cortisol, and trait dominance with status-relevant behavior.

Supplementary Tables

Table S1. Indicators of racial/ethnic and socioeconomic diversity

Self-identified Race/Ethnicity	
White/European-American	73%
African-American	2%
Asian/Asian-American	12%
Hispanic/Latino	8%
Middle Eastern/Middle-Eastern American	3%
Native American	1%
Pacific Islander	1%
Other	2%
Total non-white	28%

Education[1]	**Mother**	**Father**	**Self**
Some high school	8%	7%	
High School diploma or GED	18%	14%	73%
Some college	10%	13%	
Associate degree	13%	6%	16%
Bachelors degree	34%	32%	8%
Masters degree	11%	12%	1%
PhD or Professional School degree	6%	14%	1%
Unsure or do not know	0%	2%	

Income	**Mother**	**Father**	**Self**	**Family**
<$24,999	32%	13%	86%	15%
$25,000 to $49,999	27%	17%	3%	18%
$50,000 to $74,999	21%	18%	2%	8%
$75,000 to $99,999	9%	14%	0%	8%
>$100,000	8%	32%	0%	41%
Unsure or do not know	3%	7%	10%	20%

Note:

1. Response options to questions about parent and own educational attainment differed. Blank cells in the "Self" column were not available options for participant responses.

Table S2: Effects of testosterone treatment, basal cortisol, and opponent gender on initial phase decisions to enter competitions

	Decisions to compete: Main Effects			Decisions to compete: T/P × Cortisol			Decisions to compete: T/P × Gender			Decisions to compete: T/P × Cortisol × Gender		
	Odds Ratio	*CI*	*p*	*Odds Ratio*	*CI*	*p*	*Odds Ratio*	*CI*	*p*	*Odds Ratio*	*CI*	*p*
Fixed Effects												
(Intercept)	1.13	0.60 – 2.14	.709	1.12	0.59 – 2.12	.737	1.24	0.64 – 2.38	.525	1.25	0.65 – 2.40	.499
Testosterone Treatment (T/P)	1.67	0.86 – 3.27	.132	1.69	0.86 – 3.32	.125	1.39	0.67 – 2.87	.375	1.45	0.71 – 2.96	.308
Basal Cortisol	1.22	0.86 – 1.73	.273	1.15	0.71 – 1.88	.567	1.20	0.85 – 1.71	.300	1.24	0.74 – 2.10	.412
Opponent Gender	2.09	1.56 – 2.79	**<.001**	2.07	1.54 – 2.78	**<.001**	1.78	1.21 – 2.63	**.003**	1.75	1.22 – 2.52	**.003**
Observed	1.16	0.90 – 1.49	.264	1.16	0.90 – 1.50	.258	1.16	0.90 – 1.49	.267	1.15	0.89 – 1.49	.281
Blinding	1.07	0.55 – 2.07	.841	1.07	0.55 – 2.06	.848	1.07	0.55 – 2.08	.833	1.06	0.55 – 2.04	.872
T/P × Cortisol				1.13	0.54 – 2.34	.753				0.70	0.34 – 1.42	.318
T/P × Gender							1.40	0.81 – 2.42	.227	1.36	0.81 – 2.28	.252
Cortisol × Gender										0.88	0.61 – 1.28	.506
T/P × Cortisol × Gender										2.54	1.47 – 4.37	**<.001**
σ^2	3.29			3.29			3.29			3.29		
Observations	1840			1840			1840			1840		
Marginal R^2 / Conditional R^2	0.041 / 0.487			0.042 / 0.488			0.041 / 0.487			0.059 / 0.490		

Table S3: Time of day, time since awakening, and math skill as covariates in initial phase decisions to enter competitions

	Decisions to Compete: Time Since Awakening			Decisions to Compete: Time of Day			Decisions to Compete: Math Skill			Decisions to Compete: All Covariates		
Predictors	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*
(Intercept)	1.24	0.64 – 2.40	0.517	1.27	0.66 – 2.45	0.478	0.04	0.01 – 0.19	**<0.001**	0.03	0.01 – 0.16	**<0.001**
Testosterone Treatment (T/P)	1.47	0.71 – 3.05	0.301	1.42	0.68 – 2.94	0.347	1.42	0.73 – 2.79	0.306	1.35	0.68 – 2.69	0.391
Basal Cortisol	1.23	0.72 – 2.11	0.457	1.25	0.74 – 2.11	0.403	1.24	0.76 – 2.03	0.390	1.32	0.80 – 2.19	0.277
Opponent Gender	1.75	1.21 – 2.52	**0.003**	1.75	1.22 – 2.52	**0.003**	1.74	1.21 – 2.50	**0.003**	1.75	1.21 – 2.51	**0.003**
Observed	1.15	0.89 – 1.49	0.291	1.15	0.89 – 1.49	0.288	1.19	0.93 – 1.54	0.168	1.21	0.94 – 1.56	0.139
Blinding	1.06	0.55 – 2.06	0.858	1.05	0.54 – 2.03	0.892	1.24	0.68 – 2.28	0.485	1.22	0.67 – 2.25	0.515
T/P × Cortisol	0.69	0.34 – 1.41	0.310	0.70	0.34 – 1.44	0.338	0.66	0.34 – 1.30	0.230	0.68	0.34 – 1.33	0.255
T/P × Opponent Gender	1.35	0.80 – 2.28	0.254	1.36	0.81 – 2.28	0.249	1.33	0.79 – 2.24	0.282	1.34	0.80 – 2.26	0.270
Cortisol × Opponent Gender	0.88	0.61 – 1.28	0.505	0.88	0.61 – 1.28	0.505	0.89	0.61 – 1.29	0.539	0.89	0.61 – 1.29	0.545
T/P × Cortisol × Opponent Gender	2.54	1.47 – 4.37	**0.001**	2.54	1.47 – 4.38	**0.001**	2.51	1.46 – 4.33	**0.001**	2.51	1.46 – 4.33	**0.001**
Time since awakening	0.96	0.66 – 1.41	0.840							1.22	0.83 – 1.78	0.307
Time of Day				1.05	0.74 – 1.48	0.786				0.94	0.67 – 1.32	0.728
Math Skill							3.06	1.92 – 4.88	**<0.001**	3.26	2.02 – 5.28	**<0.001**
σ^2	3.29			3.29			3.29			3.29		
Observations	1840			1840			1840			1840		
Marginal R^2 / Conditional R^2	0.058 / 0.490			0.059 / 0.490			0.144 / 0.493			0.151 / 0.496		

Table S4: Effects of testosterone treatment, basal cortisol, opponent gender, and prior outcome on decisions to re-enter competitions after feedback

	Decisions to Compete Again: Main Effects			Decisions to Compete Again: T x C			Decisions to Compete Again: T x Gender			Decisions to Compete Again: T x W/L		
	OR	*CI*	*p*	*OR*	*CI*	*p*	*OR*	*CI*	*p*	*OR*	*CI*	*p*
(Intercept)	0.50	0.25 – 0.98	**0.044**	0.50	0.25 – 0.99	**0.045**	0.45	0.23 – 0.91	**0.025**	0.45	0.21 – 0.94	**0.035**
Testosterone Treatment (T/P)	1.04	0.54 – 2.02	0.906	1.04	0.54 – 2.02	0.905	1.26	0.62 – 2.58	0.523	1.27	0.53 – 3.02	0.596
Basal Cortisol	1.25	0.89 – 1.77	0.203	1.30	0.80 – 2.11	0.298	1.25	0.89 – 1.77	0.202	1.25	0.88 – 1.77	0.207
Opponent Gender	1.90	1.45 – 2.49	**<0.001**	1.90	1.45 – 2.49	**<0.001**	2.31	1.57 – 3.40	**<0.001**	1.90	1.45 – 2.49	**<0.001**
Observed	1.10	0.85 – 1.44	0.462	1.10	0.85 – 1.44	0.463	1.10	0.85 – 1.44	0.468	1.10	0.85 – 1.44	0.461
Prior Outcome (W/L)	4.48	1.91 – 10.52	**0.001**	4.49	1.91 – 10.55	**0.001**	4.47	1.90 – 10.50	**0.001**	6.03	1.79 – 20.31	**0.004**
Blinding	0.90	0.46 – 1.74	0.747	0.90	0.46 – 1.75	0.758	0.89	0.46 – 1.73	0.735	0.90	0.47 – 1.75	0.763
T/P × Cortisol				0.93	0.46 – 1.88	0.840						
T/P × Opponent Gender							0.68	0.40 – 1.16	0.156			
T/P × W/L										0.56	0.10 – 3.00	0.498
Cortisol × W/L												
σ^2		3.29			3.29			3.29			3.29	
Observations		1808			1808			1808			1808	
Marginal R^2 / Conditional R^2		0.073 / 0.679			0.073 / 0.679			0.074 / 0.680			0.075 / 0.679	

Table S4 (continued)

	Decisions to Compete Again: T x C x WL			Decisions to Compete Again: T x C x Gender			Decisions to Compete Again: T x C x Gender x WL		
	OR	*CI*	*p*	*OR*	*CI*	*p*	*OR*	*CI*	*p*
(Intercept)	0.46	0.22 – 0.95	**0.037**	0.46	0.23 – 0.91	**0.026**	0.42	0.20 – 0.89	**0.024**
Testosterone Treatment (T/P)	1.33	0.57 – 3.14	0.509	1.26	0.62 – 2.57	0.529	1.52	0.61 – 3.79	0.368
Basal Cortisol	1.56	0.84 – 2.91	0.162	1.31	0.77 – 2.22	0.314	1.57	0.81 – 3.04	0.186
Opponent Gender	1.91	1.45 – 2.50	**<0.001**	2.31	1.56 – 3.41	**<0.001**	2.33	1.46 – 3.71	**<0.001**
Observed	1.10	0.84 – 1.44	0.478	1.10	0.85 – 1.44	0.469	1.11	0.85 – 1.44	0.462
Prior Outcome (W/L)	5.35	1.63 – 17.51	**0.006**	4.47	1.90 – 10.52	**0.001**	5.42	1.53 – 19.18	**0.009**
Blinding	0.93	0.48 – 1.81	0.836	0.90	0.46 – 1.74	0.746	0.92	0.47 – 1.80	0.814
T/P × Cortisol	0.45	0.19 – 1.07	0.071	0.93	0.44 – 1.98	0.860	0.46	0.18 – 1.16	0.101
T/P × Opponent Gender				0.68	0.40 – 1.18	0.168	0.76	0.39 – 1.47	0.412
T/P × W/L	0.52	0.10 – 2.65	0.429				0.60	0.10 – 3.47	0.572
Cortisol × W/L	0.56	0.17 – 1.90	0.355				0.58	0.16 – 2.12	0.407
T/P × Cortisol × W/L	9.55	1.75 – 52.20	**0.009**				9.63	1.53 – 60.53	**0.016**
Cortisol × Opponent Gender				0.98	0.66 – 1.47	0.934	1.00	0.62 – 1.60	0.992
T/P × Cortisol × Opponent Gender				0.98	0.56 – 1.72	0.952	0.95	0.49 – 1.83	0.872
Opponent Gender × W/L							0.94	0.40 – 2.22	0.896

	Decisions to Compete Again: T x C x WL			Decisions to Compete Again: T x C x Gender			Decisions to Compete Again: T x C x Gender x WL		
	OR	*CI*	*p*	*OR*	*CI*	*p*	*OR*	*CI*	*p*
T/P × Opponent Gender × W/L							0.77	0.24 – 2.52	0.667
Cortisol × Opponent Gender × W/L							0.95	0.39 – 2.35	0.920
T/P × Cortisol × Opponent Gender × W/L							0.96	0.27 – 3.47	0.948
σ^2		3.29			3.29			3.29	
Observations		1808			1808			1808	
Marginal R^2		0.114 / 0.682			0.074 / 0.680			0.114 / 0.683	

Table S5: Time of day, time since awakening, and math skill as covariates of decisions to re-enter competitions after feedback

	Decisions to Compete Again: Time Since Awakening			Decisions to Compete Again: Time of Day			Decisions to Compete Again: Math Skill			Decisions to Compete Again: All Covariates		
	OR	*CI*	*p*	*OR*	*CI*	*p*	*OR*	*CI*	*p*	*OR*	*CI*	*p*
(Intercept)	0.42	0.20 – 0.89	**0.023**	0.43	0.21 – 0.91	**0.027**	0.04	0.01 – 0.18	**<0.001**	0.04	0.01 – 0.18	**<0.001**
Testosterone Treatment (T/P)	1.50	0.63 – 3.59	0.363	1.44	0.60 – 3.43	0.411	1.28	0.57 – 2.88	0.551	1.43	0.62 – 3.28	0.399
Basal Cortisol	1.40	0.74 – 2.65	0.308	1.54	0.82 – 2.87	0.177	1.54	0.85 – 2.77	0.151	1.46	0.79 – 2.67	0.223
Prior Outcome (W/L)	5.36	1.64 – 17.50	**0.005**	5.27	1.61 – 17.27	**0.006**	5.02	1.54 – 16.40	**0.008**	4.94	1.51 – 16.12	**0.008**
Opponent Gender	1.91	1.45 – 2.50	**<0.001**	1.91	1.45 – 2.50	**<0.001**	1.91	1.46 – 2.51	**<0.001**	1.91	1.46 – 2.51	**<0.001**
Observed	1.10	0.84 – 1.44	0.477	1.10	0.84 – 1.44	0.476	1.10	0.84 – 1.43	0.480	1.10	0.84 – 1.44	0.477
Blinding	1.00	0.51 – 1.94	0.991	0.97	0.50 – 1.90	0.937	1.05	0.56 – 1.98	0.868	1.12	0.59 – 2.11	0.724
T/P × Cortisol	0.43	0.18 – 1.02	0.056	0.42	0.18 – 1.01	0.054	0.43	0.19 – 0.98	**0.044**	0.04	0.01 – 0.18	**<0.001**
T/P × W/L	0.51	0.10 – 2.61	0.418	0.53	0.10 – 2.71	0.443	0.49	0.10 – 2.52	0.396	1.43	0.62 – 3.28	0.399
Cortisol × W/L	0.57	0.17 – 1.91	0.360	0.56	0.17 – 1.89	0.352	0.59	0.17 – 1.98	0.391	1.46	0.79 – 2.67	0.223
T/P × Cortisol × W/L	9.53	1.75 – 51.92	**0.009**	9.61	1.75 – 52.69	**0.009**	9.40	1.73 – 51.05	**0.009**	4.94	1.51 – 16.12	**0.008**
Time since awakening	0.76	0.53 – 1.09	0.140							0.04	0.01 – 0.18	**<0.001**
Time of Day				0.83	0.58 – 1.17	0.286				0.84	0.60 – 1.19	0.326
Math Skill							2.31	1.44 – 3.71	**0.001**	2.28	1.41 – 3.68	**0.001**
σ^2	3.29			3.29			3.29			3.29		
Observations	1808			1808			1808			1808		
Marginal R^2 / Conditional R^2	0.119 / 0.681			0.117 / 0.683			0.155 / 0.686			0.158 / 0.686		

Table S6: Other indices of cortisol level as moderators of testosterone treatment and opponent gender on initial phase decisions to enter competitions

	Decisions to compete: Pre to Post Task Cortisol			Decisions to compete: AUC_G		
	Odds Ratio	*CI*	*p*	*Odds Ratio*	*CI*	*p*
(Intercept)	1.24	0.65 – 2.35	.516	1.23	0.64 – 2.34	.536
Testosterone Treatment (T/P)	1.43	0.71 – 2.87	.320	1.44	0.71 – 2.93	.308
Cortisol	0.78	0.46 – 1.32	.353	1.27	0.77 – 2.10	.354
Opponent Gender	1.78	1.20 – 2.63	**.004**	1.77	1.22 – 2.58	**.003**
Observed	1.16	0.89 – 1.49	.271	1.17	0.91 – 1.52	.225
Blinding	1.08	0.56 – 2.10	.816	1.09	0.56 – 2.14	.791
T/P × Cortisol	2.22	1.07 – 4.58	**.031**	0.50	0.24 – 1.02	.056
T/P × Opponent Gender	1.43	0.82 – 2.48	.206	1.40	0.83 – 2.38	.209
Cortisol × Opponent Gender	1.06	0.70 – 1.61	.797	0.99	0.69 – 1.43	.963
T/P × Cortisol × Opponent Gender	0.74	0.41 – 1.33	.317	2.16	1.25 – 3.74	**.006**
σ^2		3.29			3.29	
Observations		1824			1824	
Marginal R^2 / Conditional R^2		0.055 / 0.495			0.053 / 0.496	

Table S7: Other indices of cortisol level as moderators of testosterone treatment and opponent gender on decisions to compete after feedback

	Decisions to Compete Again: Pre to Post Task Cortisol			Decisions to Compete Again: AUCg		
Predictors	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*
(Intercept)	0.44	0.22 – 0.89	**0.023**	0.45	0.21 – 0.93	**0.032**
Testosterone Treatment (T/P)	1.36	0.59 – 3.13	0.469	1.34	0.57 – 3.16	0.498
Cortisol	1.09	0.57 – 2.07	0.799	1.29	0.71 – 2.36	0.400
Prior Outcome (W/L)	5.72	1.76 – 18.62	**0.004**	5.89	1.73 – 20.04	**0.005**
Opponent Gender	1.88	1.45 – 2.43	**<0.001**	1.90	1.45 – 2.50	**<0.001**
Observed	1.09	0.85 – 1.40	0.498	1.09	0.83 – 1.42	0.528
Blinding	0.97	0.51 – 1.84	0.926	0.96	0.49 – 1.88	0.907
T/P × Cortisol	1.81	0.73 – 4.45	0.197	0.49	0.21 – 1.15	0.100
T/P × W/L	0.55	0.11 – 2.82	0.473	0.51	0.10 – 2.74	0.435
Cortisol × W/L	0.89	0.26 – 3.02	0.849	1.00	0.29 – 3.41	0.999
T/P × Cortisol × W/L	0.58	0.10 – 3.26	0.539	3.45	0.63 – 18.94	0.154
σ^2		3.29			3.29	
Observations		1792			1792	
Marginal R^2 / Conditional R^2		0.079 / 0.683			0.087 / 0.681	

Table S8: Effects of testosterone treatment, basal cortisol, and opponent gender on competition metrics

	Score			**Score**			**Satisfaction Rating**			**Satisfaction Rating**		
	Estimates	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	2.50	2.18 – 2.82	**<0.001**	2.59	2.23 – 2.95	<0.001	3.14	2.94 – 3.34	**<0.001**	3.18	2.97 – 3.40	**<0.001**
Testosterone Treatment (T/P)	0.16	-0.20 – 0.52	0.377	0.02	-0.42 – 0.46	0.922	-0.00	-0.22 – 0.22	0.984	0.06	-0.19 – 0.31	0.634
Basal Cortisol	-0.12	-0.38 – 0.15	0.382	-0.05	-0.37 – 0.27	0.776	-0.10	-0.26 – 0.06	0.227	-0.18	-0.36 – 0.00	0.056
Opponent Gender	0.08	-0.12 – 0.27	0.455	0.24	-0.08 – 0.56	0.140	0.12	0.04 – 0.20	**0.005**	0.07	-0.06 – 0.20	0.306
Observed	-0.01	-0.14 – 0.12	0.886	-0.00	-0.14 – 0.13	0.968	-0.04	-0.11 – 0.03	0.278	-0.04	-0.11 – 0.03	0.296
Blinding	-0.08	-0.40 – 0.24	0.631	-0.09	-0.41 – 0.24	0.597	-0.08	-0.29 – 0.13	0.472	-0.08	-0.29 – 0.13	0.467
T/P × Cortisol	0.05	-0.31 – 0.42	0.766	0.00	-0.43 – 0.44	0.990	0.01	-0.22 – 0.23	0.936	0.02	-0.24 – 0.27	0.906
T/P × Gender	-0.13	-0.41 – 0.15	0.358	-0.37	-0.85 – 0.10	0.124	-0.01	-0.12 – 0.11	0.915	-0.06	-0.25 – 0.14	0.575
Cortisol × Gender	-0.01	-0.21 – 0.19	0.931	0.12	-0.22 – 0.45	0.502	0.05	-0.03 – 0.13	0.224	0.16	0.02 – 0.29	**0.025**
T/P × Cortisol × Gender	0.20	-0.08 – 0.48	0.152	0.07	-0.45 – 0.58	0.800	0.06	-0.05 – 0.18	0.286	-0.03	-0.24 – 0.17	0.753
Choice (Compete vs. Piece rate)				-0.15	-0.45 – 0.15	0.329				-0.09	-0.22 – 0.04	0.164
T/P × Choice				0.24	-0.19 – 0.66	0.273				-0.09	-0.26 – 0.09	0.327
Cortisol × Choice				-0.11	-0.43 – 0.21	0.491				0.15	0.02 – 0.28	**0.028**
Choice × Gender				-0.23	-0.64 – 0.18	0.268				0.10	-0.07 – 0.26	0.247
T/P × Cortisol × Choice				0.08	-0.34 – 0.51	0.698				-0.03	-0.21 – 0.15	0.765
T/P × Gender × Choice				0.33	-0.26 – 0.91	0.275				0.06	-0.18 – 0.29	0.649
Cortisol × Choice × Gender				-0.16	-0.58 – 0.26	0.451				-0.19	-0.36 – -0.01	**0.035**

	Score			Score			Satisfaction Rating			Satisfaction Rating		
	Estimates	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
T/P × Cortisol × Gender × Choice				0.16	-0.46 – 0.78	0.604				0.15	-0.11 – 0.40	0.255
σ^2	2.00			2.00			0.33			0.33		
Observations	1840			1840			1838			1838		
Marginal R^2 / Conditional R^2	0.006 / 0.254			0.011 / 0.263			0.016 / 0.509			0.024 / 0.522		

Table S8 (con't)

	Likelihood of Winning			Likelihood of Winning		
	Odds Ratios	*CI*	*p*	*Odds Ratios*	*CI*	*p*
(Intercept)	1.46	1.05 – 2.04	**0.026**	1.47	0.97 – 2.21	0.067
Testosterone Treatment (T/P)	1.16	0.79 – 1.68	0.453	1.12	0.67 – 1.88	0.669
Basal Cortisol	0.89	0.67 – 1.17	0.391	0.91	0.62 – 1.32	0.619
Opponent Gender	1.66	1.22 – 2.24	**0.001**	1.44	0.90 – 2.31	0.128
Observed	1.02	0.84 – 1.25	0.837	1.02	0.84 – 1.25	0.839
Blinding	0.81	0.59 – 1.12	0.211	0.82	0.59 – 1.12	0.212
T/P × Cortisol	1.03	0.70 – 1.50	0.887	0.86	0.51 – 1.44	0.561
T/P × Gender	0.94	0.62 – 1.44	0.789	0.86	0.43 – 1.74	0.684
Cortisol × Gender	0.94	0.70 – 1.28	0.705	0.96	0.58 – 1.57	0.863
T/P × Cortisol × Gender	1.40	0.91 – 2.15	0.131	1.48	0.68 – 3.19	0.323
Choice (Compete vs. Piece rate)				1.00	0.64 – 1.54	0.983
T/P × Choice				1.06	0.58 – 1.96	0.849

	Likelihood of Winning			Likelihood of Winning		
	Odds Ratios	*CI*	*p*	*Odds Ratios*	*CI*	*p*
Cortisol × Choice				0.96	0.61 – 1.51	0.849
Choice × Gender				1.23	0.67 – 2.26	0.505
T/P × Cortisol × Choice				1.39	0.75 – 2.60	0.300
T/P × Gender × Choice				1.07	0.44 – 2.57	0.882
Cortisol × Choice × Gender				0.98	0.52 – 1.84	0.951
T/P × Cortisol × Gender × Choice				0.82	0.32 – 2.12	0.680
σ^2	3.29			3.29		
Observations	1840			1840		
Marginal R^2 / Conditional R^2	0.025 / 0.151			0.028 / 0.146		

Table S9: Secondary analyses of trait dominance as moderator of testosterone's effects on competitive behavior

Table S9.A: Initial phase

	Decisions to Compete: T/P × Dominance			Decisions to Compete: T/P × Dominance × Opponent Gender			Decisions to Compete: T/P × Dominance × Basal Cortisol		
Predictors	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*
(Intercept)	1.16	0.61 – 2.20	0.651	1.27	0.66 – 2.45	0.479	1.13	0.60 – 2.14	0.701
Testosterone Treatment (T/P)	1.61	0.83 – 3.12	0.162	1.34	0.65 – 2.74	0.429	1.66	0.85 – 3.21	0.136
Trait Dominance	1.44	0.90 – 2.31	0.126	1.52	0.91 – 2.54	0.113	1.38	0.86 – 2.22	0.180
Basal Cortisol	1.23	0.87 – 1.73	0.247	1.22	0.86 – 1.73	0.259	1.20	0.74 – 1.96	0.450
Opponent Gender	2.10	1.57 – 2.80	**<0.001**	1.78	1.21 – 2.62	**0.003**	2.08	1.55 – 2.79	**<0.001**
Observed	1.12	0.86 – 1.45	0.394	1.12	0.86 – 1.45	0.397	1.14	0.88 – 1.47	0.329
Blinding	1.09	0.57 – 2.09	0.799	1.09	0.57 – 2.10	0.789	1.07	0.55 – 2.06	0.845
T/P × Dominance	0.75	0.40 – 1.42	0.375	0.70	0.35 – 1.41	0.321	0.72	0.38 – 1.37	0.316
T/P × Opponent Gender				1.41	0.81 – 2.43	0.222			
Dominance × Opponent Gender				0.92	0.62 – 1.35	0.658			
T/P × Dominance × Opponent Gender				1.12	0.66 – 1.91	0.674			
T/P × Cortisol							0.98	0.48 – 2.04	0.967
Dominance × Cortisol							0.87	0.52 – 1.45	0.585
T/P × Dominance × Cortisol							1.46	0.76 – 2.81	0.252
σ^2	3.29			3.29			3.29		
Observations	1840			1840			1840		
Marginal R^2 / Conditional R^2	0.050 / 0.491			0.051 / 0.492			0.057 / 0.488		

Table S9.B: Feedback phase

	Decisions to Compete Again: T/P × Dominance			Decisions to Compete Again: T/P × Dominance × Opponent Gender			Decisions to Compete Again: T/P × Dominance × Basal Cortisol		
Predictors	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*
(Intercept)	0.49	0.25 – 0.98	**0.043**	0.46	0.22 – 0.97	**0.042**	0.50	0.25 – 0.99	**0.047**
Testosterone Treatment (T/P)	1.05	0.54 – 2.04	0.876	1.23	0.50 – 2.98	0.653	1.06	0.54 – 2.04	0.874
Trait Dominance	1.35	0.84 – 2.16	0.211	0.85	0.46 – 1.58	0.602	1.34	0.84 – 2.16	0.219
Basal Cortisol	1.32	0.93 – 1.86	0.118	1.31	0.93 – 1.85	0.128	1.32	0.81 – 2.14	0.268
Prior Outcome (W/L)	4.58	1.95 – 10.77	**<0.001**	5.57	1.68 – 18.47	**0.005**	4.66	1.97 – 11.03	**<0.001**
Opponent Gender	1.95	1.47 – 2.59	**<0.001**	1.93	1.46 – 2.55	**<0.001**	1.95	1.47 – 2.59	**<0.001**
Observed	1.11	0.84 – 1.47	0.461	1.11	0.84 – 1.47	0.449	1.11	0.84 – 1.47	0.458
Blinding	0.85	0.44 – 1.64	0.623	0.85	0.44 – 1.65	0.631	0.82	0.42 – 1.61	0.565
T/P × Dominance	0.82	0.43 – 1.57	0.548	1.28	0.54 – 3.04	0.572	0.89	0.46 – 1.72	0.722
T/P × W/L				0.65	0.12 – 3.47	0.615			
Dominance × W/L				3.79	1.12 – 12.78	**0.032**			
T/P × Dominance × W/L				0.28	0.05 – 1.45	0.129			
T/P × Cortisol							1.09	0.53 – 2.22	0.822
Dominance × Cortisol							0.94	0.57 – 1.54	0.792
T/P × Dominance × Cortisol							0.85	0.44 – 1.65	0.640
σ^2	3.29			3.29			3.29		
Observations	1808			1808			1808		
Marginal R^2 / Conditional R^2	0.081 / 0.686			0.102 / 0.687			0.084 / 0.688		

Table S10: Perceptions of opponent status in competition

Table S10.A: Ratings of extent to which opponent was "good at simple math tasks" from participants and follow-up raters

	Participants			**Participants**			**Follow-up Raters**			**Participants & Follow-ups**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	4.87	4.66 – 5.07	**<0.001**	4.98	4.78 – 5.19	**<0.001**	4.62	4.28 – 4.96	**<0.001**	4.74	4.51 – 4.97	**<0.001**
Opponent Gender (Female = 0)	-0.41	-0.68 – -0.15	**0.021**	-0.36	-0.63 – -0.10	**0.039**	-0.62	-1.09 – -0.16	**0.038**	-0.52	-0.81 – -0.23	**0.008**
Prior Outcome (W/L)				-0.29	-0.39 – -0.20	**<0.001**						
Sample: Participant vs. Follow-ups[1]										-0.25	-0.54 – 0.04	0.157
Opponent Gender x Sample										-0.21	-0.49 – 0.07	0.225
σ^2	0.71			0.65			0.54			0.69		
ICC	0.30 $_{\text{Participant}}$			0.32 $_{\text{Participant}}$			0.35 $_{\text{Rater}}$			0.30 $_{\text{Rater}}$		
	0.08 $_{\text{Opponent}}$			0.09 $_{\text{Opponent}}$			0.12 $_{\text{Opponent}}$			0.09 $_{\text{Opponent}}$		
Observations	1872			1792			256			2128		
Marginal R^2 / Conditional R^2	0.035 / 0.419			0.053 / 0.420			0.093 / 0.485			0.052 / 0.432		

Notes:

1. Sample source was contrast coded (participant sample = -0.5, follow-up rater sample = 0.5) so that the estimate of the effect of opponent gender would reflect the mean gender difference across the two samples.

Table S10.B: Other ratings of participants' perceptions of opponents in competition task

	Attractive			**Dominant**			**Intelligent**			**Mature**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	3.64	3.21 – 4.08	**<0.001**	3.87	3.54 – 4.19	**<0.001**	4.84	4.59 – 5.09	**<0.001**	4.37	4.13 – 4.61	**<0.001**
Opponent Gender (Female = 0)	0.36	-0.23 – 0.95	0.330	-0.20	-0.65 – 0.25	0.469	-0.30	-0.63 – 0.03	0.160	0.17	-0.16 – 0.49	0.412
σ^2		0.98			1.10			0.85			0.86	
ICC		0.32 $_{\text{Participant}}$			0.24 $_{\text{Participant}}$			0.25 $_{\text{Participant}}$			0.24 $_{\text{Participant}}$	
		0.23 $_{\text{Opponent}}$			0.15 $_{\text{Opponent}}$			0.11 $_{\text{Opponent}}$			0.11 $_{\text{Opponent}}$	
Observations		1872			1871			1872			1871	
Marginal R^2 / Conditional R^2		0.015 / 0.555			0.005 / 0.435			0.016 / 0.399			0.005 / 0.377	

	Warm			**"I Feel Close"**			**Respect**			**"I Performed Better"**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	3.86	3.60 – 4.11	**<0.001**	3.04	2.84 – 3.23	**<0.001**	4.42	4.27 – 4.58	**<0.001**	3.94	3.74 – 4.14	**<0.001**
Opponent Gender (Female = 0)	0.17	-0.18 – 0.52	0.433	0.11	-0.10 – 0.32	0.415	0.02	-0.14 – 0.17	0.868	0.35	0.14 – 0.56	**0.013**
σ^2		0.94			0.80			0.51			1.17	
ICC		0.21 $_{\text{Participant}}$			0.48 $_{\text{Participant}}$			0.52 $_{\text{Participant}}$			0.42 $_{\text{Participant}}$	
		0.12 $_{\text{Opponent}}$			0.03 $_{\text{Opponent}}$			0.02 $_{\text{Opponent}}$			0.02 $_{\text{Opponent}}$	
Observations		1872			1871			1871			1868	
Marginal R^2 / Conditional R^2		0.005 / 0.339			0.002 / 0.520			0.000 / 0.547			0.014 / 0.466	

Table S11. Comparison of simpler and more complex models for testosterone treatment × basal cortisol × prior outcome effect (win/lose)

	Simplified Model (i.e., as reported in text and Table S4)			**More Complex Model (singular fit)**		
Predictors	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*
(Intercept)	0.46	0.22 – 0.95	**0.037**	0.47	0.23 – 0.99	**0.047**
Testosterone Treatment (T/P)	1.33	0.57 – 3.14	0.509	1.28	0.53 – 3.06	0.582
Basal Cortisol	1.56	0.84 – 2.91	0.162	1.57	0.84 – 2.92	0.158
Opponent Gender	5.35	1.63 – 17.51	**0.006**	5.12	1.56 – 16.76	**0.007**
Observed	1.91	1.45 – 2.50	**<0.001**	1.96	1.48 – 2.59	**<0.001**
Prior Outcome (W/L)	1.10	0.84 – 1.44	0.478	1.12	0.85 – 1.48	0.418
Blinding	0.93	0.48 – 1.81	0.836	0.85	0.43 – 1.67	0.639
T/P × Cortisol	0.45	0.19 – 1.07	0.071	0.47	0.20 – 1.13	0.091
T/P × W/L	0.52	0.10 – 2.65	0.429	0.60	0.11 – 3.11	0.539
Cortisol × W/L	0.56	0.17 – 1.90	0.355	0.56	0.17 – 1.87	0.344
T/P × Cortisol × W/L	9.55	1.75 – 52.20	**0.009**	10.21	1.84 – 56.55	**0.008**
σ^2		3.29			3.29	
Observations		1808			1808	
Marginal R^2 / Conditional R^2		0.114 / 0.682			0.279 / NA	

Table S12. Model fit statistics

	AIC	BIC
Initial Phase		
Main effects	2008.9	2075.1
Testosterone treatment (T/P) × Cortisol	2010.8	2082.5
T/P × Gender	2009.5	2081.2
T/P × Cortisol × Gender	1999.2	2087.5
Feedback Phase		
Main effects	1838.9	1893.9
T/P × Cortisol	1840.9	1901.4
T/P × Gender	1838.9	1899.4
T/P × Prior Outcome (W/L)	1840.5	1901
T/P × Cortisol × W/L	1837.8	1914.8
T/P × Cortisol × Gender	1844.8	1921.8
T/P × Cortisol × W/L × Gender	1849.3	1964.8

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