Research article

Exogenous testosterone increases status-seeking motivation in men with unstable low social status

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Abstract

Testosterone is associated with status-seeking behaviors such as competition, which may depend on whether one wins or loses status, but also on the stability of one's status. We examined (1) to what extent testosterone administration affects competition behavior in repeated social contests in men with high or low rank, and (2), whether this relationship is moderated by hierarchy stability, as predicted by the status instability hypothesis. Using a real effort-based design in healthy male participants ($N = 173$ males), we first found that testosterone (vs. placebo) increased motivation to compete for status, but only in individuals with a low unstable status. A second part of the experiment, tailored to directly compare stable with unstable hierarchies, indicated that exogenous testosterone again increased competitive motivation in individuals with a low unstable status, but decreased competition behavior in men with low stable status. Additionally, exogenous testosterone increased motivation in those with a stable high status. Further analysis suggested that these effects were moderated by individuals' trait dominance, and genetic differences assessed by the androgen receptor (CAG-repeat) and dopamine transporter (DAT1) polymorphisms. Our study provides evidence that testosterone specifically boosts status-related motivation when there is an opportunity to improve one's social status. The findings contribute to our understanding of testosterone's causal role in status-seeking motivation in competition behavior, and indicate that testosterone adaptively increases our drive for high status in a context-dependent manner. We discuss potential neurobiological pathways through which testosterone may attain these effects on behavior.

Keywords

testosterone; social status; hierarchy stability; competition; dominance; AR(CAG) repeat polymorphism

1. Introduction

Status hierarchies play a prominent role in our daily lives (e.g., in our social network, at work, in politics). A high social status provides access to limited resources and rewards such as food, mates, and money, which may increase life expectancy (Sapolsky, 2004). A key neurobiological substrate that is hypothesized to play an important role in status-seeking motivation—so-called dominance behavior—is the sex steroid testosterone (Eisenegger et al., 2011). Nevertheless, little is known about testosterone's precise role in status motivation, and how it may change depending on the stability of, and an individual's position (high vs. low) within a given social hierarchy. An outstanding question of interest is whether testosterone causes differential effects on behavior for those lower or higher in the hierarchy and during periods of instability.

Research has shown that testosterone levels fluctuate with social challenges (challenge hypothesis; Wingfield et al., 1990; Archer, 2006; Wobber et al., 2010) and that these fluctuations predict competition-related motivations (e.g., Carré and McCormick, 2008; Eisenegger et al., 2017; Mehta et al., 2008; Mehta and Josephs, 2006), performance (Zilioli and Watson, 2014), and other behaviors (e.g., aggression, Carré et al., 2009; Geniole et al., 2013; see also for recent reviews Geniole and Carré, 2018; Zilioli and Bird, 2017). Nevertheless, the direction and magnitude of effects often depended on the individual's status within the hierarchy, differing for those who recently won or lost a competition (Josephs et al., 2006; Mehta and Josephs, 2006; for review Carré and Olmstead, 2015; Casto and Mehta, 2018; but also see, Carré et al., 2013)¹. Overall, these findings are consistent with the Biosocial Model of Status (Mazur, 1985), suggesting that changes in testosterone following a victory—equivalent to attaining higher status—may motivate status-relevant behavior (e.g., decisions to compete) to defend

¹ Carré and colleagues (2013) observed elevated testosterone to predict subsequent aggression in both male winners and losers.

44 and attain higher status positions, but changes following a defeat may inhibit status-relevant behavior to
45 avoid further status loss and harm (see Eisenegger et al., 2011).

46 While these studies have provided initial evidence that changes in testosterone may modulate
47 competitiveness, pharmacological administration studies examining the causal effects of the hormone
48 on competition have been relatively scarce. In one study (Mehta et al., 2015), willingness to compete
49 after a victory was increased by testosterone administration (vs. placebo) among women high in trait
50 dominance (i.e., those preferring/desiring high status, Josephs et al., 2006), but decreased after defeat
51 (independent of women's trait dominance). Hence, one's status within a hierarchy—based on previous
52 wins and losses—may moderate testosterone's effects on competitive behavior.

53 A still unanswered question and one of the aims of the present study is how exogenous
54 testosterone influences competition behavior depending on one's own status position (i.e., high or low)
55 within a social hierarchy. In addition, the stability of one's status position within a hierarchy may also be
56 important. For example, individuals with low unstable status as well as a high stable status felt
57 challenged, whereas when they had a low stable status or even a high unstable status they felt relatively
58 threatened (Scheepers, 2009). Given the notion that testosterone promotes behaviors aimed at
59 attaining and maintaining high status during social challenge, testosterone may specifically encourage
60 status-seeking behaviors in individuals with low-unstable status who have an opportunity to attain a
61 high(er) status (e.g., after a close loss), while testosterone may increase avoidance of further contests in
62 individuals with high-unstable status to avoid status loss (e.g., after a close win) (as predicted by the
63 status instability hypothesis, Zilioli et al., 2014). One study that manipulated status and stability directly
64 found that hierarchy instability (vs. stability) increased testosterone levels in high status individuals and
65 predicted subsequent performance, dominance and testosterone reactivity (Knight and Mehta, 2017).
66 Nevertheless, no studies have exogenously manipulated testosterone in addition to status and hierarchy
67 stability to examine its effects on behavior. Therefore, an important question and our second major aim

is whether testosterone predicts subsequent status-seeking behavior depending on the stability of, and an individual's position (high vs. low) within, a given social hierarchy. Based on the theoretical models and studies reviewed here, we predicted that testosterone would increase competitive behavior in those that possess an unstable low status compared to a stable low or even unstable high status, whereas in stable hierarchies, we predicted testosterone to motivate status-relevant behavior in those with high status, but not with low status. We further explored whether the expected conditional effects on competitive effort are moderated by participants' trait dominance. Based on prior studies (Carré et al., 2009; Mehta et al., 2015), we hypothesized that the effects of testosterone would be enhanced in individuals high in trait dominance, who are motivated to maintain or attain high social status, but not in individuals low in trait dominance.

To address our aims, we performed a double-blind placebo-controlled administration study in which men received either testosterone or placebo and then participated in a repeated competition against multiple players where their status was manipulated to be either high or low and, in a second part of the competition, stable or unstable. The competition required real effort and choice, and performance was not monetarily incentivized, thus providing a powerful and objective measure of their motivation and willingness to compete for status independent of monetary rewards. The present study is the first to combine testosterone administration with real-effort to assess whether testosterone's effects on competitive behavior depend on status and stability.

To gain insight into the potential mechanisms by which testosterone motivates status-seeking behavior, we also explored the moderation by genetic differences in the androgen receptor (AR) and the striatal dopamine transporter (DAT1). It is hypothesized that testosterone influences behavior by binding to ARs (Simmons and Roney, 2011). The efficiency of the AR is negatively related to the cytosine-adenine-guanine (CAG)-repeat length (Zitzmann and Nieschlag, 2003). Although only a few studies examined genetic polymorphisms in relation to testosterone and behavior, they suggest that individuals

with lower CAG-repeat length and thus higher AR efficiency showed more potential status-enhancing behaviors. For example, a correlational study by Eisenegger and colleagues (2017) found that among individuals with a low CAG-repeat polymorphism and who decided to compete, greater confidence and performance in competition was observed. Furthermore, a recent pharmacological study with a larger sample (Geniole et al., 2019) found testosterone to predict subsequent aggression among men with a low CAG-repeat polymorphism. Based on these studies, we hypothesized that men with fewer AR(CAG) repeats show enhanced effects of testosterone on behavior.

Likewise, it has been suggested that testosterone may influence behavior by interacting with dopamine and activating dopaminergic pathways such as the striatum (Mehta et al., 2015; Purves-Tyson et al., 2014; see also Losecaat Vermeer et al., 2016). In rodent studies, dopamine release and availability in the ventral striatum were increased with exogenous testosterone (de Souza Silva et al., 2009; Packard et al., 1997; Thiblin et al., 1999), and increased expression of ARs in regions of the mesolimbic system were found after winning in competition, associated with reward and status-enhancing behaviors (Fuxjager et al., 2010). In humans, exogenous testosterone increased activation of the ventral striatum to monetary rewards (Hermans et al., 2010). Yet, no human studies have examined the effects of testosterone on status-seeking behavior among individuals with varying striatal dopamine levels. The expression of DAT, which regulates striatal dopamine, is linked to a 40 base-pair variable number tandem repeat (VNTR) polymorphism of the DAT1 gene (Vandenbergh et al., 1992). Homozygous 10/10-repeat carriers of this polymorphism have higher DAT expression (i.e., lower striatal dopamine) than heterozygous individuals (i.e., 9-repeat variants, Heinz et al., 2000). We tested whether testosterone effects on competitive behavior depended on individual differences in dopamine, assessed by DAT1 polymorphism.

2. Methods and Materials

2.1. Participants

One hundred-seventy-three volunteers participated in the study. Participants were pre-screened and had no history of psychiatric, neurological, or endocrine disease, nor currently using or regular users of marijuana or any psychotropic drugs and corticosteroids, and had not taken part in a medical study in the 4 weeks preceding the study. The sample included only males, since testosterone gel administration has only been established in men (Eisenegger et al., 2013). We excluded three participants because their data was not saved due to technical problems, one participant who participated twice, and one other participant for failure to provide responses on a large number of trials (average 24%). The final sample included 168 men (Placebo ($n = 84$): mean age = 24.5, $SD = 3.51$; Testosterone ($n = 84$): mean age = 24.5, $SD = 3.36$). All participants provided written informed consent, were financially compensated for completion of the study, and received an additional 4 Euro for the separate incentivized trials to assess participants' maximum performance in the task. The study procedure was performed in accordance with the Declaration of Helsinki and approved by the Medical Ethics Committee of the University of Vienna (1918/2015).

2.2. General procedure

Participants arrived to the lab on two separate days. On the first day participants completed a batch of personality questionnaires including demographics, provided DNA for genotyping, and were measured for their Body Mass Index (BMI) and body fat using a body composition monitor (Omron BF51). On the second day, at 10:00 a.m., participants arrived to the lab in groups of nine. After participants provided consent and received instructions of the procedure of the day, they completed questionnaires assessing self-reported mood (multidimensional mood questionnaire, Steyer et al., 1994) and personality traits such as trait dominance (16-PF-R, Schneewind and Graf, 1998), which has previously been correlated with status-seeking and testosterone (Mehta et al., 2015, 2008). Twenty minutes after arrival

participants provided the first saliva sample (t_0), to assess baseline hormone concentrations. Next, participants were administered with 150 mg testosterone or placebo by random assignment (section 2.2.1). Participants waited for two hours to allow the drug to reach peak level while they stayed in the lab, were provided with food and water, and did not interact with others. To control for a potential confound of mood from the treatment (Amanatkar, Chibnall, Seo, Manepalli, & Grossberg, 2014), two-hours post-administration participants completed a second mood questionnaire (see SI). Next, and prior to the behavioral testing, participants provided a second saliva sample (t_{120}). To minimize diurnal hormone variability, the behavioral testing occurred from 1:00-3:00 p.m., and included the competition paradigm with a brief post-task questionnaire, followed by two additional tasks unrelated to the current study and therefore not reported here. At the end, we assessed participants' beliefs and confidence about the hormone that has previously been shown to affect participants' behavior (Eisenegger et al., 2010), and participants were paid for their participation. For summary statistics see Table 1.

2.2.1. Drug administration

We randomly assigned participants to receive a single dose of 150 mg testosterone (Testogel®, $n = 84$) or placebo gel ($n = 84$) in a double-blind procedure. The gel was applied transdermal to chest, shoulders and upper arms by a male research assistant. The pharmacokinetics of this dosage and administration technique have previously been established in men (Eisenegger et al., 2013), and had shown testosterone levels to peak 3 hours after administration with a significant increase after 2 hours. Therefore, our behavioral test session of the present experiment commenced 2h after drug administration. Note though that more recent work using larger sample sizes with the same dosage observed a significant increase in testosterone levels as fast as 1-hour post-administration (Bird et al., 2016; Carré et al., 2015). This notwithstanding, evidence of these studies suggest that testosterone levels were still sufficiently high in our testing window.

2.2.2. Task procedure

We developed a novel, real effort paradigm to assess status-seeking motivation in competition. This paradigm consisted of a repeated multi-player competition where participants had to exert effort to compete for rank position by responding as fast as possible to multiple targets on screen (see 2.2.3). The advantage of this task design is that it requires attention and physical effort from the participant, but no specific skill, allowing us to use their performance as an indirect, objective and continuous measure of motivation that is comparable across participants. The task was always played in groups of 9 participants simultaneously who were seated in small cubicles in the same room to increase a realistic setting and to reduce possible suspicion of participants about the rigged nature of the competition, which we probed with a questionnaire item at the end of the experiment (see SI).

Before the competition task commenced, participants completed five practice trials of the real-effort paradigm. After this, we monetarily incentivized them for one trial to provide maximum effort, as expressed by the median response time on this incentivized trial. Individuals' maximum effort was also used to assess a minimum performance threshold for in the competition (section 2.2.3). Then, to examine testosterone's effects on performance independent of status, stability, and external incentives, participants completed 15 trials of the task without monetary incentive and before they were aware of the impending competition (see SI).

Next, participants entered the competition phase of the study, which consisted of two parts. In the first part, participants played two blocks of 15 trials each in which we manipulated the rank, between-subjects, to be high or low, based on random assignment. Then, to determine whether the effects of rank were moderated by rank stability, participants completed the second part of the competition phase (one block of 10 trials) in which participants' already established rank (high or low) was made to appear stable or unstable (Fig. 1A). At the end of the second part of the competition phase,

participants were asked whether they wanted to compete one final time, in an attempt to change their rank, or if they preferred to perform the same task alone, without competition.

2.2.3. Real-effort based competition paradigm

The real-effort based competition paradigm entailed a reaction time task, in which participants had to respond as fast as possible by pressing a key when a white square briefly appeared on screen for 250 ms at random intervals. The faster participants responded at the onset of the square, the more points they could earn. Points ranged from 1-10 points with steps of 1 (henceforth, low reward) or between 10-100 points with steps of 10 (henceforth, high reward) depending on the reward level that was presented to participants at the beginning of each trial. Points could only be earned for every first response per square, while multiple keypresses to the same square were penalized with a fixed amount (i.e., per extra keypress 30% of the maximum points per reward level). This was done to ensure validity of the task and avoid the opportunity for participants to press the key as often as possible to earn maximum points by chance. Participants received feedback on screen for every response. The whole procedure was repeated ten times per trial, containing five consecutive high and five consecutive low reward squares, counterbalanced across participants (Fig. 1B). At the end of every trial, their total points were shown, and participants were asked to guess their rank position from one to nine reflecting best to worst player in the group, after which participants received feedback about their rank position presented by their name in a rank list (Fig.1B). Unbeknownst to the participants, their rank position was controlled to be either high (starting second, varying between 1 to 4) or low (starting eighth, varying between 6 to 9). To ensure instability within the higher and lower echelons of the hierarchy, participants' rank position varied across trials, with no more than two consecutive trials in the same position. Although participant's rank was controlled to be either high or low, their rank position was otherwise contingent upon their performance. To maintain high believability in the task, a minimum performance threshold

was set at 60% of participants' maximum performance – if participants response time was slower, they were dropped to position 9 on that trial (i.e., if maxRT is 300 ms, then current RT had to be < 500 ms to avoid being dropped). This occurred once for seven participants, and twice for two other participants. These trials were excluded from analysis.

Performance in competition increases when individuals can compare their behavior to others, even if their performance is kept private (Kuhnen and Tymula, 2008). To explore if testosterone influences effort depended on whether rank is public or private, in the first part of the competition phase we provided rank feedback either publicly or privately per block of 15 trials, with the order counterbalanced across blocks and participants (Fig. 1). Private feedback only included participants own rank information which was not shared with the group, whereas with public feedback, rank positions of all opponents including the participant were visible for the group. Moreover, for the public feedback condition, we told participants that they would be called forward in order of their final rank at the end of the experiment. Only prior to the start of each public or private feedback block, participants received instructions about the specific feedback condition. At the end of each block participants saw their final rank, which was always second or eighth for high and low rank condition, respectively.

<< Insert Figure 1 about here >>

The second part of the competition consisted of another public block of 10 trials, which unbeknownst to the participants was manipulated to be stable or—as in the first part of the competition—unstable. Participants first saw their final rank of the public block from the first part of the competition again, this time with the scores of their opponents, including their own score, which was the mean score of all public trials. Here, we manipulated the closeness of the scores to make rank appear stable or unstable. Participants randomly assigned to an unstable rank saw scores that were

close, whereas participants assigned to a stable rank saw scores that were more distant to theirs. To create a stable, established hierarchy for the high and low stable rank groups, their rank position only varied across the first three trials and thereafter became fixed to position one and nine, respectively. Conversely, rank position for the unstable rank groups varied after each trial within the high (1-4) or low (6-9) rank category.

2.2.4. Initial belief in status and expectations during the task

Before the competition commenced, participants estimated their rank position between 1 and 9, as an initial status belief. Testosterone (vs. placebo) did not affect participants' initial status belief ($p = .921$, Table 1). During the first part of the competition phase, participants provided their rank expectation before receiving rank feedback on every trial. As shown in Table 1, testosterone (vs. placebo) did not influence participants' rank expectations in those with a high rank, however, lowered participants' rank expectations in those with a low rank. Therefore, we included participants' trial-by-trial expectations in our statistical model.

< Insert Table 1 here >

2.2.5. Hormonal assays

Hormone samples were collected via passive drool and stored at -30 degrees Celsius. Saliva samples were analyzed for testosterone and hydrocortisone with liquid chromatography tandem mass spectrometry (LCMS/MS) (see supplement information (SI)). As shown in Table 1, baseline hormone concentrations did not differ between treatment groups (t_0), with a significant difference in testosterone concentration between treatment groups after administration (t_{120}), confirming our manipulation.

2.2.6. Genotyping

DNA was collected using sterile cotton buccal swabs (Sarstedt AG, Germany), and was extracted applying the QIAamp DNA Mini kit (Qiagen, Germany). AR(CAG)-repeat and DAT1 VNTR polymorphisms were investigated by PCR with fluorescent-labeled primers and capillary electrophoresis (details of procedure in SI). Treatment groups (Testosterone $M = 22.18$, $SD = 3.11$, Placebo $M = 22.33$, $SD = 2.83$) did not differ in mean AR(CAG) repeat length ($z = 0.34$, $p = .736$). The 9/10R and the 10/10R genotypes accounted for most of the observed DAT1 genotypes in our sample (33% ($n=56$) and 57% ($n=96$), respectively, see SI), and we thus used these two genotypes in the analyses.

2.3. Statistical analysis

Effort to compete (reaction times)

To assess motivation for status, we used effort as the dependent variable, with fast reaction times representing higher effort and slow or no responding representing lower effort. Response times < 100 ms were removed, using an *a priori* criterion based on a threshold previously suggested to yield accurate response times (Whelan, 2008). Given that reaction times can be noisy, and the median is less influenced by a few single extreme values, the outliers in response time were determined on three median absolute deviations (MAD) above the median for each participant, as advised by others (Leys et al., 2013), and then winzorised to this score. Misses were replaced to this score, as by our design participants could also decide not to respond, which would represent low motivation. However, misses following a late incorrect response for a previous square were excluded, as these misses could represent an early guess for the next square.

Behavioral analyses were performed using the R statistical software package (version 3.4.3, R Development Core Team, 2017). To control for the variance of random effects without requiring to aggregate the data (Baayen et al., 2008), we used linear mixed effects-models, including fixed and

random effects, using *lme4* (Bates et al., 2015). F-tests and p-values for regression coefficients were calculated based on Satterthwaite's approximations using *lmerTest* (Kuznetsova et al., 2017). Planned and post-hoc analysis, reported means and standard errors were computed with the *emmeans* package and slope comparisons were performed with *emtrends* (Lenth, 2016). Plots were created using *ggplot2* (Wickham, 2016).

To test if testosterone treatment increases competitive effort depending on rank position in the first part of the competition, our main model included between-subject predictors (at level two) for treatment (testosterone, placebo) and rank (high, low), and within-subject predictors (at level one) for reward level (high, low), feedback type (public, private), rank expectations, and a covariate for time, with the final model including interactions between treatment, rank and time. We also included a random intercept for participant including by-participant random slopes for reward level and feedback type to account for repeated observations and variability among participants. Although a preliminary analysis including all three-way interactions showed that the public-private rank feedback manipulation worked ($p = .003$), testosterone effects were not depended on this (see Table S3 in SI). Therefore, we reduced the model complexity by including feedback as a fixed effect only, which was confirmed by an improved model fit (M1, see SI Table S3 for model comparison and equation)².

To address our second aim if effects of testosterone on competitive effort depend on hierarchy stability—manipulated in the second part of the competition phase—we ran a similar mixed model (M2) including a between-subject predictor for rank stability (stable, unstable), and a four-way interaction (treatment $\times$ rank $\times$ time $\times$ stability) to predict effort.

² Two participants were excluded in M1, as they did not provide rank expectations in the task (testosterone, $n=83$; placebo, $n=83$).

Decision to compete

We analyzed the binary choice data with a generalized linear model (GLM1), including between-subject fixed factors for treatment, rank feedback and rank stability, and their interaction.

Covariates

Trait dominance and polymorphisms were included as covariates and analyzed separately for each of the models above. The DAT1-polymorphism was only included for M1, due to an insufficient number of subjects per cell for each genotype (i.e., < 10 participants per cell).

3. Results

Part 1: Exogenous testosterone enhances competitive effort in individuals with low rank

We found a significant three-way interaction between treatment group, rank and time ($F_{(1, 1762)} = 6.84, p = .009$), such that participants with exogenous testosterone (vs. placebo) exerted greater competitive effort (responded faster) as the task progressed, but only for individuals with a low rank position (Fig.2, Table 2). Furthermore, the effect of testosterone on competitive effort in low ranked men was enhanced in those who had higher trait dominance (treatment $\times$ time $\times$ dominance trait: $F_{(1, 707.1)} = 12.23, p < .001$, Table 2, Fig. S2), fewer AR(CAG) repeats ($F_{(1, 706.3)} = 11.33, p < .001$; Fig. S3), and a 9/10R DAT1 genotype (i.e., higher striatal dopamine, $z = 4.10, p < .001$, see SI). Low ranked men with 9/10R were also faster than high ranked men with 9/10R ($z = 3.93, p < .001$; Fig. S4). Although these conditional effects of testosterone were enhanced among men with high dominance, fewer AR(CAG) repeats and a 9/10R DAT, our main three-way interaction of treatment by rank across time remained significant in all models, suggesting that the interaction is robust.

< Insert Figure 2 and Table 2 here >

Part 2: Effects of testosterone on competitive effort depend on hierarchy stability

In the second part of the competition, our manipulation of stability further moderated the three-way interaction (i.e., treatment $\times$ rank $\times$ trial $\times$ stability interaction, $F_{(1,16215.4)} = 8.38, p = .004$). Planned contrasts showed that the tendency of testosterone to increase competitive effort across time in low-ranked men was only evident when the hierarchy was unstable, compared with low-ranked men administered with placebo (Fig.3). Again, this conditional effect of testosterone was enhanced among men with fewer AR(CAG) repeats ($F_{(1,7820.7)} = 6.41, p = .011$; Fig. S5). In fact, when the hierarchy was stable, testosterone instead decreased effort across time in low-ranked men (Fig. 3, Table 3). Further, in high ranked men testosterone (vs. placebo) increased effort overall (treatment $\times$ rank $\times$ stability, $F_{(1,159.9)} = 4.47, p = .036$), but only when their hierarchy was stable (Testosterone: $M = 371.77, SE = 5.92$; Placebo: $M = 391.31, SE = 6.19$; $z = -2.28, p = .022$), which was enhanced in low AR(CAG) repeat men ($z = 3.01, p = .003$) (see Fig. S5). In this second part, trait dominance did not further moderate the effects ($p = .334$).

< Insert Figure 3 and Table 3 here >

Testosterone increases final decision to compete in those with unstable low rank

At the end of the competition, participants had to decide if they wanted to compete again one final time to maintain or change their rank, or to play the task alone outside of competition. There was no significant three-way interaction (treatment $\times$ rank $\times$ stability, $\chi^2(1) = 1.17, p = .279$), yet, a hierarchy

stability by treatment interaction ($\chi^2(1) = 4.00, p = .046$) revealed that testosterone increased choice to compete in men who experienced an unstable hierarchy ($t(79.38) = -2.07, p = 0.042$, see Fig. 4). Since we hypothesized testosterone to increase competitive behavior in those with an unstable low status and with a stable high status, we ran the same analysis split by rank to explore whether we also find a hierarchy stability by treatment interaction in the low rank group. This analysis revealed a significant stability by treatment interaction for the low rank group ($\chi^2(1) = 4.82, p = .028$), but not for the high rank ($\chi^2(1) = 0.31, p = .576$). Although we cannot claim that there is a statistically significant difference of stability by treatment effects between the high and low rank groups (since we lack a significant three-way interaction), this would be in line with the hypothesized direction of effects: Testosterone increased decisions to compete, but only within men with an unstable low rank (Testosterone: $M = 88.2\%$, $SE = 7.8$, Placebo: $M = 59.1\%$, $SE = 10.0$, $z = 2.23, p = .026$, Fig. 4). Again, when the hierarchy was stable, testosterone instead decreased choice to compete in low-ranked men ($M = 47.6\%$, $SE = 10.9$, $z = 3.03, p = .003$, Fig. 4). Trait dominance and AR(CAG)-repeat polymorphism did not significantly moderate choice behavior.

< Insert Figure 4 here >

4. Discussion

The main aims of our study were to investigate if testosterone causally affects status-seeking motivation in repeated social contests in men with high or low status, and whether this relationship is moderated by hierarchy stability. Based on prevailing models of testosterone (Mazur and Booth, 1998; Zilioli et al., 2014), we hypothesized that testosterone reinforces status-seeking motivation predominantly when there is an opportunity to attain higher social status (such as with unstable low status), and inhibit status-seeking motivation when status may be lost or remain low (such as with

unstable high status or stable low status, respectively). Our results confirmed our predictions. Specifically, in the first part of the competition—which simulated an unstable hierarchy—testosterone increased competitive effort across multiple challenges, but only in individuals with unstable low status. This suggests that testosterone enhances motivation to compete in men with low but unstable status³, in an attempt to attain higher status. Indeed, in the second part of the competition we further tested whether these effects were specific for unstable hierarchies by directly manipulating stability to be stable or—as in the first part—unstable. Again, confirming our hypothesis, we found that testosterone’s time-dependent effects on motivation in men with low status was only present in those within an unstable hierarchy. In contrast, testosterone reduced motivation in men with a stable low status – who had no opportunity to attain higher status. Moreover, testosterone significantly increased competitive effort in individuals with a stable high status – who saw that their performance was distinctively better than of others, but testosterone decreased competitive effort in individuals with unstable high status, albeit non-significant. Here, individuals were challenged nevertheless due to a forced competition and may have induced a context of status threat and stress to avoid a status loss in individuals with an unstable high status (Scheepers, 2009). This could explain why we found no observable difference of testosterone on motivation in those men. Indeed, Knight and Mehta (2017) showed that individuals with an unstable high status had increased testosterone levels, stress (assessed by cortisol reactivity), and a reduced feeling of control, but showed no effect on performance. Together, our results show that exogenous testosterone encourages competition for status adaptively, but only in the presence of opportunities to attain high(er) social status. Thereby we provide an important extension to previous studies and behavioral models of testosterone.

³ These effects were further enhanced among those high in trait dominance, consistent with prior studies (Carré et al., 2017; Geniole et al., 2019; Mehta et al., 2015), and might be due to greater reward associated with high dominance (Geniole et al., 2019; Qu et al., 2017). Nevertheless, the effect of trait dominance was not robust across the different measures we used, which may partly be a result of reduced statistical power.

Our results on competitive effort were also partially reflected by the decisions to compete at the end of the competition, as another index of status-seeking motivation. First, as predicted by the challenge hypothesis (Wingfield et al., 1990), we found that exogenous testosterone increased the proportion of individuals deciding to compete again, and only among those with an unstable status. Importantly, based on our hypothesis, when further exploring choice to compete separately for rank, testosterone (vs. placebo) increased decisions to compete again in men with an unstable low status, to encourage a final attempt to gain higher status, but decreased decisions to compete again in men with a stable low status, when status cannot be further lost nor gained. Hence, these results further support our predictions based on the status instability hypothesis, suggesting that testosterone only increased status-seeking behavior when status can be gained. In contrast, testosterone (vs. placebo) did not differentially influence decisions to compete in men with a high status independent of the hierarchy stability. One reason could be that participants knew others could still decide to compete to influence the status hierarchy, and thereby affect the participant's ultimate high status. Hence, deciding to compete a final time may have reflected high motivation to avoid a potential status loss. Results support this by showing an overall increase in decisions to compete in those with high status, although independent of testosterone. Altogether, our results show that in men with unstable low status, testosterone not only boost performance necessary to gain higher status, but also tends to increase the willingness to decide to enter a further challenge.

Our findings contribute to other recent pharmacological studies using different behavioural paradigms, and suggest a causal role of testosterone in status-enhancing behaviors that depend on the social context. For instance increase preference for high status goods (Nave et al., 2018; Wu et al., 2017), increase willingness to punish a proposer who proposed an unfair offer in an Ultimatum Game, and increase willingness to reward those who proposed a fair offer (Dreher et al., 2016). Future studies could test if our findings of testosterone in individuals with unstable low social status also extend to

behaviors in these paradigms. Consistent with these and other studies reviewed here, exogenous testosterone may reinforce behavior for a higher social status within a dominance hierarchy, specifically in individuals who have the opportunity to improve social status.

What are the underlying neurobiological pathways through which testosterone might affect status-seeking behavior? Previous research has hypothesized that behavioral effects of testosterone are mediated by androgen-dependent pathways (Simmons and Roney, 2011). For example, individuals with fewer AR(CAG) repeats who chose to compete showed greater confidence and actual performance in competition (Eisenegger et al., 2017). However, in that study status and hierarchy stability had not been directly tested. In the current study, individuals with high androgen receptor sensitivity to testosterone (i.e., low AR(CAG)-repeat length), showed that testosterone (vs. placebo) increased motivation to compete when the competition progressed in those who had an unstable low status as well as in those who had a stable high status. A recent study in males demonstrated testosterone to promote aggression only among individuals with fewer AR(CAG) repeats (Geniole et al., 2019), which further supports our findings. In sum, these exploratory findings suggest that the conditional effects of testosterone on status-seeking motivation are moderated by androgen receptor-dependent pathways.

Furthermore, testosterone's influence on motivation might result via actions on the dopaminergic system. Research in rats have shown high density androgen receptors on dopaminergic neurons in mesolimbic areas (Creutz and Kritzer, 2004), and exogenous testosterone to increase dopamine synthesis and release (de Souza Silva et al., 2009; Purves-Tyson et al., 2014), and discount effort for high reward (Tobiansky et al., 2018). Moreover, in humans, dopamine receptor availability in the ventral striatum is correlated with status in humans (Martinez et al., 2010). One hypothesis was, thus, that the obtained effects of testosterone on behavior might be mediated by modulating striatal dopamine to enhance motivation, by discounting effort and increasing reward (Croxxon et al., 2009; see for review Losecaat Vermeer et al., 2016). Our results support this by showing that testosterone (vs.

placebo) enhanced motivation for higher status via increased effort exertion in individuals with unstable low status that had higher striatal dopamine availability (i.e., 9/10R genotype). In sum, we speculate that testosterone influences status-seeking motivation via androgen-dependent actions on striatal dopamine, thereby modulating the cost-benefit ratio of effort-for-status. Despite a small sample and consequently reduced statistical power, our study design and collection of genetic data allowed us to explore these possible pathways via which exogenous testosterone may influence status-seeking motivation in competition in men with unstable low status. Our findings -although with necessary caution- provide novel insight in the so far understudied area that uses psychopharmacogenetic approaches to understand testosterone effects on social behavior and human decision-making.

There are some limitations to the current study. First, due to our pharmacological approach we only included men. However, this could also be seen as a strength as our results are broadly in line with and extend to previous research in women (Knight and Mehta, 2017; Zilioli et al., 2014). Future studies are required to test whether our effects extend to both genders (Josephs et al., 2011). Second, we used a novel approach to assess motivation for status under testosterone administration by using real-effort. It would be important to replicate and test whether our results would extend to other measures of status-seeking behaviors. The benefit of using a simple tedious real-effort paradigm was to provide a quantifiable measure of status-seeking motivation without a potential confound of individual differences in skill or ability. Previous research has shown that these factors can moderate the testosterone-behavior relation (Van Anders and Watson, 2007; van der Meij et al., 2010). Third, as our analysis on hierarchy stability and genetic polymorphisms resulted in relatively small sample size per cell (i.e. 21 participants per group), this reduced statistical power, and the resulting findings should therefore be interpreted with caution. This also prevented us from testing all models. Though, while our sample size per cell was small, it was still in the range of other recent psychopharmacological studies on testosterone and behavior (using a range from 20-32 participants per cell; e.g. Dreher et al., 2016;

Heany et al., 2018; Mehta et al., 2015; Wu et al., 2018). Nevertheless, we acknowledge that due to general power issues in social sciences, larger sample sizes are required and starting to be used also in psychoneuroendocrinological work. Therefore, it is important to test whether these effects can be replicated and extended in larger samples (see also Geniole et al., 2019, though, for comparable results in a larger sample).

To conclude, using a combination of real effort and binary choice, we found that testosterone enhanced status-seeking by increasing effort and decisions to compete in men with unstable low status, providing novel evidence for the behavioral predictions of the status instability hypothesis. Furthermore, the effects were enhanced among those with high trait dominance, high androgen-receptor sensitivity to testosterone and high striatal dopamine availability. Taken together, we show that testosterone adaptively regulates the motivation for high(er) status in a context-dependent manner by increasing status-seeking motivation in competition in those with opportunities to improve status. In addition, the effects of testosterone on behavior and motivation for status were moderated by AR-related and dopaminergic mechanisms. The current findings have relevance for clinical populations and society, for example the influence of social challenges and hierarchies on motivation to perform could benefit groups lacking overall motivation (i.e., hypogonadal patients), but also implications for behavior in different social hierarchical groups, such as underprivileged groups who may perceive few opportunities to move up the social hierarchy.

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Figures

Fig. 1 Real-effort based competition paradigm. A) Task design and outline of the first and second part of the real-effort competition phase. All four groups played the first part of the competition phase and then in the second part of the competition phase half of the subjects of each group experienced a stable rank, and the other half an unstable rank. B) Outline of a trial. Each picture represents a screen in the experiment. A trial began with the presentation of the one of the reward levels, followed by the effort part during which 5 squares are presented at random intervals for each reward level, with each trial always consisting of both high and low reward, counterbalanced. After the effort part their total score was shown. Before the presentation of the social rank, participants had to indicate their expectation of

their current rank position between 1-9, based on their performance. Social rank feedback could be either private (only the participant's own rank position is shown to himself) or public (all players' rank positions and names are shown to all the participants). For illustration, the blue and red square represent the range of positions held by participants in the high and low rank category, respectively.

Fig. 2 Effort to compete in competition part 1. Treatment (testosterone, placebo) by rank (high, low) on effort (RT) over time (trial). Testosterone (vs. placebo) increased effort over time, but only in individuals with unstable low rank. For illustration, data is averaged across public and private feedback. Average raw data is plotted as dotted lines, overlaid by model slopes as solid lines. Shaded area represents $\pm$ SE.

** $p < .01$

Fig. 3 Effort to compete in competition part 2. Treatment effects on effort by rank and stability (stable, unstable). Testosterone administration increased effort to compete over time in the unstable low rank, but decreased effort in the stable low rank. Testosterone increased effort on average in those with a stable high rank. Average raw data is plotted as dotted lines, overlaid by model slopes as solid lines. Shaded area represents $\pm$ SE. * $p < .05$, ** $p < .01$

Fig. 4 Decision to compete again. There is a clear indication for an interaction of testosterone by stability in men with a low rank, showing that testosterone increased decisions to compete again in men with an unstable low rank, but decreased decisions to compete in men with stable low rank. A treatment x stability interaction showed that testosterone (vs. placebo) increased decisions to compete again when rank was unstable ($M = 85.4\%$, $SE = 5.6$) versus stable ($M = 65.1\%$, $SE = 6.7$; $t(79.38) = -2.07$, $p = 0.042$). Error bars represent $\pm SE$. * $p < .05$, ** $p < .01$.

Tables

Table 1. Descriptive statistics by treatment (Placebo, Testosterone)

	Placebo (<i>n</i> = 84)		Testosterone (<i>n</i> = 84)		Statistics	
	<i>Mean</i>	<i>SD</i>	<i>Mean</i>	<i>SD</i>	<i>t-value</i>	<i>p-value</i>
Age	24.54	3.47	24.42	3.30	0.23	.820
BMI	24.25	3.31	24.14	2.69	0.24	.814
Trait Dominance	24.69	4.29	24.77	4.05	-0.13	.897
Testosterone (ng/mL) - t_0 ^a	0.229	0.76	0.152	0.11	0.34	.737
Testosterone (ng/mL) - t_{120} ^a	0.427	1.13	49.16	149.39	-13.92	<.001
Cortisol (ng/mL) - t_0 ^a	3.67	2.42	3.76	2.66	0.47	.639
Cortisol (ng/mL) - t_{120} ^a	1.88	1.75	1.84	1.63	-0.15	.878
Initial belief status	4.07	1.61	4.10	1.51	-0.10	.921
Rank expectations – high rank ^b	2.87	0.62	2.90	0.61	-0.20	.844
Rank expectations – low rank ^b	6.64	1.63	7.24	0.56	-2.23	.030
Belief testosterone (<i>n</i>)	21		18		0.55	.586
Confidence about belief	56.13	26.00	55.82	25.42	0.08	.938

Note: ^a Seven samples at t_0 (2 in placebo, and 5 in testosterone), and two samples at t_{120} (testosterone only) could not be analyzed due to insufficient saliva, ^b Two participants did not provide their expectations during the task, resulting in *n* = 83 testosterone, *n* = 83 placebo.

Table 2. Mean effort over time (slopes) in the first part of the competition

	Testosterone	Placebo	Statistics	
	<i>M (SE)</i>	<i>M (SE)</i>	<i>z</i>	<i>p</i>
Rank category				
High rank	-0.14 (0.08)	-0.11 (0.08)	0.25	.994
Low rank	-0.53 (0.08)	-0.09 (0.08)	3.89	<.001
Trait dominance				
High dominance, High rank	-0.23 (0.11)	-0.14 (0.13)	-0.50	.958
High dominance, Low rank	-0.53 (0.12)	0.34 (0.11)	-5.54	<.001
Low dominance, High rank	-0.06 (0.10)	-0.08 (0.10)	0.18	.998
Low dominance, Low rank	-0.53 (0.11)	-0.68 (0.12)	0.92	.797
AR(CAG)-repeat polymorphism				
High repeat, High rank	-0.02 (0.11)	-0.09 (0.13)	0.45	.970
High repeat, Low rank	-0.56 (0.13)	-0.47 (0.12)	-0.51	.958
Low repeat, High rank	-0.25 (0.11)	-0.09 (0.10)	-1.05	.720
Low repeat, Low rank	-0.50 (0.11)	0.26 (0.11)	5.00	<.001
DAT1 polymorphism				
9/10R, High rank	0.06 (0.12)	-0.11 (0.15)	0.87	.385
9/10R, Low rank	-0.69 (0.15)	0.12 (0.13)	-4.10	<.001
10/10R, High rank	-0.26 (0.10)	-0.17 (0.11)	-0.61	.540
10/10R, Low rank	-0.48 (0.10)	-0.21 (0.10)	-1.83	.067

Table 3. Mean effort over time (slopes) by treatment, rank, stability and AR(CAG)-repeat polymorphism in the second part of the competition

	Testosterone	Placebo	Statistics	
	<i>M (SE)</i>	<i>M (SE)</i>	<i>z</i>	<i>p</i>
Stable High rank	-0.07 (0.35)	0.11 (0.37)	-0.35	.724
High AR(CAG)-repeat	0.20 (0.50)	-0.25 (0.58)	0.59	.559
Low AR(CAG)-repeat	-0.36 (0.49)	0.38 (0.47)	-1.11	.269
Unstable High rank	-0.67 (0.35)	-1.02 (0.38)	0.69	.491
High AR(CAG)-repeat	-0.36 (0.53)	-0.65 (0.62)	0.37	.715
Low AR(CAG)-repeat	-0.96 (0.46)	-1.27 (0.47)	0.47	.636
Stable Low rank	0.83 (0.37)	-0.56 (0.37)	2.67	.008
High AR(CAG)-repeat	1.00 (0.51)	-0.48 (0.50)	2.08	.038
Low AR(CAG)-repeat	0.61 (0.54)	-0.66 (0.54)	1.65	.098
Unstable Low rank	-0.95 (0.41)	0.14 (0.36)	-2.01	.044
High AR(CAG)-repeat	-1.24 (0.88)	-0.83 (0.58)	0.38	.701
Low CAG-repeat	-0.86 (0.45)	0.80 (0.45)	-2.64	.008