Research article

Title: Testosterone increases risk-taking for status but not for money

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1 **Abstract**

2 Testosterone has long been thought to increase risk-taking, but evidence supporting this association
3 is mixed. Instead, testosterone's key role may be to promote status-seeking behaviors. Here, we
4 examined to what extent testosterone administration affects risk preferences for both monetary and
5 social status outcomes, and whether this relationship is moderated by an individuals' social status.
6 Male participants ($N=166$) experienced high or low status in a competition task and then played two
7 risk tasks; one involving gambles with only monetary outcomes, and another one involving gambles
8 with non-monetary outcomes that influenced their social rank. We found that testosterone (vs.
9 placebo) altered risk preferences for gains and losses in social rank, but not for monetary gains and
10 losses. Specifically, testosterone increased risk-taking to increase social rank in individuals with high,
11 but not low social status. These results demonstrate a context-dependent role of testosterone in
12 regulating risk-taking for social status.

13 **Keywords**

14 testosterone; social status; risk; decision making; dopamine; genetic polymorphism

Introduction

Testosterone is a steroid hormone primarily associated with status-seeking behaviors such as aggression and competition (Eisenegger, Haushofer, & Fehr, 2011). In parallel, there is growing interest for its proposed role in non-social behaviors such as risk-taking. A popular lay belief is that testosterone causes excessive risk-taking (e.g. reckless driving, and competition more generally), with news outlets even blaming the hormone for the financial crisis in 2008 (e.g. Scientific American, 2009). To date, the scientific literature has been relatively mixed about whether testosterone actually increases risk-taking. A major remaining question is, under which circumstances testosterone causally affects risk-taking, and if this depends on whether risk-taking can be a means to achieve higher social status or not.

Both in the field and laboratory experimental settings (reviewed in Apicella, Carré, & Dreber, 2015; Stanton, 2017), testosterone levels correlated with changes in economic decision-making. For example, changes in testosterone predicted increased preference for risky choices involving monetary gains and losses (Apicella, Dreber, & Mollerstrom, 2014). Other work found enhanced risk-taking, but only among individuals with high and low baseline testosterone levels (Stanton et al., 2011), and testosterone to influence risk-taking only in individuals with low baseline cortisol levels (Mehta, Welker, Zilioli, & Carré, 2015). Nonetheless, there have also been reports of null results (e.g., Derntl, Pintzinger, Kryspin-Exner, & Schöpf, 2014).

Of the several investigations on the causal influence of exogenous testosterone on risk-taking, only half reported positive or mixed evidence (Cueva et al., 2015; Goudriaan et al., 2010; Nadler, Jiao, Johnson, Alexander, & Zak, 2017; van Honk et al., 2004; Wu et al., 2016), whereas others found no statistically significant effect (Boksem et al., 2013; Zethraeus et al., 2009). In sum, both correlational and testosterone administration studies demonstrate that testosterone's influence on risk-taking remains debated.

Risk-taking may function as a means for attaining higher social status (Mehta, Welker, et al., 2015). For instance, individuals' relative status has been shown to influence risk-taking in a competition to

gain a higher status (Ermer, Cosmides, & Tooby, 2008). One proposal is that testosterone may function to enhance risk-taking to attain higher social status, but not or to a lesser degree when risk-taking does not directly serve one's social status.

Theoretical models of testosterone (Archer, 2006; Mazur & Booth, 1998) postulate that testosterone fluctuates with social challenges and predicts status-relevant risky behaviors, such as aggression (reviewed in Geniole & Carré, 2018), competition (Casto, Edwards, Akinola, Davis, & Mehta, 2020; Losecaat Vermeer et al., 2020; Mehta & Josephs, 2006; Mehta, Son, et al., 2015), and social trust (Boksem et al., 2013). While some work controlled for individual risk-taking by using a separate non-social task (Boksem et al., 2013; Eisenegger, Kumsta, Naef, Gromoll, & Heinrichs, 2017), no studies to date tested whether testosterone alters risk-taking with direct status implications, and whether this depends on individuals' social status levels (see Fisk, Miller, & Overton, 2017).

According to the *Biosocial Model of Status* (Mazur & Booth, 1998), changes in testosterone following a win (i.e. equivalent to high status) may reinforce status-enhancing risky behaviors, whereas changes following a loss (i.e. equivalent to low status) may inhibit status-enhancing behaviors. This directly motivated the *risk-for-status hypothesis* in the present investigation. Extending on previous theories of the effects of testosterone, we predicted that high testosterone in individuals with high social status increases risk-taking when this could serve attaining higher status, whereas high testosterone in individuals with low social status should not, or even reduce risk-taking that can impact their social status. Importantly, these considerations would also predict that testosterone only influences risk-taking when it has direct implications for social status, and not when risk-taking has no implications to status.

To test this, we performed a double-blind placebo-controlled administration study in which men received either testosterone or placebo, and played a competition task where their social status was manipulated to be either high or low, indicated via a ranking. Following the competition, participants played two gambling tasks. The first task only contained monetary gain-loss gambles with no impact to their social rank. The second task contained gambles with gain-loss outcomes in social rank without

monetary incentives, to assess risk-taking for social status independent of monetary reward. The present study is thus the first to simultaneously manipulate testosterone and status to test their effects on risk-taking, while additionally dissociating risk preferences for money and for status.

Testosterone effects on status-seeking behaviors may depend on the functioning of the testosterone-binding androgen receptor (Eisenegger et al., 2017; Geniole et al., 2019; Losecaat Vermeer et al., 2020), and testosterone's actions on the striatal dopaminergic reward system (Losecaat Vermeer, Riečanský, & Eisenegger, 2016; Mehta, Son, et al., 2015). We thus explored whether effects of testosterone on risk-taking are also moderated by individual genetic differences, focusing on polymorphisms related to the androgen receptor, and striatal dopamine. The efficiency of the androgen receptor is negatively correlated with the cytosine-adenine-guanine (CAG)-repeat length in exon 1 of the androgen receptor gene (Zitzmann & Nieschlag, 2003), therefore we determined CAG-repeat length for each participant. Moreover, testosterone increases dopamine and activity in the ventral striatum, in animals (de Souza Silva, Mattern, Topic, Buddenberg, & Huston, 2009) and humans (Hermans et al., 2010). We thus measured a polymorphism related to DAT1 (Heinz, 2000), the dopamine transporter, as a proxy for striatal dopamine.

Method

Participants

One hundred-seventy-three male volunteers participated in the study. This sample size was directly informed by previous high-profile single-dose testosterone administration studies on status-relevant behavior and risk-taking (e.g. Boksem et al., 2013; Carré et al., 2017; Eisenegger, Naef, Snozzi, Heinrichs, & Fehr, 2010), and work on status-seeking behavior in relation to CAG-repeat polymorphism (Eisenegger et al., 2017). Participants were pre-screened and had no history of psychiatric, neurological, or endocrine disease, nor were currently using or had been regular users of marijuana or any psychotropic drugs and corticosteroids, and had not taken part in a medical study in the four weeks preceding the study. The pharmacokinetics of testosterone gel administration has been established in

men (Eisenegger, von Eckardstein, Fehr, & von Eckardstein, 2013), therefore our sample only included men. Seven participants were excluded; six participants due to a technical error, which resulted in either a partial or complete loss of their data ($n=2$), or in incorrect rank presentation ($n=4$), and one participant who was unintentionally included twice in the study. The final sample consisted of 166 men (Placebo ($N=84$): $M=24.55$ years, $SD=3.64$; Testosterone ($N=82$): $M=24.38$ years, $SD=3.33$). All participants provided consent and were financially compensated for completing the study. 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).

General procedure

Participants arrived to the lab on two separate days. On the first day, participants filled out a series 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). At the end of the day, participants received 10 Euro endowment in advance to enhance the feeling of ownership of this money which they could later lose in the subsequent experiment. They were instructed that this endowment was theirs to keep and to pay for any losses during the risk task on the second test day.

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, they completed additional questionnaires assessing self-reported mood and personality traits such as trait dominance (16-PF-R, Schneewind & Graf, 1998), which has previously shown to moderate effects of testosterone on status-seeking, mainly in individuals with high trait dominance (Mehta, Son, et al., 2015). Twenty minutes after arrival, participants provided the first saliva sample (t_0), to assess baseline hormone concentrations. Next, participants were administered with testosterone or placebo by random assignment (section *Drug administration*). Participants waited in the lab for two hours to allow the drug to reach peak level, but did not interact with others. Two-hours post-administration, participants

completed a second mood questionnaire to control for a potential confound of treatment, see Supplementary Online Material (SOM), and provided their second saliva sample (t_{120}). To minimize diurnal hormone variability, the behavioral testing occurred from 1:00-3:00 p.m. Testing included a competition paradigm with a brief post-task questionnaire (Losecaat Vermeer et al., 2020), followed by the risk paradigm and then one other task which is unrelated to the question reported here. At the end, we assessed participants' beliefs about whether they believed they had received testosterone or placebo via a binary choice, and their confidence about their answer on a scale from 0-100 (Table 1). After that we paid them for participation and paid out any gains from the risk task.

Table 1. Descriptives by treatment (Placebo, Testosterone)

	Placebo (N=84)			Testosterone (N=82)			Statistics	
	<i>n</i>	<i>M</i>	<i>SD</i>	<i>n</i>	<i>M</i>	<i>SD</i>	<i>t-value</i>	<i>p-value</i>
Age (years)	84	24.55	3.64	82	24.38	3.33	0.314	.754
BMI	80	24.27	3.35	82	24.12	2.69	0.331	.741
Trait Dominance	84	42.52	24.17	82	45.82	25.78	- 0.849	.397
Testosterone log (ng/mL) - t_0	82	0.08	0.36	77	0.05	0.43	0.447	.656
Testosterone log (ng/mL) - t_{120}	84	0.33	0.49	80	3.63	1.53	- 18.48	<.001
Cortisol log (ng/mL) - t_0	82	0.60	0.59	76	0.58	0.70	0.269	.789
Cortisol log (ng/mL) - t_{120}	84	- 0.18	0.61	80	- 0.15	0.60	- 0.284	.777
Drug belief (%)	84	0.24	0.43	82	0.23	0.42	0.096	.923
Drug belief confidence (0-100%)	84	56.40	25.24	82	55.39	26.39	0.253	.801

Note: for some comparisons *n* slightly differs from the total *N* of each group, i.e. some saliva samples were unanalyzable, and four BMI scores of the placebo group were not recorded. Drug belief (%) = percentage of participants who believed they had received testosterone, t_0 = baseline, t_{120} = post-administration.

Task procedure

Prior to the risk task, we experimentally induced high or low social status in participants via a competition task (Losecaat Vermeer et al., 2020), which included nine players and lasted for approximately 60 minutes. In brief, the competition paradigm entailed a reaction time task, in which participants had to respond as fast as possible to a target on screen appearing at random intervals. The faster participants responded, the more points they could earn. Participants repeatedly played against each other and based on their score on each round they saw they obtained either a high (varying between 1–4) or low (varying between 6–9) position in a ranking list. Unbeknownst to

participants, in reality their ranks were based on random assignment. Participants sat in the same room, in individual cubicles to make the competitive task more realistic.

Following the competition, participants performed the risk paradigm, consisting of two tasks. In the first task, participants performed a series of gambles involving monetary gains and losses. In the second task, participants performed a series of gambles involving social gains and losses (increases or decreases in the social ranking), without monetary outcomes. Participants were briefly reminded about their final position in the ranking resulting from the previously completed competition task, before receiving instructions and practice trials to perform the monetary risk task (Fig. 1a). Next, for the status risk task, we explicitly instructed participants that the outcome of the upcoming gambles would *only* affect their rank position by adding or deducting points from their score achieved in the competition, and could result in gain or loss in rank respectively. To compare high and low ranked participants, we presented participants with a new ranking consisting of players whom had previously participated in the experiment and had the same rank position. Specifically, both high and low-ranked participants were compared to eight participants with an identical rank position to themselves. In this new ranking, the name of each player and their score was shown, including the difference in points compared to the participant, who was placed in the middle (i.e. 5th). This was done to provide the opportunity to enhance and reduce further status by gaining or losing position in the ranking based on the outcome of these gambles. This new rank list was shown at all times at the top of the screen (Fig. 1b).

Risk paradigm

We developed a novel paradigm consisting of a monetary and status risk task in fixed order. For both, participants had to choose whether to accept or reject a gamble with an equal probability to win or lose. If participants decided to reject the gamble they would simply not gain or lose anything. No immediate feedback was given to avoid carry-over effects of the outcome to the next trial. At the end

of the experiment, one gamble of each task was randomly chosen and played in case the participant had chosen to accept the gamble.

Figure 1. Risk paradigm. (a) Example of a monetary risk trial of a high ranked participant. After competition participants saw their name in the rank list. Followed by this rank list, participants could decide to accept the gamble with equal probability to win or lose or reject it (i.e. choose € 0). (b) Example of a status risk trial of a high ranked participant. At the beginning, the participant saw a new ranking, consisting of players who had previously participated in the experiment and ended up in the participants' rank category (i.e. here high). While the participant's precise rank is number one, his performance is ranked 5th among the other players of his rank category (i.e. whom also ended up as number one in their competition). Participants could decide to accept or reject gambles with equal probability to win or lose status points for rank position. No immediate feedback was provided in both tasks.

I. Monetary risk task

Sixteen trials, each containing a monetary gain/loss gamble were presented. The gambles had a fixed gain of € 12 and a loss ranging from € -1 to € -16, with steps of € 1 (Fig. 1a). On each trial, participant's rank was shown at the top of the screen. However, participants were instructed that the outcome of these gambles only had consequences for their monetary endowment of € 10, but not for their status in the experiment.

II. Status risk task

Sixteen trials, each containing a gain/loss gamble in terms of points were presented. The gain and loss points ranged from 30 to 120 (in steps of 30 points) and had consequences for participants' position in the rank list. Points would be either added or subtracted from the participants' score depending on

the gain or loss outcome and determined their final social status in the experiment. On each trial, the players' rank, including their score and difference in points to the participant's score was shown, for the participants to see the potential impact a specific gamble would have on their position on the ranking. The task also contained two gambles with an expected value of zero, but one with high and one with low variance (i.e. risk level). The high variance gamble provided a gain or loss of 120 points (i.e. gain/loss of 4 positions, resulting in first or last place), whereas the low variance gamble provided a gain or loss of 30 points (i.e. gain/loss of 1 position, resulting in 4th or 6th place respectively). There were also two catch trials to check if participants paid attention in which with an equal probability one could win 1 position or lose none, or lose 1 position or win none. Catch trials were excluded from analysis. The outcomes of the status gambles had no monetary consequences throughout, neither immediately nor later in the experiment.

Drug administration

We randomly assigned participants to receive a single dose of 150 mg testosterone (Testogel®, N=82) or placebo gel (N=84) in a double-blind procedure. The gel was topically applied 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). This showed testosterone levels to peak 3 h post-administration with a significant increase after 2 h. Therefore, behavioral testing commenced 2 h post-administration, with the risk task 195 min post-administration. Recent work using larger samples and same dosage observed a significant increase in testosterone levels as fast as 1 h post-administration (see e.g. Carré et al., 2017). Notwithstanding, evidence of these studies when taken together suggest that testosterone levels were still sufficiently high in our testing window.

Hormonal assays

Hormone samples were collected via passive drool into a 2 mL polypropylene vial and stored at -30 degrees Celsius. Saliva samples were analyzed for testosterone and hydrocortisone with liquid

chromatography tandem mass spectrometry (LCMS/MS) (see SOM for details). Saliva values were winsorized based on three median absolute deviations from the median and log-transformed. Table 1 shows baseline hormone concentrations (t_0) did not differ between treatment groups, while testosterone concentration after administration (t_{120}) was significantly increased in the testosterone vs. placebo group, confirming our manipulation was successful.

Genotyping

DNA was collected using sterile cotton buccal swabs (Sarstedt AG, Germany), and purified applying the QIAamp DNA Mini kit (Qiagen, Germany). CAG-repeat and DAT1 VNTR polymorphisms were investigated by multiplex PCR with fluorescent-dye-labeled primers and capillary electrophoresis (detailed in SOM). Treatment groups (Testosterone $M = 22.11$, $SD = 3.12$, Placebo $M = 22.44$, $SD = 2.80$) did not differ in mean CAG-repeat length ($t(161.32) = 0.72$, $p = .474$). For the DAT1 VNTR polymorphisms, 9/10-repeat carriers (9/10R: 34%; testosterone $n=29$ placebo $n=27$) and 10/10-repeat carriers (10/10R: 57%; testosterone $n=48$, placebo $n=46$) occurred most in our sample (see also SOM). Therefore we used these two genotypes in the analyses, with 10/10-Repeat carriers assumed to have higher DAT, expression and hence lower striatal dopamine than 9/10-Repeat carriers (Heinz, 2000).

Statistical analysis

To assess whether testosterone administration alters risk-taking, we ran a generalized mixed effects model on the decision to accept or reject the gamble, using *lme4* (Bates, Mächler, Bolker, & Walker, 2015) in the R statistical software package (version 3.4.3, R Development Core Team, 2017). The model included between-subject predictors (at level two) for Drug (testosterone, placebo) and Rank (high, low), and a within-subject predictor (at level one) for Expected Value of the gamble, including their interactions. The model also included a random intercept for participant including by-participant random slopes for expected value of the gambles to account for repeated observations and variability among participants. We ran this model separately on the behavior for the monetary gamble task and

for the status gamble task, because these were played independently in a fixed order and had different magnitudes and expected values, and thus were not entirely comparable. P values for regression coefficients were determined with Likelihood Ratio Tests. Reported estimates are odds ratios (OR) and Wald 95% confidence intervals (CIs), and means are estimated marginal means unless otherwise stated. Planned and post-hoc comparisons were obtained using *emmeans*, based on two-tailed tests where no other statistical corrections (e.g., adjustments for multiple comparisons) were made. The total sample broken down for each group: Testosterone ($n=82$) including high rank $n=45$ and low rank $n=37$; Placebo ($n=84$) including high rank $n=40$, low rank $n=44$.

Moderators

Baseline cortisol, trait dominance, CAG-repeat and DAT1 polymorphisms were separately included in the model as described above, to explore whether they influenced the effect of testosterone on risk-taking depending on social status. Expected value of the gambles was included as predictor and random effect only, without interactions, to reduce model complexity, which was supported by model comparison (full model incl. interactions, $\chi^2(3) = 4.76$, $p=.199$).

Results

Social rank influences monetary risk-taking, independent of testosterone

Participants with a low rank position showed increased risk-taking for monetary gambles as compared to those with high rank (low rank $M = 35.7\%$, 95% CI = [21.9; 52.4], high rank $M = 13.8\%$, 95% CI = [6.9; 23.5], $p=.007$, Table 2, Fig. 2a). As shown in Table 2, testosterone (vs. placebo) did not differentially influence risk preferences for monetary gambles, nor was this influenced by whether participants had a high and low rank (drug x rank, $p > .05$, Fig 2b). Participants accepted more gambles with increasing expected value (Fig. 2c), but this was not significantly moderated by testosterone or the participant's rank (Table 2).

Trait dominance, baseline cortisol and individual polymorphisms of CAG-repeat and DAT1 genotypes, related to androgen function and dopamine, did not interact with drug or rank to predict risk-taking (see SOM)¹.

Figure 2. Mean (raw) proportion risk-taking for monetary outcomes. (a) Risk-taking for low compared to high rank category. Every dot represents participants' mean. (b) Risk-taking by drug group (testosterone or placebo) of high and low rank showed no significant interaction. (c) Risk-taking by expected value of the gamble (x-axis, range -2 to 5.5), for drug and rank. The horizontal line represents the point of indifference (i.e. the expected value where proportion of gambling is 0.50). Testosterone had no statistical significant effect on risk-taking by rank and expected value. Error bars and shaded bands represent 95% confidence intervals. Asterisks indicate a significant difference between rank (** $p < .01$).

Table 2. Results of generalized mixed effects models for Money and for Status, involving Drug (testosterone, placebo), Rank (high, low) and Expected Value (EV)

Predictors	Risk-taking for money (N=166)					Risk-taking for status (N=166)				
	Odds Ratios	SE	95% CI	z	p	Odds Ratios	SE	95% CI	z	p
Intercept	0.29	0.26	[0.17, 0.48]	-4.74	<.001	0.16	0.21	[0.11, 0.25]	-8.59	<.001
Rank	0.52	0.24	[0.32, 0.84]	-2.70	.007	0.73	0.18	[0.51, 1.04]	-1.75	.081
Drug	0.88	0.24	[0.55, 1.41]	-0.52	.602	1.21	0.18	[0.84, 1.73]	1.02	.305
Expected Value (EV)	7.31	0.14	[5.58, 9.56]	14.49	<.001	1.16	0.01	[1.14, 1.19]	14.04	<.001
Rank x Drug	1.22	0.24	[0.76, 1.96]	0.84	.399	1.63	0.18	[1.13, 2.33]	2.64	.008
Rank x EV	1.13	0.10	[0.94, 1.37]	1.29	.197	1.01	0.01	[1.00, 1.03]	1.76	.078
Drug x EV	1.00	0.10	[0.82, 1.20]	-0.04	.967	1.00	0.01	[0.98, 1.01]	-0.46	.649
Rank x Drug x EV	1.07	0.10	[0.89, 1.29]	0.70	.481	0.99	0.01	[0.98, 1.00]	-1.31	.189
Marginal R ² / Conditional R ²	0.594 / 0.910					0.671 / 0.895				

Note: sum-to-zero contrasts were applied (i.e. effect coding). Testosterone and High rank were coded as 1. Expected Value was centered at 0.

¹ Including baseline cortisol or DAT1 resulted in a marginal effect of rank ($p=.081$, $p=.0506$, respectively).

Testosterone increases risk-taking for status outcomes in individuals with high social status

Testosterone influenced risk-taking for status depending on participants' rank (Table 2), such that testosterone (vs. placebo) increased risk-taking in participants with a high rank (OR = 3.85, 95% CI = [1.36, 10.92], $z = 2.10$, $p = .036$), but not in participants with a low rank (OR = 0.55, 95% CI = [0.20, 1.49], $z = -1.07$, $p = .285$, Fig. 3a). Rank had a similar effect on risk-taking for status as on risk for money, but this just failed to reach significance ($p = .081$). Participants accepted more gambles with increasing expected value, though no interaction with drug or rank was observed (Table 2, Fig. 3b).

Figure 3. Mean (raw) proportion risk-taking for status. (a) Risk-taking for each drug group (testosterone or placebo) of high or low rank. Testosterone (vs. placebo) increased risk-taking in participants of high rank, but not low rank. Every dot represents participants' mean. (b) Risk-taking by expected value of the gamble (x-axis), for drug and rank. Expected value influenced risk-taking, but not differentially for drug or rank. The horizontal line represents the point of indifference (i.e. the expected value where proportion of gambling is 0.50). Error bars and shaded bands represent 95% confidence intervals. Asterisks indicate a significant difference between drug (* $p < .05$).

Baseline cortisol, trait dominance, CAG-repeat and DAT1 polymorphisms did not interact with drug and rank to predict risk-taking². Despite a nonsignificant interaction, when observing the data of high and low cortisol groups (based on median split), the conditional effect of testosterone seemed more pronounced in individuals with low baseline cortisol (for details see SOM). However, testosterone influenced risk-taking depending on DAT1 genotype (95% CI = [0.48, 0.97], $p = .035$), with

² The main drug x rank interaction changed to marginal significance when including trait dominance ($p = .056$) or DAT1 ($p = .081$), but drug x rank interaction was significant in all other models. Importantly, including all four as covariates in one model demonstrated that the drug x rank interaction effect was robust ($p = .024$, see SOM).

testosterone (vs. placebo) appearing to increase risk-taking in 10/10-carriers, and reduce risk-taking in 9/10-carriers.

Discussion

In the current study we investigated whether exogenous testosterone influenced risk-taking for choices that could alter the individual's social status level, and for choices with pure monetary outcomes, and whether this was dependent on the individual's current social status (i.e. high or low rank). This was mainly motivated by previous mixed results regarding the influence of testosterone on risk-taking, which had not accounted for the potentially important moderating role of social status. Based on prevailing models and research on testosterone function and status-seeking (Archer, 2006; Mazur & Booth, 1998), we formulated a *risk-for-status* hypothesis that predicted testosterone to influence risk-taking when this could serve to attain or maintain high social status. Conversely, this hypothesis predicted that testosterone would not (or less so) influence risk-taking that does not provide an opportunity to improve social status (e.g. gambling to only gain money).

Results confirmed our risk-for-status hypothesis. Exogenous testosterone (vs. placebo) enhanced risk-taking for status in individuals with high social rank, but not in individuals with low social rank. Importantly, monetary risk-taking was unaffected by testosterone administration. Our findings crucially add to previous theories and studies on testosterone and status-seeking (Eisenegger et al., 2011), by showing that testosterone influenced risk preferences for options with a possible impact to social status in men with a high social rank, but not for options with monetary outcomes without direct status consequences.

Our findings are of relevance for previous research reporting an overall positive but weak association of testosterone and monetary risk-taking (Cueva et al., 2015; Goudriaan et al., 2010; Mehta, Welker, et al., 2015; Nadler et al., 2017; van Honk et al., 2004; Welker, Roy, Geniole, Kitayama, & Carré, 2019). Besides differences in methodologies and sex/gender of the samples (previous work mainly used female participants), most of the studies have not manipulated the relative social status

or accounted for the implications for one's social status as a contextual factor in their design and analysis. Therefore, one possible reason for the previously reported inconsistencies in the literature for testosterone and risk-taking is whether individuals could influence their social status through risk-taking. In the current study, we explicitly told participants that the monetary gambles would *not* influence their status level, thereby excluding any perceived challenge or opportunity to improve status. By combining a pharmacological intervention with a risk task containing gambles with status outcomes, we showed that the social implications of choices are an important moderator of testosterone function in risk-taking. We therefore recommend future studies to consider social status in their design and analysis, to further elucidate testosterone's role in risk-taking.

Low social status has in previous studies been associated with increased monetary risk-taking (Welker, Goetz, & Carré, 2015). Likewise, we found increased risk-taking for monetary gambles in individuals with an experimentally manipulated low compared to high social status. Importantly, their status level was irrelevant to the choices at hand. Previous research for monetary choices has shown that people typically avoid risk following a gain, but take risk following a loss, even when these prior gain and loss outcomes were unrelated to the current choice (Losecaat Vermeer, Boksem, & Sanfey, 2014; Tversky & Kahneman, 1981). Relatedly, the anticipation of a social loss increased risk-taking (i.e. overbidding) in an experimental auction (Delgado, Schotter, Ozbay, & Phelps, 2008). Our results add to these findings, demonstrating that a high or low social status influenced risk preferences in a similar direction as previously experienced monetary losses. Importantly, this was the case even though status information had no relevance to the decision.

Previously identified moderators of testosterone (i.e. trait dominance, baseline cortisol, CAG-repeat) did not influence effects of testosterone and rank on risk-taking in the present experiment. Although we found no statistical interaction with basal cortisol, applying a median split, the conditional effect of testosterone on risk-taking for status tends to be more pronounced in individuals with low cortisol, consistent with predictions of the dual-hormone hypothesis (Mehta, Welker, et al., 2015). In contrast to previous work on aggression and competition (Geniole et al., 2019; Losecaat Vermeer et

al., 2020), we found no association between high androgen receptor sensitivity to testosterone (CAG-repeat) and status-driven risk-taking. DAT1-genotype (hypothesized to affect striatal dopamine availability) influenced risk-taking for status depending on drug administration. While risk-taking was significantly different between DAT1-genotype individuals under placebo, testosterone administration appeared to reverse this effect. Testosterone increased risk-taking in 10/10R-carriers (i.e. lower endogenous dopamine), and to a smaller degree reduced risk-taking in 9/10R-carriers (i.e. higher endogenous dopamine), although this was nonsignificant at post-hoc level. Previous research demonstrated increased striatal activity during reward anticipation following testosterone administration, specifically in low intrinsically motivated women (Hermans et al., 2010). Consistent with this, our findings may suggest that testosterone acts on striatal dopamine pathways to motivate risk-taking when anticipating reward of a potential status gain, specifically in those with low baseline dopamine availability. Future studies are required to replicate and test if this relation holds using larger samples.

It is important to note that, first, our sample only included men because the pharmacokinetics of the testosterone gel is only known in men. Although sex/gender differences for risk-taking are thought to be small (for review see Fisk et al., 2017), future research will be required to test whether our effects generalize to females. Second, to compare high and low-ranked individuals on risk preferences for identical risk choices and status outcomes (i.e. magnitudes, change in positions), high and low-ranked participants were placed in the middle of the new hierarchy containing only players of the same rank category. One possibility is that this induced an unstable hierarchy and a perceived small loss (gain) in status in the high (low) ranked participants. Consequently, testosterone may have reinforced risk-taking in the high-ranked participants to “redeem” their top position, but inhibited risk-taking in the low-ranked to secure the middle position and avoid further status loss, consistent with previous findings (Losecaat Vermeer et al., 2020).

Concluding, exogenous testosterone increased risk-taking for status, but not for options without direct status implications, in men with high but not low social status. These findings provide important

insight for research in the field and theoretical models of testosterone in risk-taking as a strategy for status-seeking.

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Open practices statement

The current design and analysis plans were not formally preregistered.

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527

Supplementary online material**Mood**

Drug administration did not influence self-reported mood, alertness and calmness (multidimensional mood questionnaire, Steyer, Schwenkmezger, Notz, & Eid, 1994) (all F values < 1). From pre- to post-administration alertness decreased ($F(1,164) = 9.77, p = .002$), calmness increased ($F(1,164) = 10.97, p = .001$), but participant's mood did not statistically change ($F(1,164) = 2.11, p = .148$).

Hormone analysis

Saliva samples analyzed for testosterone and hydrocortisone using liquid chromatography tandem mass spectrometry (LCMS/MS), with an Agilent 6460 with electrospray ionization in positive mode coupled to a 1290 UHPLC system. Collision energy was optimized for specific MRM transitions of Hydrocortisone (363.2/121.1 m/z ; 363.2/91.1 m/z), Testosterone (289.2/109.1; 289.2/97.1 m/z), 2,3,4- $^{13}C_3$ -Hydrocortisone (366.2/124 m/z) and 2,3,4- $^{13}C_3$ -Testosterone (292.2/100 m/z). Agilent Poroshell 120 EC-C18 was used for chromatographic separation under reversed phase conditions. For the internal standard preparation and internal standard mixture was prepared containing 2,3,4- $^{13}C_3$ -Hydrocortisone; 2,3,4- $^{13}C_3$ -Testosterone, 2,4,16,16,17- d_5 -17 β -Estradiol and 2,4,16,16- d_4 -Estron with a final concentration of 5ng/mL each. Samples were prepared by adding 100 μ L internal standards (5 ng/mL) to 500 μ L plasma or saliva and the steroids were extracted using 4 mL MTBE. After 10 minutes overhead shaking, samples were centrifuged for 5 minutes at 3000 rpm and the top MTBE layer was transferred to a test tube. MTBE was evaporated using a centrivap concentrator at 40°C (Labconco). The residual sample was then re-dissolved in methanol and analyzed by LC-MS/MS.

Genotyping

Buccal swabs were collected using sterile cotton swabs (Sarstedt AG, Germany) and stored at room temperature. DNA was extracted using the QIAamp DNA Mini kit (Qiagen, Hilden, Germany) following

the kit's protocol: "DNA Purification from Buccal Swabs" and eluted in a final volume of 50 µL of QIAamp buffer AE (Qiagen, Hilden, Germany). Human DNA was quantified using the Applied Biosystems (AB) 7500 real-time PCR system (Thermo Fisher Scientific, Waltham, MA) and the Quantifiler HP (Human Plus) Quantification Kit (AB) following manufacturer's recommendations. Both, androgen receptor (AR)CAGn and DAT1 VNTR were simultaneously co-amplified in a single tube reaction. Accordingly, template DNA (10 ng per sample) was subjected to 35 cycles of PCR amplification performed in a total volume of 25 µL consisting of 1 × GeneAmp PCR Buffer (AB), 0.25 mM each dNTP, 0.5 U AmpliTaq Gold Polymerase (AB) and target specific primers (including fluorescently-labeled forward primers, details are provided in Table S1). The following thermal protocol was applied using the Veriti 96-well thermal cycler (AB): 35 cycles at 95 °C for 30 seconds, 55 °C for 1 minute, and 72 °C for 1 minute. Before the first cycle, an initial denaturation (95 °C for 5 minutes) was included, and the last cycle was followed by a final extension step at 72 °C for 45 minutes. Aliquots of PCR products were diluted with Hi-Di formamide (AB), mixed with internal lane standard LIZ 600 v.2 (AB) and separated on the ABI 3500 Genetic Analyzer applying standard conditions. The number of repeats predicted by the GeneMapper ID-X software (AB) was in full agreement to the actual repeats determined by direct sequencing of PCR products using the BigDye Terminator Sequencing Kit v3.1 (AB) in selected DNA samples.

Table S1. Panel of loci and primer sets used for the typing of repeat length polymorphisms

Marker	Location ^a	Primer sequence 5'-3' ^b	Dye	Orientation	Conc.(nM) ^c
AR(CAG)n	chrX:67545237-67545434(+)	CGCGAAGTGATCCAGAACC	6-FAM	forward	200
		<u>GTTTCT</u> AGAACCATCCTCACCTGCT		reverse	
DAT1 VNTR	chr5:1393559-1394008(-)	TGTGGTGTAGGGAACGGCCTGAGA	6-FAM	forward	400
		TGTTGGTCTGCAGGCTGCCTGCAT		reverse	

^a Chromosome number and genomic location of targeted sequence (orientation provided in brackets) according to UCSC version hg38 (<http://genome.ucsc.edu/>)

^b The non-specific primer tail is underlined in Italics

^c The final primer concentrations in the reaction mix

Moderators

Baseline cortisol

The model included 158 participants, as some samples ($n=8$) were not analyzable due to low quality or too little saliva. As shown in Table S3, baseline cortisol did not interact with drug or rank. The effect of rank when gambling for money was marginally significant ($p=.061$). For status, the conditional effect of testosterone and rank remained significant ($p=.024$). Although a nonsignificant drug x rank x cortisol interaction for status, when applying a median split on cortisol the effect of testosterone in the high-ranked group appears to be more pronounced among low baseline cortisol individuals (Fig. S1).

Figure S1. Risk-taking for status outcomes, split by rank and baseline cortisol (based on median split). Every dot represents participants' mean. Error bars represent 95% CI. Asterisks indicate significant difference between drug (** $p < .01$).

Trait dominance

As shown in Table S3, trait dominance did not influence risk-taking for money nor for status, nor did it interact with drug or rank. The main effect of rank when gambling for money remained significant ($p=.027$). For status, the conditional effect of testosterone and rank reduced to marginal significance (drug x rank, $p=.056$).

CAG-repeat length

As shown in Table S3, CAG-repeat length did not influence risk-taking for money nor for status, nor did it interact with drug and rank. The main effect of rank when gambling for money ($p=.018$), and the drug x rank interaction for status ($p=.029$) remained significant.

DAT1 genotype

We included data from the 10/10 (testosterone $n=48$, placebo $n=46$) and 9/10-carriers (testosterone $n=29$, placebo $n=27$), as they represented the largest sample (total $n=150$), similar to other work (Eisenegger et al., 2013; Heinz, 2000). Sixteen participants not included consisted of 9/9-carriers (testosterone $n=3$, placebo $n=8$), 10/11-carriers (testosterone $n=2$, placebo $n=2$), and one 9/11-carrier (placebo).

As shown in Table S3, DAT1 genotype did not influence risk-taking for money nor for status, nor did it interact with drug and rank. The main effect of rank when gambling for money was marginal significant ($p=.051$).

For status, DAT1 interacted with drug to predict risk-taking ($p=.035$). Although not reaching statistical significance at post-hoc comparisons, decomposing the interaction suggest that 10/10-carriers with testosterone (vs. placebo) were almost twice more likely to take risk (OR = 1.97, SE=0.85, 95% CI = [0.85;4.59], $z=1.57$, $p=.116$), whereas 9/10-carriers with testosterone (vs. placebo) seem 56% less likely to take risk (OR=0.44, SE=0.25, 95% CI = [0.15; 1.32], $z = -1.47$, $p=.143$). Including DAT1 resulted in a marginal significant testosterone by rank effect ($p=.081$).

Importantly, when including baseline cortisol, dominance, CAG-repeat and DAT1 as covariates in one model, the conditional effect of testosterone and rank on risk-taking for status was significant (OR=0.67, 95% CI = [0.47,0.95], $z = -2.25$, $p=.024$), whereas the main effect of rank for monetary gambles seemed less robust ($z = -1.51$, $p = .130$). To note, this model could only be analyzed on a subset of the sample ($n=142$), which reduced statistical power.

623 Table S3. Results of generalized mixed effects models for Money and for Status, for each moderator

Baseline cortisol (n=158)	Risk-taking for money					Risk-taking for status				
<i>Predictors</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>
Intercept	0.27	0.27	0.16,0.45	-4.86	<.001	0.18	0.21	0.12,0.27	-8.09	<.001
Rank	0.65	0.23	0.41,1.02	-1.88	.061	0.88	0.17	0.63,1.22	-0.78	.433
Drug	0.82	0.23	0.52,1.29	-0.87	.387	1.14	0.17	0.82,1.58	0.76	.449
Cortisol log (t0)	1.23	0.36	0.60,2.51	0.56	.573	1.09	0.27	0.64,1.84	0.31	.757
Expected Value	7.62	0.15	5.72,10.15	13.88	<.001	1.16	0.01	1.14,1.19	13.62	<.001
Rank * Drug	1.22	0.23	0.78,1.92	0.87	.386	1.47	0.17	1.05,2.04	2.25	.024
Rank * Cortisol log (t0)	1.24	0.36	0.61,2.52	0.59	.555	1.25	0.26	0.74,2.10	0.84	.398
Drug * Cortisol log (t0)	0.60	0.36	0.30,1.22	-1.41	.159	0.94	0.27	0.55,1.59	-0.24	.810
Rank * Drug * Cortisol log (t0)	0.83	0.36	0.41,1.69	-0.50	.614	0.73	0.26	0.43,1.23	-1.18	.237
Marginal R ² / Conditional R ²	0.594 / 0.913					0.662 / 0.892				
Trait dominance (n=166)	Risk-taking for money					Risk-taking for status				
<i>Predictors</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>
Intercept	0.29	0.28	0.17,0.50	-4.45	<.001	0.18	0.22	0.12,0.28	-7.82	<.001
Rank	0.58	0.24	0.36,0.94	-2.22	.027	0.83	0.18	0.58,1.19	-1.02	.309
Drug	0.82	0.24	0.51,1.31	-0.84	.401	1.22	0.18	0.86,1.74	1.13	.259
Trait dominance	1.00	0.01	0.98,1.02	0.13	.899	1.00	0.01	0.99,1.01	0.08	.937
Expected Value	7.33	0.14	5.60,9.60	14.49	<.001	1.16	0.01	1.14,1.19	13.95	<.001
Rank * Drug	1.39	0.24	0.86,2.23	1.35	.177	1.41	0.18	0.99,2.01	1.91	.056
Rank * Trait dominance	1.00	0.01	0.98,1.02	-0.05	.959	0.99	0.01	0.98,1.01	-0.70	.487
Drug * Trait dominance	0.99	0.01	0.98,1.01	-0.63	.526	1.00	0.01	0.99,1.02	0.44	.660
Rank * Drug * Trait dominance	1.01	0.01	0.99,1.03	0.78	.434	0.99	0.01	0.98,1.01	-0.86	.387
Marginal R ² / Conditional R ²	0.592 / 0.910					0.661 / 0.893				
CAG-repeat polymorphism (n=166)	Risk-taking for money					Risk-taking for status				
<i>Predictors</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>
Intercept	0.29	0.26	0.17,0.48	-4.75	<.001	0.17	0.21	0.11,0.25	-8.54	<.001
Rank	0.59	0.22	0.38,0.91	-2.38	.018	0.85	0.17	0.61,1.18	-0.98	.326
Drug	0.89	0.22	0.58,1.37	-0.53	.595	1.14	0.16	0.83,1.58	0.81	.415
CAG-repeat	1.02	0.08	0.88,1.19	0.32	.748	0.99	0.06	0.88,1.10	-0.22	.827
Expected Value	7.38	0.14	5.63,9.68	14.44	<.001	1.16	0.01	1.14,1.19	13.96	<.001
Rank * Drug	1.27	0.22	0.83,1.96	1.10	.272	1.43	0.17	1.04,1.98	2.18	.029
Rank * CAG-repeat	0.91	0.08	0.78,1.05	-1.29	.199	0.91	0.06	0.82,1.02	-1.62	.104
Drug * CAG-repeat	1.11	0.08	0.96,1.29	1.42	.155	0.97	0.06	0.87,1.09	-0.50	.614
Rank * Drug * CAG-repeat	1.09	0.08	0.94,1.27	1.17	.242	1.05	0.06	0.94,1.17	0.83	.405
Marginal R ² / Conditional R ²	0.594 / 0.911					0.663 / 0.893				
DAT1 genotype (n=150)	Risk-taking for money					Risk-taking for status				
<i>Predictors</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>	<i>OR</i>	<i>SE</i>	<i>95% CI</i>	<i>z</i>	<i>p</i>
Intercept	0.31	0.28	0.18,0.54	-4.16	<.001	0.18	0.22	0.11,0.27	-7.85	<.001
Rank	0.62	0.24	0.38,1.00	-1.95	.051	0.82	0.18	0.57,1.16	-1.12	.262
Drug	0.80	0.24	0.50,1.29	-0.91	.365	0.96	0.18	0.68,1.36	-0.21	.835
DAT1	0.70	0.24	0.43,1.13	-1.46	.144	0.73	0.18	0.52,1.04	-1.76	.078
Expected Value	7.38	0.15	5.54,9.83	13.68	<.001	1.16	0.01	1.14,1.19	13.48	<.001
Rank * Drug	1.15	0.24	0.71,1.85	0.57	.569	1.36	0.18	0.96,1.93	1.75	.081
Rank * DAT1	1.03	0.24	0.64,1.66	0.13	.897	1.09	0.18	0.77,1.54	0.49	.623
Drug * DAT1	1.39	0.24	0.86,2.23	1.34	.179	1.46	0.18	1.03,2.06	2.11	.035
Rank * Drug * DAT1	1.44	0.24	0.89,2.32	1.50	.134	1.30	0.18	0.92,1.84	1.48	.138
Marginal R ² / Conditional R ²	0.596 / 0.910					0.671 / 0.893				

624 Note: zero-to-sum contrast were applied (i.e. effect coding). Testosterone and high rank were coded as 1.

625 Continuous variables Expected value, baseline cortisol, trait dominance and CAG-repeat length were centered at

626 0.

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