Slot machine gambling and testosterone: Evidence for a 'winner-loser' effect?

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Abstract

The "winner-loser effect" refers to a phenomenon in testosterone research, where the outcome of a social competition induces increases (wins) and/or decreases (losses) in testosterone levels. Here, we sought to test to what extent changes in testosterone occur in response to gambling behavior. More specifically, we hypothesized that the winner-loser effect would extend to slot machine gambling as a solitary (noncompetitive) gambling activity in players who 'anthropomorphized' the slot machine, thus treating the machine as a human opponent. Male participants ($n = 113$) were recruited into a quasi-experimental design involving 15 minutes of authentic slot machine gambling, incentivized by a $10 cash bonus for participants who finished in profit. In addition to salivary measures of testosterone, salivary cortisol and self-reported anthropomorphization of the slot machine were tested as potential moderators. Contrary to predictions, winning and losing slot machine sessions did not exert significant differential effects on testosterone, and this pattern was not moderated by cortisol levels or slot machine anthropomorphization. Exploratory analyses tested relationships between subjective gambling experiences in the sessions and testosterone change. Higher Positive Affect and Flow predicted greater testosterone declines from pre- to post-gambling. The testosterone results add to a growing literature on the boundary conditions of the winner-loser effect, and inform future studies on testosterone reactivity in relation to gambling and disordered gambling. The tendency to anthropomorphize slot machines is a neglected cognitive distortion in gambling that merits further study.

Keywords: Gambling, Slot machines, Testosterone, Winner-loser effect, Anthropomorphism

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Testosterone primarily functions as an androgenic hormone in the development and maintenance of the male sex organs and secondary sexual characteristics, but is actually produced in both men and women (Nelson, 2011). In both sexes, the adrenal glands produce small amounts of androgen hormones including testosterone, while the reproductive glands (ovaries in women and testes in men) produce much larger quantities. In women, the aromatase enzyme produced in the ovaries converts a large proportion of testosterone to estrogens (Nelson, 2011), causing average testosterone levels in women to be several times lower than those of men (e.g., Clifton et al., 2016). This disparity in testosterone is widely thought to underlie sex differences in behavioral tendencies, but such differences are likely multiply determined. As one relevant example in humans, risk aversion in adults appears to show associations that vary according to prenatal testosterone exposure, sex, and basal testosterone levels (Sapienza, Zingales, & Maestripieri, 2009). Testosterone also exerts downstream effects on dopamine (see Sinclair, Purves-Tyson, Allen, & Weickert, 2014), a neurotransmitter that is decisively involved in reward processing and central to contemporary models of problem gambling and addictions more broadly (Clark, 2014; Murch & Clark, 2016). This ability of testosterone to alter dopamine functioning implies a modulatory role in reward-related behavior (Macoveanu et al., 2016; Peper, Koolschijn, & Crone, 2013).

Testosterone is further implicated in modulating decision-making, in ways that are directly relevant to gambling behavior (see Stenstrom & Saad, 2011). For example, basal testosterone positively predicted risk-taking in experimental investments and in lottery games with real monetary rewards (Apicella et al., 2008; Sapienza et al., 2009) as well as poorer performance on the Iowa Gambling Task (Evans & Hampson, 2014; Stanton, Liening, &

Schultheiss, 2011). In female participants, sublingual testosterone administration impaired performance on the Iowa Gambling Task and on a poker simulation (van Honk et al., 2016; Van Honk et al., 2004). These laboratory studies have been corroborated by field work that has found morning testosterone levels to be predictive of subsequent daily profits in male financial traders, in measures taken on the trading floor (Coates & Herbert, 2008). Real-world gambling, in which individuals often make a succession of financial decisions on uncertain events, has received less attention regarding testosterone. Currently, only one study to our knowledge has directly assessed the relevance of basal testosterone levels to the pathophysiology of Gambling Disorder, a condition that is listed as a behavioral addiction in the *Diagnostic and Statistical Manual of Mental Disorders* (5th ed.; American Psychiatric Association, 2013). Blanco, Ibáñez, Blanco-Jerez, Baca-Garcia, and Sáiz-Ruiz, (2001) found no differences in basal (morning) testosterone levels in 29 males seeking treatment for Gambling Disorder, compared to a healthy, age-matched control group. However, that study did not examine the relevance of acute testosterone reactivity.

Acute testosterone fluctuations can result from the 'winner-loser effect,' a social endocrinological phenomenon in which the outcomes of a social competition can increase (in winners) and decrease (in losers) testosterone levels (Archer, 2006; Mazur & Booth, 1998). The effect has been elicited in both males and females, and across a variety of competitive contexts, including laboratory contests with controlled outcomes (e.g., Tetris, a reaction time task) and field studies using dyadic and team-based competitions (e.g., soccer, tennis singles, chess) (see Carré & Olmstead, 2015; Geniole, Bird, Ruddick, & Carré, 2017). At the same time, the winner-loser effect has failed to replicate in a proportion of studies (Carré & Olmstead, 2015) and a recent meta-analysis indicated heterogeneity in more than 2500 participants, with a small overall effect size ($d = .22$ for women, $d = .23$ for men) (Geniole et al., 2017). Importantly, psychological

variables substantially moderate how humans appraise competitive outcomes, which may explain the inconsistencies in these past observations (Salvador, 2012; Salvador & Costa, 2009). For example, greater attributions of outcomes to personal ability and effort (vs. external factors such as luck) positively predicted testosterone change following competitions (Salvador, Costa, Hidalgo, & González-Bono, 2017; Serrano, Salvador, González-Bono, Sanchís, & Suay, 2000). The direction of testosterone change can even be fully reversed if the wins and losses are surprising or ambiguous (Zilioli, Mehta, & Watson, 2014). Contextual elements also appear to play a role, as the closeness of outcomes has moderated testosterone fluctuation in a prior study (Wu, Eisenegger, Zilioli, Watson, & Clark, 2017). Interestingly, Stenstrom and Saad (2011) argue that Blanco et al.'s (2001) interpretation of basal levels in Gambling Disorder may be complicated by the presence of sustained financial losses, such that true levels in that study may have been underestimated as a direct result of the winner-loser effect.

The winner-loser effect may be relevant in gambling games, and in slot machine gambling specifically. In North America, Electronic Gaming Machines (an umbrella term that includes modern slot machines) generate the majority of gambling revenue (MacLaren, 2016) and are robustly linked with gambling pathology (Binde, Romild, & Volberg, 2017; Markham, Young, & Doran, 2016). As an ostensibly solitary experience, slot machine gambling includes contextual elements, such as salient cues for winning and losing money, which might cause transient fluctuations in testosterone that are relevant to changes in risk-taking (e.g., Apicella, Dreber, & Mollerstrom, 2014). To what extent should the winner-loser effect transfer to slot machine gambling? Using a coin toss game in which a $5 cash prize was offered if over 30 of 60 heads were thrown, winners experienced significant increases in testosterone and losers experienced decreases (McCaul, Gladue, & Joppa, 1992). This study is notable in transferring a

competition phenomenon to an outcome determined by chance. However, a small study by

(Mazur & Lamb, 1980) did not find a significant winner-loser effect with a laboratory lottery

game, in which a $100 prize was awarded randomly to 7 of 14 participants. Steiner, Barchard,

Meana, Hadi, and Gray (2010) assessed the winner-loser effect in poker competitions, as a social

mode of gambling. Among 32 men who competed in one-on-one matches, testosterone levels

increased in both winners and losers, between measures taken at baseline and 5 minutes after the

matches. To our knowledge, no other research has directly examined the winner-loser effect in

the context of gambling games.

In applying the winner-loser effect to solitary forms of gambling like slot machines, a

relevant source of individual differences may exist in the extent to which people humanize

inanimate objects, termed 'anthropomorphism' (Waytz, Cacioppo, & Epley, 2010). If this

tendency applies to slot machines, it could logically create a competitive experience from what is

objectively a solitary activity. Previous observations using the 'think-aloud' procedure describe

anthropomorphic comments in rationalizing gambling successes and failures (e.g., "this machine

doesn't like me," "I don't think it wants to pay out at all") (Delfabbro & Winefield, 2000;

Griffiths, 1994; Ladouceur, Gaboury, Dumont, & Rochette, 1988). Griffiths (1994) found that

these verbalizations made up a larger proportion of think-aloud statements in regular (vs.

nonregular) gamblers – although the infrequent gamblers nonetheless displayed meaningful

levels of anthropomorphism towards the games (see also Ladouceur et al., 1988). Recently, Riva,

Sacchi, and Brambilla (2015) tested directly whether anthropomorphic primes influenced

gambling on an internet slot machine game (see also Kim and McGill, 2011). Across four

experiments, the anthropomorphic prime led participants to place more bets (i.e., more spins),

and ultimately incurr more losses than the players who read a neutral description of the slot

machine. Riva et al.'s study is important in experimentally manipulating anthropomorphic thinking but does not establish whether individual differences in the natural tendency to anthropomorphize are relevant to gambling behavior.

In the current study, we recruited male novice gamblers to play a slot machine for 15 minutes, for the chance to win a $10 cash prize. We used an authentic slot machine situated within a laboratory environment, acknowledging Geniole et al.'s (2017) meta-analysis of the winner-loser effect showing stronger effects in field studies like sports venues ($d = .46$). It is not possible to specify the outcomes on real slot machines, and so this ecological validity trades off with the quasi-experimental nature of winning and losing. Furthermore, winners are inherently underrepresented in authentic slot machine play. Thus, we elected to test novice gamblers due to feasibility of recruiting a sufficient sample size to test the winner-loser effect, compared to the smaller recruitment numbers likely for a study in regular or problem gamblers (c.f. Blanco et al., 2001). In light of recent evidence that testosterone fluctuations are modulated by hypothalamic-adrenal axis functioning (Mehta & Prasad, 2015; Zilioli & Watson, 2012), we also examined cortisol levels. Considering these elements together, we formulated three predictions: (1) that winning or losing sessions in slot machine gambling would increase and decrease (respectively) salivary testosterone. We also predicted two moderation effects: (2) baseline salivary cortisol should moderate testosterone fluctuation by attenuating acute increases, and (3) anthropomorphization of the slot machine should moderate divergent effects of wins and losses on testosterone.

Methods

Participants

Male students ($N = 124$) were recruited through a combination of a psychology subject panel (for course credit) and a campus advertisement (reimbursed $15 per hour). Exclusion criteria were (i) aged under 19 years (the legal age for gambling in British Columbia), (ii) a score > 7 on the Problem Gambling Severity Index (PGSI; Ferris & Wynne, 2001), indicating a high risk for problem gambling, (iii) did not have normal or corrected-to-normal eyesight, (iv) use of medications that are known to affect hormone functioning, (v) smoked more than five cigarettes per day, or (vi) had health problems including oral bleeding or an endocrine disorder. These criteria resulted in 10 participants being excluded from the study. One participant was consistently noncompliant and was also omitted from analyses. Thus, our final sample comprised 113 participants (age $M = 21.03$, $SD = 2.45$). All participants provided written informed consent before commencement of the study procedure, which was reviewed and approved by the University of British Columbia Behavioral Research Ethics Board (H15-03434).

Modelling Winners and Losers

To produce a gambling scenario wherein participants could either win or lose a cash bonus, participants received a cash endowment to play a genuine "Dragon's Fire" slot machine (WMS Gaming Inc., Waukeagan, IL) in our Casino Lab. Following a 15-minute period of gambling, participants were awarded a cash bonus of $10 if their credit score exceeded their initial endowment. This binary prize structure was used to maximize the impact of a winner-loser effect in the context of a game with a continuous outcome. The return-to-player percentage of the slot machine was set to 87.1%, meaning that 12.9% of the amount bet would be lost over an infinite number of spins. Based on the return-to-player, it was expected that most participants

would experience a net loss during the gambling session, such that there would be an unbalanced number of winners and losers. To reduce volatility in the outcome across participants, we instructed participants to not alter the bet setting of the machine, which was set to place the minimum bet on the maximum number of paylines (40 cents bet per spin) - the preferred strategy among regular slot machine gamblers (Livingstone, Woolley, Zazryn, Bakacs, & Shami, 2008).

Procedure

      Pregambling phase. Following consent and the PGSI to ensure eligibility, the participant rinsed their mouth of any food residue that might interfere with hormone assays. Following this, a male research assistant led the participant to the Casino Lab containing four authentic slot machines. There, the participant completed questionnaire measures including the Gambling Competitiveness Scale (GCS; Parke, Griffiths, & Irwing, 2004). The experimenter attached some psychophysiological monitoring equipment (not reported here), and the participant was given 5 minutes alone to provide a baseline saliva sample (T1).

      Gambling phase. The participant was seated at the slot machine and received brief verbal directions for the gambling session while the experimenter loaded the cash into the machine ($40, 4000 credits in $n = 44$; $60, 6000 credits in $n = 69$[1]). Each participant was told that the slot machine was authentic, that they would play for a fixed period, and that any winnings above their initial endowment would be converted to a cash prize of $10. The participant then played the slot machine alone for exactly 15 minutes, based on testosterone time course data by Zilioli and Watson (2012, 2014), after which time the experimenter returned holding the cash prize. For participants who exceeded their starting credit amount, the experimenter emphatically awarded the prize, stating "you've won the 10 dollars". For

participants who finished in loss, the experimenter emphasized that the $10 prize had not been

achieved.

   Postgambling phase. Immediately following the gambling session, the participant

provided a state rating of the gambling session using the in-game version of the Game

Experience Questionnaire (GEQ; IJsselsteijn, de Kort, & Poels, 2013), as well as ratings of slot

machine anthropomorphization (Riva et al., 2015) and mood (PANAS-X; Watson & Clark,

1994). The second saliva sample (T2) was taken 15 minutes after the gambling session had

ended, and a third sample was taken 30 minutes after the session ended (T3). Each participant

completed a bio-demographic questionnaire (see below). Following completion of the study, the

participant was debriefed about the specific aims of the study and the workings of modern slot

machines.

Saliva Samples and Hormone Assays

   Saliva samples were used to derive estimates of unbound serum testosterone and cortisol.

One day prior to their study appointments, an email reminder was sent to participants instructing

them to refrain from flossing, exercising, and consuming alcohol on the day of the study, and to

avoid brushing their teeth, eating, or drinking (aside from water) 45 minutes before participation.

To reduce the influence of diurnal rhythms, all testing occurred between 13:00 h and 19:30 h

(Dabbs, 1990; Horrocks et al., 1990). Participants were directed to provide 2.0mL saliva samples

using the passive drool method into sterile polypropylene vials during 5-minute periods.

Following collection, saliva was immediately frozen and stored at a constant temperature of -

20°C until analysis. All samples were assayed in duplicates for testosterone and cortisol using

enzyme-linked immunosorbent assay kits (Salimetrics LLC, State College, PA). Plates were

prepared as per the manufacturer's instructions and read on an Envision 2105 Multimode Plate

Reader (PerkinElmer, Woodbridge, ON). Average intra-assay coefficients of variation were 4.14% for testosterone and 4.94% for cortisol. High and low control samples were assayed on each plate in quadruplicates and used to determine inter-assay coefficients of variation, which were 16.88% for testosterone and 6.48% for cortisol.

Hormone measurements were normally distributed for testosterone and positively skewed for cortisol, as has been seen in previous research. Cortisol values were normalized by applying a log (n + 1) transformation (Mehta, Welker, Zilioli, & Carré, 2015; Wu et al., 2017; Zilioli & Watson, 2012). For testosterone, four participants had one or more values that differed by more than three standard deviations from the timepoint mean, and were excluded (2.7% of samples). For cortisol, three participants had outlier values that were excluded on the same basis (1.5% of samples).

Covariates of Hormone Levels

The bio-demographic questionnaire was adapted from Schultheiss and Stanton (2009) assessing variables known to influence hormone levels, which we considered as potential covariates in analyses of the winner-loser effect: age, body mass index ($M = 23.80$, $SD = 5.27$), recent caffeine consumption (binarized to the last 12 hours, $n = 52$), regular cigarette smoking ($n = 5$), recreational drug use ($n = 22$), involvement in a serious, committed relationship with one individual ($n = 32$), and sexual activity during the previous 24 hours ($n = 10$). We additionally considered regularity of sexual behavior (the modal response was 2 to 4 times per week), time of day (Diver, Imtiaz, Ahmad, Vora, & Fraser, 2003), sleep schedule (modal preferred waking time was 9:05 am-10:30 am) and hours sleep ($M_{hours} = 7.25$, $SD = 1.50$) (Leproult, Copinschi, Buxton, & Van Cauter, 1997), physical fitness assessed by number of hours of physical activity per week (the modal response was greater than 4 hours) (Tremblay, Copeland, & Van Helder, 2004), and

sexual orientation (heterosexual status, yes *n* = 105, no *n* = 8) (van Anders & Watson, 2006), as possible covariates in our analyses.

Statistical Analyses

Testosterone and cortisol changes from pre- to post-gambling were analyzed using hierarchical linear modeling (HLM). This strategy allows repeated-measures data to be modeled as a function of variation at within- and between-person levels simultaneously, and to test whether variables of interest predict variation at these different levels (Goldstein, 2010; Raudenbush & Bryk, 2002; Singer & Willett, 2003). HLM is ideal for analysis of repeated measurements of hormone time-courses (Hruschka, Kohrt, & Worthman, 2005), offering several advantages over repeated-measures ANOVA: HLM does not require balanced data or that complete data be present in all participants (Goldstein, 2010), and HLM offers increased power due to its greater accuracy in modelling variance and covariance components (Gueorguieva & Krystal, 2004; Quené & Van Den Bergh, 2004).

To assess testosterone change from pre- to post-gambling, a level-1 model was constructed using the three testosterone measurements from each participant and corresponding saliva sampling times (coded as minutes from baseline: T1 = 0, T2 = 30, T3 = 45). Testosterone change trajectories for each participant were modeled linearly over time (as is recommended for short time periods; Raudenbush and Bryk, 2002), as a function of fixed effects (namely, our predictor variables at level 2) and random effects (the intercept and slopes of the individual change trajectories, which were assumed to vary across participants). Thus, testosterone change at level 1 was modeled as

$$(Testosterone)_{ti} = \pi_{0i} + \pi_{1i}(Time)_{ti} + e_{ti}, \tag{1}$$

wherein salivary testosterone at measurement t for person i is predicted by participants'

testosterone intercept, π_{0i}, and the slope of testosterone change over saliva sampling occasions

(reactivity), π_{1i}. The intercept in the linear model (π_{0i}) represents an estimate of salivary

testosterone at baseline, as the baseline saliva measurement (T1) was coded as 0. With no

additional predictors included at any other levels, this level-1 equation also served as an

'*unconditional*' model.

To test hypothesis 1, that the outcome of the gambling session would produce a winner-

loser effect on testosterone, level-2 equations for intercept and slope were introduced to the

model that each contained a dummy coded predictor variable representing the outcome (winners

= 1, losers = 0). From this, variance in intercepts and slopes of testosterone change at level 1

could be predicted by outcome of the gambling sessions using the following level-2 equations:

$$\pi_{0i} = \beta_{00} + \beta_{01}(Outcome)_i + r_{0i} \tag{2}$$

$$\pi_{1i} = \beta_{10} + \beta_{11}(Outcome)_i + r_{1i} \tag{3}$$

For hypotheses 2 (cortisol) and 3 (anthropomorphization), additional models were

constructed to test moderation effects, with baseline (T0) cortisol and anthropomorphization

(along with their corresponding Outcome interaction terms) added individually as predictors to

the level-2 equations for the test of hypothesis 1. Based on guidelines for multilevel models with

level-2 interaction terms (Enders & Tofighi, 2007), level-2 variables were grand-mean centered.

Bio-demographic covariates were considered for inclusion in these models based on a

procedure by Hackman et al. (2012): of the variables listed in the section on covariates, age

(slope), recent caffeine consumption (slope), and sexual orientation (intercept and slope)

produced both significant coefficients and improved model fit ($p < .10$), when tested individually

as level-2 predictors. When these three variables were included concurrently in the models,

caffeine consumption and sexual orientation were significant at $p < .05$ and thus were retained as covariates.

Twelve participants (11 winners and 1 loser) did not gamble continuously throughout the gambling session, either because they ran out of credits early (1 participant) or because they voluntarily elected to stop play before the experimenter returned. It is possible that these participants experienced some appraisals of their skill or strategy as contributing factors to their outcome, and these appraisals can influence the winner-loser effect (Gonzalez-Bono, Salvador, Ricarte, Serrano, & Arnedo, 2000; Salvador et al., 2017). Thus, an additional dummy variable was coded (0 = played full session, 1 = stopped early) and considered as another covariate in the bio-demographic models, but all ps > .43.

Estimation and testing of the hierarchical model parameters was carried out using HLM 7.03 (Raudenbush, Bryk, & Congdon, 2017) with full maximum likelihood estimation and robust standard errors. Descriptive analyses of the data and variable centering were performed using SPSS 24.0 (IBM, Armonk, NY) prior to import into the HLM software. For the hierarchical linear models, residuals at each level and Mahalanobis distance were calculated to investigate whether any outlying data points excessively influenced the models. Such cases were not found to unreasonably influence hypothesis test results, nor were any hypothesis tests in the primary models unduly affected by the either the inclusion of control variables or the exclusion of hormone outliers. Alpha was set at .05.

Results

Preliminary Analyses

The remaining credits on the slot machine (i.e., final score minus endowment) were normally distributed with a mean in the loss range ($M = -742.55$, $SD = 2308.58$), as expected

from the return-to-player ratio of the machine. There were 50 overall 'winners' (net credit $M =$

1311.28, $SD = 1552.10$) and 63 overall 'losers' (net credit $M = -2372.57$, $SD = 1273.60$). Winners

and losers did not differ significantly on bio-demographic variables (all $ps \geq .068$) or measures of

gambling behavior on the PGSI ($t(111) = 1.42$, $p = .16$) and GCS ($t(111) = 1.21$, $p = .23$).

Notably, winners and losers had comparable baseline levels of testosterone ($t(109) = .27$, $p = .79$)

and cortisol ($t(109) = .24$, $p = .81$). There was substantial variability in slot machine

anthropomorphization scores ($M = 1.96$, $SD = .79$), which were positively skewed with no

outliers. Winners and losers did not differ in the extent to which they anthropomorphized the slot

machine ($t(111) = .05$, $p = .96$). As expected, winning sessions were associated with greater

postgambling measures of Positive Affect ($t(111) = 2.79$, $p = .006$) and lower measures of

Negative Affect ($t(111) = -2.90$, $p = .005$), on the PANAS-X. On the GEQ, winners endorsed

greater feelings of Competence ($t(111) = 9.84$, $p < .001$) and lower Tension/Annoyance ($t(111) =$

-3.15, $p = .002$) during the slot machine sessions. Other GEQ subscales did not differ (Immersion

$t(111) = 1.63$, $p = .11$, Flow $t(111) = 1.05$, $p = .30$, Challenge $t(111) = .79$, $p = .43$).

Effects of Wins and Losses on Testosterone and Cortisol

Following exclusion of hormone outliers, there were 330 level-1 observations,

comprising complete salivary testosterone data for 109 participants, partial data for 3

participants, and no testosterone values for one participant. For cortisol, there were a total of 334

level-1 observations. One participant had insufficient level-2 data to be included in analyses of

hormone changes. Time of day was negatively correlated with baseline cortisol ($r(109) = -.318$, p

$< .001$), but was not correlated with baseline testosterone ($r(109) = -.113$, $p = .24$). Table 1 lists

descriptive statistics for testosterone and untransformed cortisol concentrations.

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The *unconditional* model for testosterone change from pre- to post-gambling showed a

significant negative slope (B = -.14, p = .008), indicating an overall testosterone decline during

the study. Within this model, there was sufficient variability in both testosterone intercept (σ_0^2 =

1812.30, p < .001) and slope (σ_1^2 = .21, p < .001) to allow prediction by person-level variables at

level 2 in subsequent models.

Gambling outcome and the significant control variables were added to the model to test

whether winners and losers diverged in testosterone reactivity (Table 2, "No Moderators"

model). Hypothesis 1 was not supported: the association between Outcome and testosterone

reactivity (slope) was not significant, (B = -.036, p = .73), indicating that testosterone change did

not differ in winners and losers (see Figure 1). In the equivalent model with cortisol as the

dependent variable, the *unconditional* model showed that participants' cortisol declined through

the session (B = -.0013, p < .001). When Outcome and significant control variables were added

to the model, there were no associations between Outcome and baseline cortisol (B = .0041, p =

.76) or cortisol reactivity (B = -.000036, p = .87).

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Testing Moderators of the Winner-Loser Effect

Cortisol. Hypothesis 2 predicted that baseline cortisol levels would moderate (attenuate)

changes in testosterone in response to winning, which would be expressed as a significant

association between the Cortisol $\times$ Outcome interaction term and the slope of testosterone

change. Since hypothesis 1 was not supported, hypothesis 2 was modified to include Cortisol and the Cortisol × Outcome interaction term, to determine whether wins and losses would generate divergent effects on testosterone after controlling for baseline cortisol (see Wu et al., 2017). Two additional participants lacked sufficient level-2 data (baseline cortisol) to be included in this model. As in previous studies (e.g., Mehta et al., 2015; Popma et al., 2007; Zilioli & Watson, 2012), baseline testosterone and cortisol were correlated ($r(107) = .35, p < .001$). The results of the HLM (Table 2, "Cortisol" model) reflect this as a significant estimate of the association between baseline cortisol and testosterone intercept ($B = 168.44, p < 0.001$). However, neither baseline cortisol ($B = -1.05, p = .077$) nor the Cortisol × Outcome interaction ($B = -.13, p = .91$) predicted testosterone change. The effect of Outcome on baseline testosterone ($B = 2.11, p = .78$) and reactivity ($B = -.052, p = .62$) remained nonsignificant. Thus, addition of cortisol to this model did not support hypothesis 2 that the winner-loser effect on testosterone would be moderated by cortisol.

Slot Machine Anthropomorphization. Hypothesis 3 posited a similar moderation of testosterone reactivity by the tendency to humanize the slot machine, and was tested by incorporating Anthropomorphization (and its interaction with Outcome) in place of the cortisol predictors in the previous model. Testosterone reactivity was neither associated with Anthropomorphization ($B = .0039, p = .95$) or the Anthropomorphization × Outcome interaction term ($B = .011, p = .94$) (Table 2, "Anthropomorphization" model). Baseline testosterone was significantly predicted by the Anthropomorphization × Outcome interaction term ($B = -27.93, p = .005$), but the lower-order effect of Anthropomorphization was not significant ($B = -2.31, p = .65$). To explore this interaction, we re-centered Outcome in two subsequent models, to derive Anthropomorphization coefficient estimates separately for winners and losers. With winners

coded 0 and losers coded 1 (thereby representing coefficient estimates for the winners), higher slot machine anthropomorphization was associated with lower testosterone at baseline (B = -17.66, p = .008), while the association between Anthropomorphization and testosterone change remained nonsignificant (B = .010, p = .93). Conversely, in losers (as coded 0), Anthropomorphization was neither associated with baseline testosterone (B = 10.27, p = .17) or testosterone change (B = -.0013, p = .97). Thus, hypothesis 3 was also not supported: a tendency to think about the slot machine as more human-like did not predict testosterone fluctuation, and thus did not moderate any winner-loser effect.

---------- INSERT TABLE 2 ABOUT HERE ----------

Effects of Subjective Gambling Experiences on Hormone Fluctuation

As winners and losers differed substantially in their gambling experiences on the GEQ and postgambling affect (PANAS-X), a further set of post-hoc models were run to explore whether those ratings predicted testosterone fluctuation. Each rating scale and the interactions with Outcome were entered as fixed effects for intercept and slope, in separate models. Hormone covariates were retained from the previous models. With respect to testosterone reactivity, higher ratings of Positive Affect on the PANAS-X (B = -.013, p = .035) and GEQ Flow (B = -.089, p = .035) following slot machine play predicted steeper testosterone declines. All other slope coefficient estimates for rating scale, Outcome, and Outcome interaction terms were not significant (all ps ≥ .088). However, in models for GEQ Competence, Immersion, and Tension, by including outlying hormone values and/or excluding the hormone covariates, one or more

significance tests for fixed effects in each model were influenced. Thus, caution is warranted in interpreting these models.

Discussion

In the present study, we sought to test whether a winner-loser effect could be elicited in slot machine gambling as a solitary competitive experience. Our primary motivation was to indirectly assess the potential of gambling wins and losses to elicit testosterone changes relevant to subsequent gambling tendencies, such as risk taking (Apicella et al., 2014). We predicted that increases and decreases in testosterone would occur in response to wins and losses, respectively. In a moderately large group ($n = 113$) of young adult males, winning or losing outcomes from playing an authentic slot machine for 15 minutes had no differential influence on testosterone levels. Rather, testosterone declined for all participants during this period, and did so equally for winners and losers. Several potential confounds of testosterone fluctuation (e.g., trait differences, subjective experience, cortisol state, etc.) were monitored and/or controlled for, and the data were modeled using a highly sensitive HLM strategy that is well suited for analysis of hormone time-courses. The winner-loser effect was not observed either with or without controlling for cortisol status, with the former model motivated by the dual-hormone hypothesis (Mehta & Josephs, 2010). We investigated whether the individual differences in the tendency to humanize the slot machine (anthropomorphization) explained variability in testosterone reactivity to winning or losing the gambling session. Clear variation was observed in slot machine anthropomorphization, but this was not associated with testosterone reactivity. On the other hand, anthropomorphization of the slot machine was predicted by an outcome-moderated association with baseline testosterone. This result was driven by winners, among whom higher baseline testosterone was associated with lower slot machine anthropomorphization scores.

Overall, we find no evidence to support the hypothesis that humanizing slot machines is sufficient to create a competitive experience as manifested in testosterone change.

The parsimonious interpretation of our data is that winning (vs. losing) outcomes in the context of slot machine gambling are not sufficient to trigger testosterone changes. In support of this conclusion, our methodological approach appeared sufficient to identify Positive Affect and Flow as related to testosterone change. Indeed, these two variables are often positively associated during gambling (Murch, Chu, & Clark, 2017; see also Dixon et al., 2018), and we observed a similar correlation between Flow and postgambling ratings of Positive Affect on the PANAS-X ($r(111) = .42$, $p < .001$). Thus, their co-occurrence as predictors of testosterone decline mitigates against the possibility of a Type I error. Our null finding for the basic winner-loser effect is in line with a previous study monitoring testosterone during a poker competition (Steiner et al., 2010). The disparity with the conventional effect in social competitions may be reconciled in several ways. One is the absence of human opponents within our slot machine design. Although the winner-loser effect has been elicited in sports fans watching matches (Bernhardt, Dabbs, Fielden, & Lutter, 1998), and in sports players watching personal videos of their own previous achievements (Carré & Putnam, 2010), in both cases, these procedures involve witnessing a traditional interindividual competition, which may generate a vicarious or imagined sense of social involvement.

Second, subjective appraisals of involvement and one's performance as having contributed to the win are known mediators of the winner-loser effect (Gonzalez-Bono, Salvador, Serrano, & Ricarte, 1999; van Anders & Watson, 2007), and these could have been diminished by the restrictions we placed on participants' betting strategies and reward possibilities. We approached this issue via the role of anthropomorphization, which did not reliably predict

testosterone reactivity, suggesting that perceived intentionality may not necessitate an actual sense of competition against the machine. However, anthropomorphism is a complex construct that may engender other perceptions in gambling games besides competition, such as companionship (Griffiths, 1993), which would qualify as anthropomorphism but not predict moderation of the winner-loser effect. Another key factor here is that the random nature of slot machine payouts may generally limit the ability of slot machine gambling to elicit subjective experiences of involvement or influential ability in wins and losses. Pressing the spin button on a slot machine may not be sufficient to provoke these experiences. As mentioned previously, McCaul et al. (1992) provide the only evidence that chance-based, noncompetitive outcomes can elicit a winner-loser effect, but in their study, the experimenter performed the coin tosses that may have been sufficient to engender a competitive experience. In the present study, the experimenter was absent during the period of slot machine play.

Our results may also be interpreted considering involvement of a *reverse* winner-loser effect. Some studies have demonstrated post-competition increases in testosterone levels of losers but not winners (e.g., Oliveira et al., 2013, 2014), and Zilioli et al. (2014) showed that a similarly reversed winner-loser effect was more pronounced in participants who experienced surprise after a competition designed to produce ambiguous wins and losses. More recently, Wu et al. (2017) showed that cortisol levels and the closeness (clear versus narrow) of Tetris competition outcomes moderated testosterone fluctuation. Individuals who had higher baseline cortisol and won the Tetris competition narrowly, experienced the greatest change in testosterone, which *decreased* in line with a reverse winner-loser effect. In the context of the present results, the random nature of slot machine wins and losses may enhance the uncertainty about winning and losing during the gambling session. Furthermore, although the number of

winners and losers were close in proportion (63 losers, 50 winners), net slot machine scores had a mean in the loss range (-742.55 credits), with an important consequence that wins occurred by a narrower margin than losses. This ambiguity could explain why testosterone was seen to decline on average. Our laboratory slot machines include 'Loss Disguised as Win' events on which only a proportion of the original bet is won (a net loss) but rewarding feedback (jingles, visual animations) are presented (Dixon, Harrigan, Sandhu, Collins, & Fugelsang, 2010). These events may have further promoted a sense of ambiguity in loss outcomes. Losses Disguised as Wins have been shown to produce similar elevations in physiological arousal as true wins and overestimations of the number of wins achieved, in novice gamblers (Dixon et al., 2010; Jensen et al., 2013). Thus, it is possible that for losing participants, gambling sessions may have still been laden with enough winning stimuli, such as bonus rounds, near misses, and Losses Disguised as Wins, that a cumulative perception of winning was created. These game features, especially bonus rounds, may have also contributed to feelings of surprise while gambling or toward the final outcomes, which could contribute to a reverse winner-loser effect as in the study by Zilioli et al. (2014).

To what extent could our design have missed an effect of slot machine gambling on testosterone (i.e. a Type II error)? First, we modeled testosterone change linearly in our analyses, but it is plausible that testosterone trajectories for winners and losers could diverge on a nonlinear basis. Our modelling decisions were informed by the principle that at least four repeated measurements are a mathematical requirement for interpretable quadratic growth models (Maxwell, Delaney, & Kelley, 2018). Visual inspection of our data does indicate quadratic trends in testosterone change, and so future studies may fruitfully include additional testosterone timepoints in order to model nonlinearity. Second, our incentivization procedure

may have been insufficient to elicit a winner-loser effect. We binarized the bonus prize to

increase the salience of wins and losses. However, this win-loss scheme, together with the

provision of a sum of money to gamble with, produced indeterminate positive expected value

outcomes, which may have influenced the participant's perception of the outcome. Our

incentivization procedure also prevented participants from receiving full jackpots, which when

achieved, may have signaled the $10 prize as a net *loss* of money. Similarly, although the

participant's initial endowment was displayed by the machine in credits, to distance this number

from the dollar amount, winners may have still recognized that the cash bonus was a fraction of

their starting amount.

Several limitations in this study are worthy of discussion. First, our participants were

novice, nonproblem gamblers, and it is an empirical question whether experienced slot machine

gamblers are more reactive to session outcomes; 'loss chasing' is a cardinal symptom of

disordered gambling that implies an emotional response to losing, but recent work (Murch et al.,

2017) also highlights a state of immersion in the game, where players could lose track of their

financial outcomes. Furthermore, pathological gamblers report greater competitiveness than

nonpathological gamblers (Parke et al., 2004), and hence regular and problem gamblers might

experience slot machine gambling as a more competitive activity than our participants did. Riva

et al. (2015) found that regular gamblers self-reported a greater tendency to anthropomorphize

slot machines, compared to nonregular gamblers (see also Griffiths, 1994). Differences such as

these may inform a higher likelihood of finding, for instance, a moderation effect of

anthropomorphization in experienced gamblers. A winner-loser effect may be also more evident

in experienced gamblers due to increased age, which is a known moderator (Geniole et al.,

2017). Second, the use of authentic slot machines as a mode of generating gambling wins and

losses created a tradeoff between enhanced ecological validity and reduced experimental control. The quasi-experimental determination of wins and losses provided a realistic experience (c.f. Geniole et al., 2017), but heterogeneity within winning and losing sessions may have influenced winner-loser effects. As is expected in authentic slot machine gambling, all participants experienced random sequences of winning and losing trials, but this may have produced variable experiences among winning and losing subgroups in the number and type of game features experienced (e.g., bonus rounds, near misses). Furthermore, participants may have spent varied proportions of time above or below the winning credit threshold, and they may have achieved winning status only near the end of their sessions. Thus, the trajectory of participants' credit balance may not be reflected in the net outcome used to determine who won and lost, and it is possible that testosterone slopes were influenced by different patterns in balance change around the winning threshold. Our approach to balancing these tradeoffs was to limit participant betting to one denomination (40 cents), thereby reducing variability in experiences introduced by participants' ability to vary their bet sizes. However, this may have promoted gambling behaviors that are inconsistent with authentic gambling settings. Because of these design features, caution is warranted in generalizing the current findings to other gambling settings.

In conclusion, this study examined how slot machine gambling affects testosterone, a hormone that is widely implicated in both risk-taking and outcome processing. Although minimal associations were observed between gambling outcomes and hormone fluctuation, this study adds to a growing body of research highlighting boundary conditions to the winner-loser effect. Furthermore, extensions of this research may yet uncover specific gambling conditions that elicit testosterone changes, which may be relevant to predicting changes in gambling

strategy. Further research is recommended to elucidate the bidirectional relationships between

testosterone and aspects of slot machine gambling.

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Footnote

[1] The first 44 participants received 4000 credits. This endowment was switched to 6000 credits to accommodate a minority of participants for whom 4000 credits did not last the entire gambling session. Participants who received 6000 credits were .97 years older on average, $t(111)$ = 2.09, p = .04. No differences between the two groups that received each amount were observed on gambling experiences (Game Experience Questionnaire) or any other variables of interest in this study (all ps > .09). Additionally, there was a comparable proportion of winners and losers who received each amount of credits, $\chi^2(1, n = 113) = 0.33, p = .57$.

Table 1

Descriptive Statistics for Testosterone and Cortisol Measurements in Winners and Losers

	All participants		Winners		Losers	
	M (SEM)	SD	M (SEM)	SD	M (SEM)	SD
Pre-gambling testosterone (pg/mL)	141.77 (4.06)	42.81	140.55 (6.16)	43.58	142.77 (5.44)	42.51
Post-gambling testosterone, +15 min (pg/mL)	133.63 (3.27)	34.19	134.21 (4.98)	34.87	133.16 (4.38)	33.91
Post-gambling testosterone, +30 min (pg/mL)	135.66 (3.43)	35.99	133.97 (4.99)	34.94	137.01 (4.74)	37.05
Pre-gambling cortisol (µg/dL)*	.1851 (.0087)	.0920	.1834 (.0142)	.0997	.1865 (.0110)	.0862
Post-gambling cortisol, +15min (µg/dL)*	.1331 (.0057)	.0597	.1347 (.0092)	.0648	.1318 (.0071)	.0558
Post-gambling cortisol, +30 min (µg/dL)*	.1205 (.0049)	.0515	.1222 (.0080)	.0567	.1190 (.0060)	.0473

Note. * Untransformed hormone measurements were used to calculate the mean, standard error of the mean (SEM), and standard deviation.

Table 2

Effects of Gambling Outcome, Cortisol, and Slot Machine Anthropomorphization, on Testosterone

Fixed Effects	1. No Moderators			2. Baseline Cortisol			3. Anthropomorphization		
	Estimate	SE	*p*	Estimate	SE	*p*	Estimate	SE	*p*
Pre-gambling testosterone, π_{0i}									
Intercept	142.14	4.10	< .001	141.36	3.71	< .001	142.25	3.97	<.001
Cortisol				168.44	42.76	< .001			
Anthropomorphization							-2.31	5.01	.65
Gambling outcome	-1.89	8.19	.82	2.11	7.50	.78	-1.88	7.88	.81
Cortisol × Gambling outcome				10.90	82.17	.90			
Anthropomorphization × Gambling outcome							-27.93	9.83	.005
Sexual orientation	-30.60	9.39	.001	-20.85	10.06	.041	-29.29	9.46	.003
Testosterone reactivity, π_{1i}									
Intercept	-.14	.049	.004	-.13	.049	.008	-.14	.049	.004
Cortisol				-1.05	.59	.077			
Anthropomorphization							.0039	.068	.95
Gambling outcome	-.036	.10	.73	-.052	.10	.62	-.035	.10	.74
Cortisol × Gambling outcome				-.13	1.18	.91			
Anthropomorphization × Gambling outcome							.011	.14	.94
Caffeine within past 12 hours	.22	.087	.014	.22	.089	.014	.22	.085	.012
Sexual orientation	.54	.15	< .001	.47	.15	.003	.54	.15	< .001
Random Effects	Estimate		*p*	Estimate		*p*	Estimate		*p*
Level 1									
Within-person (σ_e^2)	88.95			89.64			88.93		
Level 2									
Pre-gambling testosterone (σ_0^2)	1774.52		< .001	1416.40		< .001	1654.03		< .001
Testosterone reactivity (σ_1^2)	.18		< .001	.17		< .001	.18		< .001
Covariance, σ_1^0	-9.50			-7.53			-9.44		

Figure 1. No-moderators model of linear trajectories for testosterone in winners (solid line, n = 50) and losers (dashed line, n = 61), from baseline to post-gambling (+30 minutes). Circles and error bars indicate means and standard error of the observed data. Winners and losers did not differ with regard to baselines testosterone levels ($t(109)$ = .27, p = .79), or testosterone reactivity over the course of the experiment (B = -.036, p = .73).