SLOT MACHINE GAMBLING AND TESTOSTERONE:

EVIDENCE FOR A 'WINNER-LOSER' EFFECT?

by

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Abstract

Testosterone can be seen to modulate cognition and behaviour in many ways. One likely effect is to promote risky decision-making. According to a phenomenon termed the "winner-loser effect," testosterone has also been observed to fluctuate in response to winning or losing competitions with others, with wins causing increases and losses causing decreases. Surprisingly, few studies have investigated the effects of gambling on testosterone levels, or whether individual differences in testosterone are related to risky gambling strategies. More specifically, the winner-loser effect may extend to slot machine gambling as a solitary gambling activity if players tend to 'anthropomorphize' slot machines, i.e. to treat the machine as a human agent with intentions and feelings. This study used a quasi-experimental design to measure testosterone fluctuations in response to winning and losing during a period of authentic slot machine gambling. Cortisol and anthropomorphism were investigated as potential moderators of a winner-loser effect on testosterone. Male participants ($n = 120$) provided saliva samples before and after a period of gambling on an authentic slot machine. Participants also provided measures of real-world gambling involvement, subjective experiences during slot machine play, and anthropomorphic tendencies. Contrary to predictions, winning and losing were not significantly associated with divergent effects on testosterone, even after considering cortisol levels and anthropomorphization of the slot machine. An exploratory analysis supported a link between positive affect (higher in winners) and decreases in testosterone, which suggested that the winner-loser effect may be reversed in slot machine gambling. In addition, baseline testosterone predicted a slower rate of gambling. The results of this study add to a growing literature on the

boundary conditions of the winner-loser effect, which inform future examinations of the role of

testosterone in gambling behaviour.

Lay Summary

The "winner-loser effect" is a phenomenon in which winning a competition increases testosterone levels, and competitive losses decrease testosterone. Given testosterone's established associations with risky behaviour, it is surprising that few studies have investigated the effects of gambling on testosterone levels, or whether differences in testosterone predict riskier gambling. The goals of this research were to clarify the roles of testosterone in slot machine gambling, as the most prevalent mode of gambling in casinos. Contrary to predictions, a winner-loser effect was not observed, even after considering several putative moderators of this effect. An exploratory analysis suggested that the effects of slot machine wins and losses on testosterone could even be reversed as a function of positive mood. Additionally, baseline testosterone predicted a less-risky gambling strategy. By helping to elucidate the conditions that elicit the winner-loser effect, this work informs future inquiries into the complex relationships between testosterone and gambling.

Preface

This thesis is an original work by the author, Mario Ferrari, whom carried out the design, testing, hormone and statistical analyses, and writing, under the supervision of Dr. Luke Clark. Study apparatus for recording slot machine spin presses was developed by W. Spencer Murch. Dr. Eve Limbrick-Oldfield provided statistical consultation for hierarchical linear modeling. Training on immunoassay procedures for measuring steroid hormones was provided by Dr. Michael Chen. Data collection was carried out with the assistance of Chris de Groot, Amit Chandna, and Cameron Drury.

Data were collected at the Centre for Gambling Research at the University of British Columbia. Hormone measurements were performed at the British Columbia Institute of Technology Natural Health and Food Products Research Group Laboratory.

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Chapter 1: Introduction

Indirect evidence implicates testosterone, a sex steroid hormone regulated by the hypothalamic-pituitary-gonadal axis, as a relevant factor in the etiology and/or maintenance of problem gambling and gambling-related decision-making. The dispositional characteristics of individuals with gambling problems have been found to include increased impulsivity, competitiveness, and antisocial behaviour (Parke, Griffiths, & Irwing, 2004; Steel & Blaszczynski, 1998). In the general population, these characteristics have each been associated with basal levels or transient fluctuations of circulating testosterone in humans (for a review, see Carré & Olmstead, 2015; examples include Gunilla Stålenheim, Eriksson, Von Knorring, & Wide, 1998; Takahashi, Sakaguchi, Oki, Homma, & Hasegawa, 2006). At the same time, research conducted over the past two decades convincingly implicates a variety of hormones in the modulation of human and non-human cognition, behaviour, and neural morphology (for reviews, see Frick, 2012; Galea et al., 2008). It may be surprising, then, that hormones, especially testosterone, have received so little attention from researchers seeking to investigate cognition and behaviour in realistic gambling games and environments.

Of the few of studies that have directly investigated testosterone in relation to gambling behaviour, the results have been limited by several methodological shortcomings, including reliance on basal testosterone levels rather than testosterone reactivity, the use of ecologically unrealistic gambling tasks, and inferences derived from the use of supraphysiological (i.e., much greater than naturally occurring levels) testosterone doses. For the most part, these studies have focused quite specifically on simplified poker competitions in a laboratory setting. For example, van Honk et al. (2016) investigated the effects of sublingual testosterone administration on gambling behaviour in women. During a two-person poker match, females in the testosterone

condition bluffed less often and less randomly compared to females in the placebo condition. In poker, bluffing is generally considered a beneficial strategy. So in other words, these authors found that artificial increases in testosterone adversely affected strategy selection in women, albeit in a specific form of gambling that has both a strong social and skillful component (Bolen & Boyd, 1968; DeDonno & Detterman, 2008; Hannum & Cabot, 2009). The uniqueness of poker in this regard highlights the problems with generalizing results from poker research to other gambling modalities. In North America, most casino patrons participate in and prefer solitary forms of gambling, specifically slot machines (American Gaming Association, 2013). Slot machines (more broadly termed Electronic Gambling Machines, EGMs) are also widely accepted as the form of gambling that is most closely associated with gambling problems (Dowling, Smith, & Thomas, 2005; Navas et al., 2017; Storer, Abbott, & Stubbs, 2009). Consequently, prior emphasis on poker in gambling endocrinology research may have limited relevance to individuals with gambling problems.

This thesis aims to further elucidate the role of testosterone in problem gambling behaviour by examining some rudimentary relationships between testosterone levels and relevant aspects of slot machine gambling. First, in Section 1.1, definitions of gambling and problem gambling are explained. Subsequent sections of this introduction will explore several lines of research that strengthen the link between testosterone and slot machine gambling. Section 1.2 provides a brief introduction to how testosterone affects neural processes, and highlights evidence supporting a relationship between testosterone and risky decision-making, along with gambling-relevant research examples. Section 1.3 introduces a phenomenon known as the 'winner-loser effect,' which links competition outcomes (i.e., wins and losses) with divergent effects on testosterone, such that testosterone levels tend to increase in winners (victories) and

decrease in losers (defeats). In addition to reviewing the conceptual underpinnings of this phenomenon, section 1.3 also reviews previous studies on the winner-loser effect that are relevant to the consideration of gambling behaviour, in addition to the specific conditions that elicit the effect most strongly. Section 1.4 will consider how individual differences in the tendency to anthropomorphize (i.e. humanize) slot machines, as well as other inanimate objects, may contribute to gambling distortions and testosterone involvement.

The importance of testosterone fluctuation within gambling contexts is multifaceted. The possibility that changes in testosterone may result predictably from slot machine wins and losses, and depend on one's tendency to anthropomorphize, highlights the need to study these phenomena jointly. This need also arises from potential consequences of testosterone changes, which may, through modulation of individuals' decision-making, foster riskier gambling decisions, and consequently, problem gambling.

1.1   Gambling, Gambling Disorder, and Problem Gambling

Common understanding of what constitutes gambling behaviour is generally in keeping with dictionary definitions, which describe gambling as "to stake something on a contingency" or "to bet on an uncertain outcome" (Merriam-Webster Online, 2017). By these definitions, gambling comprises a broad assortment of behaviours, many of which are prevalent across cultures, now and in previous time periods (Binde, 2005a, 2005b). Millennia-old dice and gaming boards have been unearthed in Egypt, India, Greece, and Iraq, dating as far back as 2000 BC, and Chinese gambling involvement has been dated as far back as 4000 years ago (McMillen, 2005). In the last century, commercialized gambling has seen increasing liberalization in many

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countries, marking a significant shift in conceptualization of gambling from a morally dubious activity to a mainstream form of recreation and entertainment (Banks, 2017).

In one of the first attempts to call attention to the ubiquity of gambling worldwide, Bolen and Boyd (1968) produced a formal definition of gambling that has since been used by many researchers: gambling is the wagering of something of value on an outcome determined, to varying amounts, by chance. From this, the gambler is anyone who wagers something of value upon a chance-based outcome of an event or game. These definitions nevertheless struggle to separate gambling from other risky decisions that affect most people, such as deciding to propose marriage, applying to university, or to buy real estate. Bolen and Boyd preferred to define gambling broadly in order to highlight and examine a universality of gambling behaviour, independent of the relevance of gambling to addiction or mental health.

Within this thesis, and typically within the gambling literature, use of the term 'gambling' is constrained further to maintain a frame of reference to behaviour that is relevant to the symptoms and signs of problem gambling. In this way, gambling is defined as only those activities in which the wagers and prizes involve money, given that monetary consequences are central in several of the problem gambling criteria (see below). Moreover, gambling is restricted by legal definitions as activities that are legalized and regulated as such by the Canadian Government, which serves to exclude some relevant behaviours (e.g., investing in the stock market) that could fall within the scope of standard psychological definitions. In Canada, these forms of gambling currently include monetary wagers made on games in casinos or on the internet, on sports matches, at race tracks, on electronic gambling machines, or by means of government operated lotteries, or other licensed raffles and activities, for which monetary prizes are awarded. Although the legality of certain gambling modalities varies by country and

state/province, the government-recognized forms of gambling within Canada are similar to those of other Western jurisdictions.

Among Canadians of legal age for gambling, the 12-month prevalence of participation in one or more legalized gambling activities is 75% (Cox, Yu, Afifi, & Ladouceur, 2005; Williams, Volberg, & Stevens, 2012). Similar estimates have been obtained for other Western countries (e.g., Abbott, Volberg, & Rönnberg, 2004; Wardle et al., 2007; Welte, Barnes, Wieczorek, Tidwell, & Parker, 2002). Even though most of these gamblers do not experience negative repercussions from their gambling, for some, gambling becomes a source of problems in personal (i.e., mental and physical health), social, or occupational functioning (Shaffer & Martin, 2011). For this subset of individuals, several terminological distinctions have been made and used within the gambling literature.

The American Psychiatric Association's current classification, called 'Gambling Disorder,' represents a recent reconceptualization of the previous term, 'Pathological Gambling' (American Psychiatric Association, 1980, 2013). With this change, the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) requires only four (instead of five) of the original criteria for Pathological Gambling be met for a diagnosis of Gambling Disorder. The DSM-5 has also repositioned Gambling Disorder alongside the substance use disorders, reflecting a burgeoning literature that supports the substantial behavioural and neurobiological similarities between individuals with gambling problems and substance addictions (Grant, Brewer, & Potenza, 2006; Potenza, 2006). According to the DSM-5, Gambling Disorder constitutes four or more of nine criteria (see Table 1.1) occurring within a 12-month period.

1. Needs to gamble with increasing amounts of money in order to achieve the desired excitement.
2. Is restless or irritable when attempting to cut down or stop gambling.
3. Has made repeated unsuccessful efforts to control, cut back, or stop gambling.
4. Is often preoccupied with gambling (e.g., having persistent thoughts of reliving past gambling experiences, handicapping or planning the next venture, thinking of ways to get money with which to gamble).
5. Often gambles when feeling distressed (e.g., helpless, guilty, anxious, depressed).
6. After losing money gambling, often returns another day to get even ("chasing" one's losses).
7. Lies to conceal the extent of involvement with gambling.
8. Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling.
9. Relies on others to provide money to relieve desperate financial situations caused by gambling.

Table 1.1 DSM-5 criteria for Gambling Disorder

Others have taken a more inclusive approach to defining disordered gambling, recognizing that gambling involvement that does not qualify a person for a DSM diagnosis can still result in significant levels of harm (Toce-Gerstein, Gerstein, & Volberg, 2003). This approach to classifying harm from gambling is often reflected by the term 'problem gambling,' which captures a broader range of gambling behaviour severity, spanning from having moderate difficulties with gambling, to meeting full clinical criteria for gambling addiction (Cowlishaw et al., 2012; Hodgins, Stea, & Grant, 2011). Accordingly, 'problem gambling' has been used widely and sometimes interchangeably with clinical distinctions of gambling involvement. Much like the DSM-5's Gambling Disorder, 'problem gambling' is conferred by both subjective symptoms (e.g., feelings of stress or anxiety over one's gambling) and objective signs (e.g., chasing losses with additional bets, selling belongings, or taking loans to sustain gambling

behaviour), and is often measured using scales that are either directly modeled on DSM criteria, or have good sensitivity and specificity when compared to DSM cutoffs (Ferris & Wynne, 2001; Lesieur & Blume, 1987).

Within this thesis, the broader and more inclusive designation, 'problem gambling,' will be used consistently to refer to signs and symptoms captured by either problem gambling or DSM classifications of disordered gambling, despite that problem gambling may not reflect the exact nomenclature of gambling involvement used in some of the studies that are reviewed.

1.2   Testosterone, Decision-Making, and Gambling Behaviour

The psychological and physiological processes underlying decision-making have received much attention in gambling and behavioural endocrinology research. Although risky decisions are a central and ubiquitous feature of gambling, the relevance of hormone functioning to decision-making has received less attention. Nonetheless, several hormones are implicated in decision-making. For example, recent work has found associations between estradiol and progesterone, and social allocation of money (Anderl, Hahn, Klotz, & Rutter, 2015), and that oxytocin administration has been seen to modulate individuals' selection of allies (De Dreu, Greer, Handgraaf, Shalvi, & Van Kleef, 2012). Steroid hormones, which include sex hormones like testosterone, have been hypothesized to regulate financial decision-making (Boksem et al., 2013; Coates & Herbert, 2008). Specifically, a burgeoning literature supports testosterone's ability to influence decision-making, in ways that are directly relevant to gambling behaviour. Despite this, little regard has been given to the study of testosterone levels in realistic gambling situations.

Testosterone is one of many hormones secreted by the endocrine system that promote changes in neural activity that affect both sexes. During intrauterine development, the presence of testosterone in males is largely responsible for sexual differentiation of the reproductive organs, in addition to masculinization of brain structure and, subsequently, gender identity, behavioural patterns (e.g., childhood play behaviour, aggression, etc.) and cognition (Hines, 2006; Swaab, 2007). In women, testosterone is produced postnatally by the ovaries and adrenal cortices, though in quantities that are an order of magnitude lower than in men (Clifton et al., 2016), who also produce testosterone in the adrenal cortices but experience substantial testosterone secretion from the Leydig cells of the testes. This divergence begins just before puberty, when girls experience a 2- to 3- fold increase in testosterone production while boys undergo a 10-fold increase, on average. In both sexes, endogenous adulthood testosterone levels appear to be implicated similarly in modulating several neural processes, including sexual desire and functioning (Isidori et al., 2005; Randolph, Zheng, Avis, Greendale, & Harlow, 2015; Wåhlin-Jacobsen et al., 2015), as well as overall cognitive functioning in older age (Barrett-Connor & Goodman-Gruen, 1999; Moffat et al., 2002; Wolf & Kirschbaum, 2002).

The early organizational effects of prenatal testosterone exposure on the nervous system are an important consideration in understanding the later activational effects of testosterone on behaviour and cognition in adulthood. Specifically, early developmental effects of hormones can shape behaviour and cognitive tendencies directly, and shape the response to hormone exposure in adulthood. Because of natural sex differences in pre- and post-natal testosterone levels, this hormone may be implicated in some overlapping processes in women and men, while in other cases its influence is moderated by sex. As one example, Thilers, MacDonald, and Herlitz (2006) found that, in a sample of 1107 men and 1276 women aged 35 to 90, testosterone levels were

positively associated with episodic memory, semantic memory, and visuospatial ability in men, but negatively associated with these aspects of cognition in women. Other aspects of cognition, such as risk aversion, may show comparable associations with testosterone levels in both sexes, but the strength of these associations appears to vary according to both testosterone levels and age (e.g., Sapienza, Zingales, & Maestripieri, 2009; Thilers et al., 2006).

Once in circulation, testosterone and its derivatives (e.g., dihydrotestosterone, estrogens, etc.) are able to pass through the blood-brain barrier (Hobbs, Curtis, Jones, & Plymate, 1992) where they have a number of poorly-understood effects, including an influence on dopamine neurotransmission (for reviews, see Sinclair, Purves-Tyson, Allen, & Weickert, 2014; Zheng, 2009). Dopamine functioning is decisively involved in reward processing and is central to contemporary models of problem gambling and, more broadly, addiction (Clark, 2014; Murch & Clark, 2016). Thus, the ability of testosterone to alter dopamine functioning implies a modulatory role in reward-related behaviour (Bayer, Bandurski, & Sommer, 2013; Dreher et al., 2007; Peper, Koolschijn, & Crone, 2013). In rats, testosterone appears to have hedonic or rewarding properties (Frye, Rhodes, Rosellini, & Svare, 2002; Schroeder & Packard, 2000). In humans, testosterone is similarly involved in brain reward processing. For instance, compared to participants in a placebo group, those who received implants containing a gonadotrophin-releasing hormone agonist—a substance that downregulates sex steroid production—experienced decreased neural activation to monetary rewards in a card-gambling task (Macoveanu et al., 2016). In this study, Macoveanu et al. were able to demonstrate that changes in testosterone caused by the implants were correlated positively with decreases in BOLD-responses in the insula. Activation of the insula occurs in response to reward, and the salience/awareness of rewards, including money (for a review, see Sescousse, Caldú, Segura, & Dreher, 2013), and has

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been well-associated with reward-related decision-making in brain imaging research (Clark, Lawrence, Astley-Jones, & Gray, 2009; Fauth-Bühler et al., 2014; Furl & Averbeck, 2011). Insula function is also implicated in gambling-related cognitive biases (Clark, Studer, Bruss, Tranel, & Bechara, 2014), risk prediction (Preuschoff, Quartz, & Bossaerts, 2008), and selection of risky bets (Clark et al., 2008; Kuhnen & Knutson, 2005).

Decision-making studies also associate higher circulating testosterone levels with a tendency to make risky decisions. For instance, in a study of 98 men, basal testosterone was found to predict increased risk-taking in an experimental investment game with real monetary payoffs (Apicella et al., 2008). A larger study by Sapienza, Zingales, and Maestripieri (2009) involving 460 graduate students also found a positive correlation between basal testosterone and a measure of financial risk-taking. Interestingly, these laboratory studies have been corroborated by field work that has found morning testosterone measures to be predictive of subsequent daily profits in male financial traders, in measures taken on the trading floor (Coates & Herbert, 2008). Thus, males may undergo a shift in risk preferences on days when they experience elevated testosterone. However, these positive associations between testosterone and different indices of risky decision-making have not been unanimously supported. Derntl, Pintzinger, Kryspin-Exner, and Schöpf (2014) failed to find a relationship between testosterone and risky decision-making in both men ($n = 45$) and women ($n = 71$), using a battery of laboratory risk-taking measures, including two gambling tasks. Similarly, a study by Zethraeus et al. (2009) failed to find a relationship between testosterone and financial risk-aversion, in women. It may be that failed replications of an association between testosterone and risky decision-making reflect some of the complexities of hormone-behaviour interactions mentioned earlier. Moderating factors, like

biological sex or cortisol levels (e.g., Mehta & Josephs, 2010; Weller et al., 2014), may be need to be considered in addition to testosterone levels.

Research on the relationship between testosterone and Iowa Gambling Task performance generally support an association between testosterone and risk-taking, but also highlights sex differences. Although imbued with the term 'gambling,' the Iowa Gambling Task broadly assesses reward-related decision-making relative to uncertain outcomes (Bechara, Damasio, Damasio, & Anderson, 1994). This task involves choosing cards from four decks that each differ in the number of cards that grant and deduct hypothetical monetary rewards from the player. A tendency to select from decks that result in more losses suggests riskier or poorer strategy. In two studies, individual differences in endogenous testosterone levels appear to predict poorer performance on the Iowa Gambling Task for women and men (Evans & Hampson, 2014; Stanton, Liening, & Schultheiss, 2011). Similarly, supraphysiological doses of testosterone administered sublingually appear to cause poorer Iowa Gambling Task performance in women (Van Honk et al., 2004). However, this same effect was not found in men who had their testosterone artificially increased to high-normal levels using an aromatase inhibiting drug that blocks the conversion of testosterone to estradiol (Goudriaan et al., 2010). Sapienza et al. (2009) suggest that sex differences in correlations between testosterone and risk-taking may result from testosterone's organizational effects. For example, preferences for risky choices have been associated with a putative approximation of prenatal androgen exposure (a lower ratio of the second and fourth finger lengths; Coates, Gurnell, & Rustichini, 2009; Garbarino, Slonim, & Sydnor, 2011; Manning, 2002). Nonetheless, together this research highlights the potential for individual differences in testosterone to account for variation in the way people strategize or behave when gambling.

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In May 2017, a search query of PsycINFO and MEDLINE databases for journal articles containing any text with the terms 'testosterone' and 'gambling,' yielded 168 results. From these, only one study was found to include both testosterone measurements and participants meeting criteria for problem gambling. In their investigation, Blanco, Ibáñez, Blanco-Jerez, Baca-Garcia, and Sáiz-Ruiz (2001) found that 29 male, treatment-seeking pathological gamblers did not differ significantly from a group of healthy, age-matched volunteers in terms of plasma testosterone levels sampled once, at baseline, in the morning (09.00am). This appears to be the only study that has directly examined whether individual differences in testosterone are linked with problem gambling. Interestingly, and as others have pointed out (e.g., Stenstrom & Saad, 2011), Blanco et al.'s findings may be confounded by the winner-loser effect in such a way that measures of testosterone could have been underestimated in the pathological gamblers. Specifically, Stenstrom and Saad argue that, because pathological gamblers are more likely to experience losses financially, occupationally, and socially, investigators might expect that—*under baseline conditions*—these individuals could display decreased testosterone. Because Blanco et al. recruited gamblers seeking treatment, one could expect those participants to have accrued significant losses before seeking treatment, and that those losses may have caused a persistent (i.e., in the order of days; Mazur & Lamb, 1980) lowering of testosterone. Other important characteristics of pathological gamblers were not considered in Blanco et al.'s study, like cortisol levels (Geisel, Panneck, Hellweg, Wiedemann, & Müller, 2015; Paris, Franco, Sodano, Frye, & Wulfert, 2010), which can alter testosterone production and modulate its effects on behaviour (Mehta & Josephs, 2010; Mehta & Prasad, 2015, see below).

Given this accumulation of evidence that supports testosterone's ability to promote risky choice in laboratory tasks and real-world financial situations, a logical extension of this work is

to investigate behavioural effects of testosterone in realistic gambling scenarios. At present, only a handful of studies have done this, and these studies have focused almost exclusively on laboratory task performance. Interestingly, van Honk et al. (2016) provide the first evidence that artificial changes in testosterone can impact decision-making during gambling in a realistic way. Considered alongside the criticisms of Blanco et al.'s (2001) investigation of testosterone levels among pathological gamblers (i.e., that a potential winner-loser effect was overlooked), the current state of gambling endocrinology research substantiates a need to shift investigative focus from individual differences in testosterone levels to the consequences of transient changes in this hormone. This need is also highlighted by recent evidence that demonstrates a link between individual differences in testosterone reactivity and risk-taking: Apicella, Dreber, and Mollerstrom (2014) showed that changes in testosterone levels following a rock-paper-scissors competition correlated negatively with the number of times participants chose smaller, certain monetary rewards (ranging from $1 to $10) over a gamble with a 50% chance of winning $10, in an incentivized risk-aversion task. However, to make inferences about the real-world effects of testosterone on gambling behaviour, researchers first need to identify whether meaningful changes in testosterone levels are pertinent to gambling involvement. One way of doing so is to investigate whether gambling wins and losses may encourage natural hormone fluctuations. A substantial literature on the winner-loser effect informs this possibility. Accordingly, the following discussion will explore what is known about this phenomenon, and the relevance of this knowledge to gambling research.

1.3   Winning, Losing, and Testosterone Fluctuation

The winner-loser effect appears to be a prevalent phenomenon observed in both animal and human studies of endocrinological responses to social competitions (Archer, 2006; Salvador & Costa, 2009; van Anders & Watson, 2006b). In humans, winner-loser effects have been elicited in both men and women, and across a variety of contexts, including sports competitions (Booth, Shelley, Mazur, Tharp, & Kittok, 1989; Elias, 1981; Mazur & Lamb, 1980), chess matches (Mazur, Booth, & Dabbs, 1992), and rigged laboratory task competitions (Gladue, Boechler, & McCaul, 1989). Some evidence suggests that direct involvement in the competition is not even a pre-requisite for win- and loss-based testosterone fluctuations. For instance, sports *fans* have been observed to experience a winner-loser effect on testosterone after watching a match involving their favourite team (Bernhardt, Dabbs, Fielden, & Lutter, 1998). However, despite the level of recognition given to the 'winner-loser effect,' a number of studies have been unable to demonstrate a change in testosterone levels following competition (e.g., Salvador, Simón, Suay, & Llorens, 1987; Suay et al., 1999). Geniole, Bird, Ruddick, and Carré (2016) recently conducted a meta-analysis that reflects the heterogeneity of winner-loser effect findings in studies collectively involving more than 2500 participants. These authors found that winners of a competition tend to experience modest increases in testosterone, compared to losers ($d = .22$ for women, $d = .23$ for men), and that the winner-loser effect is strongest for studies conducted in naturalistic settings like sports venues ($d = .46$), compared to studies conducted in laboratories, for which the winner-loser effect was weak ($d = .08$).

Until recently, the evolutionary relevance of a winner-loser effect, especially in humans, remained speculative. More than two decades ago, biologists began to contend that, based on their observations in animals, social and physiological aspects of winning aggressive interactions

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or dominance contests increase the likelihood of animals winning future antagonistic encounters (Chase, Bartolomeo, & Dugatkin, 1994; Hsu & Wolf, 2001; Lehner, Rutte, & Taborsky, 2011). Observations made within the past 15 years have convincingly shown that testosterone mediates this increased likelihood of subsequent victory, in animals. Influxes of testosterone caused by wins appear to initiate a feedback loop wherein androgenic priming promotes aggressive behaviour that helps the animal to win future contests (Earley, Lu, Lee, Wong, & Hsu, 2013; Fuxjager, Oyegbile, & Marler, 2011; R. Oliveira, Silva, & Canário, 2009; Oyegbile & Marler, 2005; Trainor, Bird, & Marler, 2004). This phenomenon was first characterized as one of the main principles of the 'Biosocial Model of Status,' which synthesized the research of numerous human and nonhuman studies to propose that the winner-loser effect on testosterone occurs after status gains or losses, and serves to alter male behaviour in ways that help maintain or increase their status (Mazur, 1985). Mazur's model also stipulates that this relationship between testosterone and status is reciprocal. That is, losing dominance competitions should suppress testosterone, and thus, subdue behaviour in order to minimize further defeat. As Casto and Edwards (2016) indicate, these aspects of the Biosocial Model of Status are important in highlighting that perception of status or status changes (i.e., of winning or losing) can influence short-term testosterone fluctuations, which can then affect future status-seeking behaviour.

Researchers have suggested that, like other primates, humans are situated within status hierarchies, wherein opportunities to compete for status are abundant. Status in these cases (animal or human) is defined by influence over other individuals or access to something of value (Mazur & Booth, 1998). In the modern age, status hierarchies take on more complex forms, like sports leagues, social networks, businesses, or governments (for a review, see Booth, Granger, Mazur, & Kivlighan, 2006). In line with this view, converging evidence is beginning to support

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behavioural consequences of the winner-loser effect in humans (Mazur et al., 1992; Page &

Coates, 2017; Zilioli & Watson, 2014). For example, in Zilioli and Watson's (2014) study,

testosterone increases following a Tetris competition on one day positively predicted skill

improvements in a second Tetris competition the next day. These findings have some subtle but

important implications for gambling research. Much like in sports or video games, one of the

reasons people gamble is to compete, either against others (Lee, Lee, Bernhard, & Yoon, 2006;

McBride & Derevensky, 2009; Neighbors, Lostutter, Cronce, & Larimer, 2002), or against 'the

house' when gambling solitarily (Cotte, 1997). Correlations between trait competitiveness and

problem gambling severity (Harris, Newby, & Klein, 2015; Parke et al., 2004) suggest that

problem gamblers are more inclined to approach gambling as a competitive activity. Crucially,

transient increases in testosterone appear to be able to influence competitive behavior in the

future. Whether this influence is beneficial or maladaptive in a gambling context remains

unexplored. Some have speculated that the effect of transient testosterone increases can be both

beneficial and disadvantageous to financial decision-making. According to Coates and Gurnell

(2017), win-based testosterone increases could be helpful in attaining short term financial trading

gains, but in the long run, foster over-confidence and larger, riskier bets with lower risk-reward

trade-offs. Given that surprisingly few researchers have sought to explore whether the winner-

loser effect applies to gambling, even a basic understanding of how gambling wins and losses

can cause marked fluctuations in testosterone has not been established.

Some prior investigations of the winner-loser effect are relevant to the consideration of

gambling behaviour. A small experiment in 28 participants by McCaul, Gladue, and Joppa

(1992) measured testosterone changes to winning or losing a chance-based game in which a $5

cash prize was offered on the cumulative outcome of 60 coin tosses; if more than 30 heads were

thrown, the participant won the cash prize. Results showed that prize winners experienced significant increases in testosterone and losers experienced decreases, 20 minutes following the end of the coin-tossing game. In other words, this study was the first to show that the winner-loser effect can occur following wins and losses that were determined by chance rather than ability. More research is needed to identify the boundary conditions for this effect in relation to real-world gambling. For example, Mazur and Lamb (1980) did not find a significant winner-loser difference with a laboratory lottery game, in which a $100 prize was awarded randomly to 7 of 14 participants. Like McCaul et al.'s study, Mazur and Lamb's experiment was limited by small sample size. However, McCaul et al.'s study was further limited by low sensitivity to modest testosterone changes due to the sample timing relative to the lottery outcome, which appears to have been 30 minutes or greater. In order to detect post-competition testosterone fluctuations, measurements are typically needed within the span of 0 to 30 minutes after winning or losing (Geniole et al., 2017).

In a more direct evaluation of the winner-loser effect in gambling, Steiner, Barchard, Meana, Hadi, and Gray (2010) measured testosterone in 32 men before and after they competed in one-on-one poker matches. Testosterone levels increased in both winners and losers between measures taken at baseline to 5 minutes after the matches. In other words, Steiner et al. did not detect a winner-loser effect, but an indiscriminate increase in testosterone caused by the poker competition. Again, methodological limitations including small sample size, the lack of a real monetary prize, and variable competition duration may have reduced their ability to detect a winner-loser effect.

A handful of recent studies have revealed some specific conditions necessary for wins and losses to influence testosterone levels. In particular, testosterone responses to competition

appear to depend heavily on psychological processes, rather than on simply winning or losing (Salvador, 2012; Salvador & Costa, 2009). For example, positive affect has been suggested to mediate the effect of winning on testosterone increases (Booth et al., 1989; Mazur & Lamb, 1980; McCaul et al., 1992). Subjective appraisals have been identified as moderators of the winner-loser effect (that promote larger testosterone fluctuations), including high motivation to win (Suay et al., 1999), significant involvement in the competition or contribution toward the outcome (Salvador, Costa, Hidalgo, & González-Bono, 2017; van Anders & Watson, 2007), and attributing competition outcomes to one's own skill or effort (González-Bono, Salvador, Ricarte, Serrano, & Arnedo, 2000; González-Bono, Salvador, Serrano, & Ricarte, 1999). Numerous situational variables may also influence the magnitude of the winner-loser effect, like an opponent's confidence (van der Meij, Buunk, Almela, & Salvador, 2010), or the location of the competition (Carré, 2009). Interestingly, the winner-loser effect appears to be reversible when wins or losses are close or uncertain (Zilioli, Mehta, & Watson, 2014): losers can experience increases in testosterone and winners can experience decreases, if the outcomes are surprising or ambiguous.

Further, increasing empirical support for the relevance of cortisol levels to testosterone's fluctuation and effect on behaviour, has helped to explain previous inconsistent empirical support for the winner-loser effect. In humans, cortisol levels vary diurnally, peaking shortly after awakening then declining throughout the day, and increase acutely in response to physical and psychological stressors (Tsigos & Chrousos, 2002). According to a premise called the 'Dual-Hormone Hypothesis,' cortisol levels are posited to interact with testosterone in such a way that testosterone's influence on status-seeking behaviour is blocked or subdued when cortisol levels are relatively high (Carré & Mehta, 2011; Mehta & Josephs, 2010; Mehta & Prasad, 2015). In

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other words, this hypothesis predicts that, for testosterone levels to influence status seeking behaviours including risk-taking, physical aggression, and dominant behaviour, individuals' cortisol levels need to be low, relative to testosterone levels. Support for testosterone × cortisol interactions have been seen in investigations of gambling-relevant behaviours including, among others, making risky choices, overbidding in auctions, economic punishment, and willingness to compete again after competition losses (for a more comprehensive review of the dual-hormone hypothesis and its implicated behaviours, see Mehta & Prasad, 2015). Importantly, testosterone and cortisol appear interact with contextual factors, such as victory and defeat. For example, a study using a multiplayer variant of the game Tetris has shown that basal cortisol can modulate post-competition increases in testosterone (Zilioli & Watson, 2012). More specifically, the winner-loser effect may depend on individual differences in cortisol status in such a way that high baseline cortisol levels preclude detectable increases in testosterone following wins.

In summary, the winner-loser effect appears to be a multifaceted biopsychological experience that occurs in a variety of situations and may extend to gambling behaviour. Currently, little research exists to inform whether gambling wins and losses can elicit meaningful testosterone changes, although the literature linking testosterone and risky behaviours strengthens the plausibility of this hypothesis. Certainly, gambling venues offer ubiquitous opportunities to compete against other gamblers, or even to compete against the dealers or 'the house.' However, attempts to study the effects of table or dealer game outcomes on testosterone levels come at the cost of generalizability to the larger populace of gamblers (e.g., slot machine gamblers) who experience problems as a result of their gambling involvement. In some respects, slot machine gambling is an inherently solitary experience wherein the chance of winning a bet is random. If perceived this way, only limited and indirect evidence suggests that this type of

experience is sufficient to elicit a winner-loser effect: Research has shown that winner-loser

effects are observable in absence of direct involvement in a competition with another person

(Bernhardt et al., 1998) and as a result of random wins and losses (McCaul et al., 1992).

However, the heterogeneity of findings regarding the winner-loser effect and the recognition of

several moderators of this phenomenon signify the importance of subjective experience in the

elicitation of this type of testosterone fluctuation. The next section will consider a further

possible moderator, in the gambler's tendency to view the slot machine as an animate intentional

being and competitor.

1.4   Anthropomorphism of Slot Machines

Individual differences have been described in the extent to which people humanize

inanimate objects, termed 'anthropomorphism.' If this tendency applies to slot machines, it could

logically create a competitive experience from what is ostensibly a solitary activity. In addition,

gambling on a slot machine might represent a competition against 'the house'—a metaphysical

extension or mental representation of the casino operator. Either cognitive style would be

expected to invoke the same endocrinological effects that underlie the winner-loser effect. In

other words, anthropomorphism, the extent to which gamblers believe the slot machine vies for

their money, may moderate a winner-loser effect on testosterone. Thinking about a slot machine

in this way would also constitute a specific instance of a gambling-related cognitive distortion

(see Fortune & Goodie, 2012; Toneatto, 1999).

Anthropomorphism is defined as the attribution of human characteristics, real or

imagined, to any non-human entity (Epley, Waytz, & Cacioppo, 2007; Kracher, 2002; Urquiza-

Haas & Kotrschal, 2015). Epley et al. (2007) proposed a theory to elucidate the circumstances

that tend to elicit anthropomorphic thinking. Their theory postulates two primary motivators for humanizing objects and animals that, importantly, support an understanding of why slot machines are a prevalent trigger for this cognitive tendency. First, these authors suggest that anthropomorphizing fulfills a ubiquitous desire for social connection, an idea otherwise referred to as sociality motivation (Baumeister & Leary, 1995; Epley et al., 2007). In other words, people anthropomorphize to feel less alone, and based on this motivation, people are presumed to be more likely to anthropomorphize when they feel deprived of social connections. Research has corroborated this idea by showing that chronic or induced feelings of loneliness cause people to anthropomorphize pets and object more (Epley, Akalis, Waytz, & Cacioppo, 2008; Epley, Waytz, Akalis, & Cacioppo, 2008). Considered alongside evidence of both a link between problem gambling and loneliness (Dowling & Brown, 2010; Porter, Ungar, Frisch, & Chopra, 2004; Trevorrow & Moore, 1998), and socializing being a prominent gambling motive (Lee et al., 2006; Neighbors et al., 2002), one might reasonably expect anthropomorphic thinking to be more prevalent among gamblers. Additionally, the inherently solitary nature of slot machine gambling may encourage feelings of isolation, which further promotes anthropomorphization.

Epley et al.'s (2007) second proposed motivator, referred to as effectance motivation, reflects a desire to understand why objects and animals behave in certain ways. In other words, people also anthropomorphize to gain a sense of understanding or control over seemingly uncontrollable or random happenings. In support of this idea, individuals with high need for control were more likely to anthropomorphize unpredictable nonhuman behaviour (Epley, Waytz, et al., 2008; Waytz, Morewedge, et al., 2010). Epley et al.'s effectance motivation is applicable to an explanation of why gamblers might imbue slot machines with intentions to win their money. For slot machine gamblers, unpredictable wins and losses could elicit a desire to

understand and explain win or loss streaks (e.g., "maybe the machine just doesn't want me to win today"). By attributing human-like motives to the machine, the gambler may gain a sense of control over their wins and losses. Interestingly, others have proposed that increased cognitive load may increase the tendency to anthropomorphize (Waytz, Gray, Epley, & Wegner, 2010). This could further implicate increases anthropomorphic thinking in gambling venues as electronic gambling machines become increasingly complex.

Some previous observations support this framework, showing that gamblers do specifically anthropomorphize slot machines. In a broad investigation of irrational thinking among slot machine gamblers, Walker and Phil (1992) had participants verbalize their thoughts aloud while they gambled on a slot machine. These authors were the first to call attention to the ubiquity of anthropomorphic comments. Using this 'think aloud' method, other researchers have observed similar patterns in the way gamblers think about and rationalize slot machine payouts (Delfabbro & Winefield, 2000; Griffiths, 1994). Importantly, their statements indicate a range of beliefs that imbue slot machines with thoughts, intentions, and emotions (e.g., "this machine doesn't like me," "I don't think it wants to pay out at all"). Crucially, these studies indicate that gamblers spontaneously rationalize slot machine payouts in terms of beliefs about slot machines having feelings or motives.

Kim and McGill (2011) tested the theory of anthropomorphism generated by Epley et al. (2008; 2007) using pictures of slot machines. First, in support of observations from past think-aloud studies, Kim and McGill found that making a slot machine appear more like a robot (i.e., more human-like) led to greater endorsement of statements including, the machine "has free will," "has intentions", and "looks like human". In keeping with Epley et al.'s theory of effectance motivation, Kim and McGill also found that self-perceived power over others was an

important moderator of participants' beliefs about slot machines depicted in photos. For example, high-power individuals were more willing to gamble on a slot machine that looked more like a human. These high-power individuals also considered a humanlike machine to be a less risky gamble. Conversely, when participants reported low social power, they were less likely to indicate their willingness to gamble on a humanlike machine and they believed that gambling on a humanlike machine would be riskier. Stated differently, participants' willingness to gamble was dictated jointly by whether they considered the slot machine to be a social entity and their self-perceived ability to exert control over others.

Electronic gambling machines vary in appearance and this may shape their tendency to elicit anthropomorphic thinking. Common slot machine themes often involve real or fictional human or animal characters. Depictions like these are often animated to move in coordination with key elements of the game, such as wins, losses, and bonus rounds. This coordination could understandably inform a sense that the game, or a character within it, is sentient and bears influence over those important elements. Considering Kim and McGill's (2011) finding that this mode of thinking interacts with self-perceived traits to influence perceptions of gambling risk and willingness to gamble, aspects of a slot machine's appearance and personal traits may jointly dictate which machines gamblers choose to gamble on. In effect, research has shown that people allocate money preferentially based on anthropomorphic considerations rather than scientific evidence (Martín-López, Montes, & Benayas, 2007). Similarly, animal conservation efforts have seen attention and funding bias for species that are more visually and phylogenetically similar to humans (Martín-Forés, Martín-López, & Montes, 2013). Thus far, the research discussed supports the hypothesis that anthropomorphism is a pertinent phenomenon to gambling behaviour and that this cognitive distortion may be a risk-factor for increased spending.

Recently, Riva et al. (2015) have directly tested whether anthropomorphic primes influenced slot machine gambling. Corroborating previous qualitative reports, their pilot study indicated that regular slot machine players anthropomorphized slot machines more than nonregular players. In three subsequent experiments, participants were assigned to two groups who played an authentic internet slot machine game. Players who were primed with an anthropomorphized description of the slot machine game placed more bets (i.e., more spins), gambled for longer, and ultimately lost more money than players who read a neutral description of the slot machine. Riva et al. also found that high-arousal, positive emotions mediated the relationship between anthropomorphism and increased gambling. These findings support the idea that humanizing electronic gambling machines is a risk-factor for financial losses and other gambling problems. Crucially, these findings represent an important first step in determining the proximal mechanisms that explain how anthropomorphism can affect gambling behaviour. However, Riva et al.'s research has only investigated the effects of artificially induced anthropomorphic thinking (via the priming instructions). Their study does not address whether individual differences in this tendency are relevant to gambling behaviour in naturalistic settings.

In summary, anthropomorphism appears to be implicated in slot machine gambling. Converging evidence suggests that anthropomorphizing slot machines influences gamblers' beliefs about these machines, in addition to their gambling behaviour. Limited evidence also indicates that anthropomorphic thinking is a risk-factor for gambling persistence, and subsequently, monetary losses (Riva et al., 2015). However, research has not yet begun to examine how individual differences in tendency to anthropomorphize might moderate important subjective experiences during gambling. One such experience may be the feeling of being in a

competition with the slot machine, which, based on evidence presented earlier, could trigger a lasting hormonal response that alters gambling behaviour.

1.5   Experimental Plan

This study was designed to help determine the relevance of fluctuations in testosterone to gambling behaviour on modern slot machines. Specifically, this experiment tested for a winner-loser effect in 120 male novice gamblers who were playing for a chance to win a $10 cash prize. By using an authentic slot machine to produce monetary wins and losses, and by allowing wins and losses to occur naturally, the experiment tested for endocrinological changes resulting from slot machine gambling under ecologically realistic laboratory conditions. In doing so, this study also contributes to a small literature that examines applied extensions of the winner-loser effect. In light of recent evidence that testosterone fluctuations can be modulated by hypothalamic-adrenal axis functioning (Mehta & Prasad, 2015; Zilioli & Watson, 2012), gambling endocrinology research has not yet considered cortisol functioning in the investigation of winner-loser effects. Considering these elements together, I predicted in this study that: (1) winning or losing sessions in slot machine gambling would elicit divergent effects on salivary testosterone levels. Specifically, I predict that winners and losers will experience increases and decreases (respectively) in salivary testosterone. Furthermore, I also predicted that: (2), as has been seen in previous research, baseline salivary cortisol will moderate testosterone fluctuation in such a way that high basal cortisol attenuates testosterone increases seen in the winner-loser effect.

The second aim of this study was to investigate anthropomorphism as a specific cognitive distortion in slot machine gambling. Specifically, this aim was to test whether the winner-loser effect on testosterone levels was moderated by individual differences in participants' tendency to

anthropomorphize the machine. By humanizing the machine, gamblers might feel that they are competing against an entity with thoughts and intentions of awarding or withholding prizes. In this case, testosterone should fluctuate after wins and losses as classically shown in sports and laboratory competitions.

Associations among gambling-relevant cognitive measures, behaviour, and testosterone levels during slot machine gambling were also considered in this investigation. As mentioned previously, the literature on testosterone, risk-taking, and gambling behaviour has scarcely looked beyond contrived gambling procedures such as the Iowa Gambling Task. Because of this, little is known about how testosterone relates to other gambling-relevant indices of risk-proneness, such as gambling-related cognitive distortions. Based on prior evidence of an association between testosterone and risky decision-making and behaviour, I hypothesized that: (4) testosterone levels during slot machine gambling would positively predict riskier gambling behaviour (i.e., more slot machine spins).

Chapter 2: Method

2.1   Participants

One hundred and twenty-four healthy male students (age $M = 21.25$, $SD = 2.91$) from the University of British Columbia were recruited using the Psychology Human Subjects Pool and Psychology Paid Participants Studies List. Depending on their mode of recruitment, participants were compensated with either psychology course credit or $15 per hour. Participants were not allowed to take part in this study if they were younger than 19 years of age, the legal age for gambling in British Columbia, or if they scored seven or greater on the Problem Gambling Severity Index (PGSI; detailed below in section 2.3.1), indicating a high risk for problem gambling. Three respondents were prevented from participating on this basis. Additional exclusion criteria were i) did not have normal or corrected-to-normal eyesight, ii) using medications that are known to affect hormone functioning, iii) smoked more than five cigarettes per day, or iv) had health problems including oral bleeding or an endocrine disorder. These exclusions resulted in 6 participants being discontinued based on a bio-demographics questionnaire.

Some further steps were taken to mitigate known confounds in hormone measurement. One day prior to their study appointments, an email reminder was sent to all participants instructing them to abstain from flossing, exercising, and consuming alcohol on the day of the study, and to abstain from brushing their teeth, eating, or drinking (aside from water) one hour before participation in the study. One participant was consistently non-compliant and was also excluded from analyses. My final sample comprised 113 participants. All procedures were reviewed and approved by the University of British Columbia Behavioural Research Ethics Board.

2.2   Modelling Winners and Losers

To produce a gambling scenario wherein participants could either win or lose a cash bonus, participants received an endowment ($40, 4000 credits in $n = 44$; $60, 6000 credits in $n = 69$[1]) 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 outcome was used to maximize the impact of a winner-loser effect in the context of a game with a continuous outcome. The Dragon's Fire game is a popular slot machine in BC gambling venues; it is a multi-line game with a maximum of 40 paylines across five computer-animated reels, with a bet denomination of one cent per line. The return-to-player percentage was set to 87.1%, meaning that an average of 12.9% of each bet placed 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.

A consequence of the ecologically-realistic design of this gambling scenario is that we could neither predict nor manipulate the wins and losses delivered by the machine. These aspects qualify the design of this study as quasi-experimental and analogous to the many studies of the winner-loser effect in real sports settings. To reduce volatility across gambling sessions, we asked participants to bet according to the 'maxi-min' strategy, which involves betting on the

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[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. No differences between the two groups that received each amount were observed on any variables of interest in this study (all $ps > .1$). Additionally, there was a comparable proportion of winners and losers who received each amount of credits, $\chi^2(1, N = 113) = 0.33, p = .57$.

maximum number of paylines with the minimum bet possible, which on our game resulted in 40 cents bet per spin. Among regular slot machine gamblers this is a preferred strategy (Livingstone, Woolley, Zazryn, Bakacs, & Shami, 2008) that increases the machine's hit percentage while minimizing long streaks of losses (Templeton, Dixon, Harrigan, & Fugelsang, 2014).

2.3   Questionnaire and Task Measures

2.3.1   Problem Gambling Severity Index

The Problem Gambling Severity Index (Ferris & Wynne, 2001) is a 9-item questionnaire that measures the prevalence of an individual's problem gambling symptoms over the previous 12 months. For each of its items, the PGSI uses a 4-point scale ranging from 0 (never) to 3 (almost always), to assess agreement with a statement about one's gambling (e.g., "Has gambling caused you any health problems, including stress or anxiety?"). PGSI scores of 0 to 2 indicate no or low risk, scores in the range of 3 to 7 connote a moderate risk, and a score greater than seven (8 to 27) suggests a high risk of developing or having gambling problems (Ferris & Wynne, 2001). The PGSI is psychometrically superior to older screening instruments such as the South Oaks Gambling Screen, for identifying and distinguishing at-risk and problem gamblers (Currie, Casey, & Hodgins, 2010).

2.3.2   Gambling Related Cognitions Scale (GRCS)

The Gambling Related Cognitions Scale includes 23 statements for which individuals rate their agreement using 7-point Likert-scales. The GRCS items span five domains (represented by five subscales) of gambling-related beliefs that, along with GRCS total scores,

have been determined to have good psychometric properties for distinguishing non-clinical

problem and non-problem gamblers (Raylu & Oei, 2004). Three of the GRCS's subscales

(predictive control, illusion of control, and interpretative bias) measure elements of illusory

control over gambling outcomes, while the remaining two subscales (perceived inability to stop

and gambling expectancies) address addictive aspects of gambling. Table 2.1 below summarizes

the five subscales of the GRCS.

Subscale	Examples
Interpretative bias	Believing that wins result from personal strengths, and losses happen by chance or due to other external influences
Illusion of control	Believing that superstitious rituals or systems of luck (e.g., carrying a lucky penny) can influence wins and losses
Predictive control	Believing that wins or losses will predict future gambling outcomes (e.g., many losses will be followed by a series of wins)
Gambling expectancies	Believing that gambling is a crucial way to maintain daily functioning (e.g., by reducing stress)
Perceived inability to stop	Believing that one's desire to gamble is too strong to ever stop

Table 2.1 Examples of beliefs corresponding to each of the GRCS subscales

2.3.3   Gambling Competitiveness Scale (GCS)

The Gambling Competitiveness Scale is a brief questionnaire that evaluates gambling-

related competitiveness in terms of both general competitive tendencies (e.g., "I am

competitive") and predispositions toward gambling for competitive reasons (e.g., "I like to

gamble to beat the system"). Respondents read six statements and report their agreement with

each statement using a 10-point scale. GCS scores are totaled across the items and have been

found to distinguish individuals with Gambling Disorder from those without (Parke et al., 2004).

2.3.4   Individual Differences in Anthropomorphism Questionnaire (IDAQ)

The Individual Differences in Anthropomorphism Questionnaire contains 30 items that provide a stable measure of individual differences in the tendency to attribute humanlike mental states to three categories of commonly anthropomorphized agents: natural entities (e.g., entities having to do with nature, such as wind), technological objects (e.g. computers) and nonhuman animals (Waytz, Cacioppo, & Epley, 2010). The IDAQ was developed to reflect Epley, Waytz, & Cacioppo's (2007) theory of anthropomorphism, and thereby to also predict this theory's three proposed consequences of anthropomorphic thinking: treating anthropomorphized agents with moral regard (e.g., affording them rights such as freedom), trusting and holding nonhuman entities responsible for their actions (e.g., punishing anthropomorphized agents), and being influenced socially by anthropomorphized agents (e.g., responding in socially desirable ways in their presence). In their validation of the IDAQ, Waytz, Cacioppo, & Epley demonstrated moderate correlations between measures of these behavioural consequences and IDAQ scores. Likewise, the IDAQ has been found to have good internal reliability ($\alpha \geq .82$ across all validation studies) and reasonable temporal stability at 12 to 19 weeks ($r = .55$, $p < .0001$) (Waytz, Cacioppo, et al., 2010).

2.3.5   Slot Machine Anthropomorphization Scale (SMAS)

Previously, Riva et al. (2015) adapted the IDAQ to quantify the extent to which participants in their study specifically anthropomorphized slot machines. Their scale, referred to in this thesis as the Slot Machine Anthropomorphization Scale, includes 15 items that each reflect or elaborate on the specific agentive vocabulary used in the IDAQ (e.g., has free will, consciousness, emotions, intentions) in ways that refer to a specific episode of slot machine play

(i.e. state-related, rather than trait-related). For example, the SMAS includes items that have been directly adapted from the IDAQ (e.g., "the slot machine experiences emotions") and ones that appear to extend key anthropomorphism concepts, such as free will (e.g., "the slot machine rewards whoever she wants"). In contrast to IDAQ items, which use 10-point scales, SMAS items have respondents rate their agreement with 15 statements by using five-point scales. Riva et al (2015) found this measure to have a level of internal reliability ($\alpha = .84$) comparable to that of the IDAQ.

2.3.6   Game Experiences Questionnaire (GEQ)

The Game Experiences Questionnaire is a 14-item questionnaire that distinguishes between seven dimensions of player experience during a digital game: positive and negative affect (e.g., "I felt good" or "I felt bored," respectively), challenge (e.g., "I had to put a lot of effort into it"), competence (e.g. "I felt skillful"), flow (e.g., "I felt completely absorbed"), sensory and imaginative immersion (e.g., "I found it impressive"), and tension (e.g. "I felt frustrated") (Ijsselsteijn et al., 2008). Respondents use 5-point Likert scales to rate their endorsement of each item thinking back to a game they just played. The GEQ has been used previously to measure the experiences of slot machine players (e.g., Dixon, Collins, Harrigan, Graydon, & Fugelsang, 2013), and is reported to have good test-retest reliability and convergent validity with physiological measures of affect and arousal during gaming experiences (Ijsselsteijn et al., 2008).

2.3.7 Positive and Negative Affect Schedule – Expanded Form (PANAS-X)

The expanded form Positive and Negative Affect Schedule is a widely used, 60-item questionnaire that measures mood broadly along two dimensions: positive and negative affect. The measure distinguishes among specific subcategories of positive and negative affect by yielding 11 subscales each corresponding to a more specific positive or negative mood state (e.g., guilt, joviality, irritability, enthusiasm, etc.). After reading each item, respondents report the extent that they feel the emotion listed in the item on a 5-point Likert scale. The PANAS-X has been thoroughly validated against a variety of affect measures, including peer-judgments of mood, other comparable affect questionnaires, and measures of personality and emotionality (Watson & Clark, 1994; Watson, Clark, & Tellegen, 1988). Accordingly, PANAS-X scales for positive and negative affect were used in lieu of GEQ positive and negative affect scales, for all analyses.

2.3.8 Play Behaviour During the Gambling Session

To derive a measure of participants' play behaviour, their slot machine spins were monitored throughout the gambling session. This was achieved via a sensor attached to the spin button on the slot machine. Each time the spin button was depressed, a 'Makey Makey' (Joylabz, Cambridge, MA) relayed a signal to a recording laptop that was synchronized with the start time of each gambling session. Button press data was recorded with and extracted from AcqKnowledge® 4.4 (Biopac Systems Ltd., Goleta, CA). To mitigate the recording of accidental presses or of button pressing in excess of actual betting, button press events were not considered if they occurred within .5 seconds from the previous press (Murch, Chu, & Clark, 2017). Video

recordings of the gambling sessions were also taken in order to have a transcript for examining atypical button pressing patterns (e.g., rapid button pressing or long breaks in gambling)

2.4   Procedure

2.4.1   Pre-Gambling Phase

To control for diurnal hormone fluctuation, all testing occurred between 13:00 h and 19:00 h (Campbell, Walker, Riad-Fahmy, Wilson, & Griffiths, 1982; Dabbs, 1990; Horrocks et al., 1990). On arrival at the laboratory, participants were greeted by a male experimenter who provided a verbal overview of the study session before obtaining written consent. Participants then completed the PGSI and, if eligible to continue, were provided with water to rinse their mouths of any food residue that could interfere with hormone assays. Following this, participants were led to a lab containing four authentic slot machines. There, they first completed a series of trait questionnaires (GRCS, GCS, and IDAQ) before gambling to prevent this experience from influencing responses on these measures. Next, the experimenter assisted in attaching physiological monitoring equipment, and participants were given 5 minutes alone to provide a baseline saliva sample (T1).

2.4.2   Gambling Phase

Participants were seated at the slot machine and received brief verbal directions for the gambling session while the experimenter loaded cash into the machine. Each participant was told that the slot machine was authentic and unmodified, and that any winnings above their initial endowment would be converted to a cash prize of $10. The experimenter explained that the slot machine session would last for a fixed period of time, and they would be notified when this limit

is reached. Participants were then allowed to gamble alone for 15 minutes (based on testosterone time course data by Zilioli & 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.

2.4.3   Post-Gambling Phase

Immediately following the gambling session, participants were assisted in removing the physiological monitoring equipment and then completed a series of self-report state measures (GEQ, SMAS, PANAS-X) that surveyed participants' experiences during the gambling session. Participants provided another saliva sample (T2) exactly 15 minutes after the end of the gambling session, then completed two computerized tasks, not reported here. At exactly 30 minutes past the end of the gambling session, participants provided a third saliva sample (T3) and completed a bio-demographic questionnaire asking about relevant covariates for hormone measurement (e.g., height, weight, oral and overall health, alcohol and stimulant consumption, and sleep pattern). Following completion of the study, participants were debriefed orally and in writing about the specific aims and predictions of the study.

2.5   Saliva Samples and Hormone Assays

Saliva samples were used to derive estimates of unbound serum testosterone and cortisol. Amounts of these hormones in saliva are highly correlated with unbound serum levels (Duplessis, Rascona, Cullum, & Yeung, 2010; Shirtcliff, Granger, & Likos, 2002). Participants

provided saliva samples using the passive drool method into sterile polypropylene vials over 5

minute periods. Following collection, saliva was immediately frozen and stored at a constant

temperature of -20°C until analysis at the British Columbia Institute of Technology Natural

Health and Food Products Research Group Laboratory, where I performed all hormone assays.

All samples were assayed in duplicates for testosterone and cortisol using commercially

available enzyme-linked immunosorbent assay kits (Salimetrics® LLC, State College, PA).

Immunoassay is a cost-effective and reliable method for quantifying concentrations of analytes

in bodily fluids. The assay kits used in this study employ a competitive binding technique in

which hormone molecules from participants' saliva compete with an enzyme-linked antigen to

bind to hormone-specific antibodies that have been fixed to the wells of a microtiter plate. After

a period of incubation that allows these molecules to compete for binding positions on the

surfaces of the plate wells, unbound antigens are washed from the plate and a chemical substrate

is added to every well. At this stage, the enzyme from the enzyme-antigen conjugate reacts with

the added substrate to produce a color change according to how much of the conjugate is bound

in each well. Because of this, the colour change is inversely proportional to the amount of

hormone bound to the antibodies in the well and can be used to derive a measurement of

hormone concentration in the saliva. This extrapolation is based on comparison of the optical

density (i.e., the precise amount of colour or 450nm light measured by a plate reader) of each

well against a calibration curve established using a series of plate wells that make up a gradient

of known quantities of the hormone.

Assay precision is typically assessed using two statistics, the intra-assay and inter assay

coefficients of variation. The former is an estimate of precision that considers the coefficients of

variation of every hormone measurement included in the study and reflects the average

discrepancy between assay duplicates. Similarly, the inter-assay coefficient of variation is an index of assay-to-assay (or plate-to-plate) variability that is typically derived using measurements on each plate of identical known quantities of the analyte being measured. In this study, average intra-assay coefficients of variation were 4.24% for testosterone and 4.86% 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 11.40% for testosterone and 6.47% for cortisol. These values do not exceed the widely recognized limits of 10% and 15% for acceptable intra-assay and inter-assay coefficients of variation, respectively (Hsing et al., 2007; Schultheiss & Stanton, 2009).

2.6 Covariates of Hormone Levels

Several variables that have been observed to influence testosterone levels were considered as covariates in analyses of the winner-loser effect, including body mass index ($M = 23.80$, $SD = 5.27$), age (Gapstur et al., 2002; Ukkola, Gagnon, & Rankinen, 2001), sleep duration/schedule ($M_{hours} = 7.25$, $SD = 1.51$), time of day (Diver, Imtiaz, Ahmad, Vora, & Fraser, 2003; Luboshitzky, Zabari, Shen-Orr, Herer, & Lavie, 2001), physical fitness assessed by number of hours of physical activity per week (the modal response was greater than 4 hours) (Nindl et al., 2001; Tremblay, 2004), caffeine consumption within the last 12 hours ($n = 51$) (Beaven et al., 2008; Bonati et al., 1982), regular cigarette smoking ($n = 5$) (English et al., 2001), recreational drug use ($n = 22$) (Fronczak, Kim, & Barqawi, 2012), sexual activity during the previous 24 hours ($n = 10$) (Dabbs & Mohammed, 1992), partnered status, defined as being in a serious, committed relationship with one individual ($n = 32$), and sexual orientation (van Anders

& Goldey, 2010; van Anders & Watson, 2006a). Sexual orientation was coded according to whether or not participants endorsed non-heterosexual status ($n = 8$).

2.7   Statistical Analyses

2.7.1   Models of Hormone Fluctuation

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, 2011; Raudenbush & Bryk, 2002; Singer & Willett, 2003). Hierarchical linear modeling is ideal for analysis of repeated steroid hormone measurements (Hruschka, Kohrt, & Worthman, 2005) and provides several advantages over repeated measures ANOVA, which the existing literature on winner-loser effects has relied upon. Specifically, hierarchical linear models do not require balanced data or that complete data be present in all participants (Goldstein, 2011), and this modeling strategy typically provides greater power for identifying effects and contrasts due to its greater accuracy in modelling variance and covariance components (Gueorguieva & Krystal, 2004; Quené & Van Den Bergh, 2004).

To assess testosterone reactivity from pre- to post-gambling, a level-1 model was constructed using the three testosterone measurements from each participant and corresponding saliva sampling times. With these two variables, testosterone change trajectories for each participant were modeled linearly over time and considered to depend on several unique parameters, including fixed effects (namely, predictor variables at levels 1 and 2) and random

effects (the intercept and slopes of the individual change trajectories), which were assumed to vary across participants. Given the suitability of linear models for data involving relatively few repeated measurements taken over a short time period (Raudenbush & Bryk, 2002), testosterone reactivity was modeled linearly, as opposed to using a quadratic growth model. Saliva sampling times were coded in minutes according their departure from baseline (i.e., T1 = 0, T2 = 30, T3 = 45). The resulting level-1 model was as follows:

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

According to this model, the salivary testosterone level at measurement t for person i is predicted by the individual's testosterone intercept, π_{0i}, and the linear rate of change over saliva sampling occasions, π_{1i}. Because 0 was used to code for time at T1 (the baseline saliva measurement) the intercept in this case (π_{0i}) reflects salivary testosterone at baseline. This level-1 equation served as an "unconditional" model with no additional predictors included at any other levels. Separate level-2 equations were then introduced to this model that allowed variation in the level-1 parameters, specifically slopes and intercepts of testosterone change trajectories, to be predicted by level-2 (person-level) variables; for example, winning versus losing. In accordance with guidelines for multilevel models (Enders & Tofighi, 2007), continuous level-2 variables were grand-mean centered and all other model variables were binary coded (as 0 or 1) and included uncentered.

Separate models were constructed to test hypotheses 1 to 3. To test hypothesis 1, that the outcome of the gambling session would produce a winner-loser effect on testosterone, the first model added a single level-2 predictor—a dummy variable indicating the outcome of the gambling session (winners = 1, losers = 0). From this, variance in individual slopes of

testosterone change (reactivity) at level 1 could be predicted by the outcomes of the gambling

sessions. This resulted in the following level-2 equation for this model:

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

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

In hypotheses 2 and 3, similar models were constructed to test moderation effects of baseline

cortisol and slot machine anthropomorphization. Each variable was included as a level-2

predictor variable (in separate models), along with the corresponding interactions with the

gambling outcome variable. For example, the level-2 equation including cortisol took the

following form:

$$\pi_{0i} = \beta_{00} + \beta_{01}(Cortisol)_i + \beta_{02}(Outcome)_i + \beta_{03}(Cortsiol \times Outcome)_i + r_{0i}$$

$$\pi_{1i} = \beta_{10} + \beta_{11}(Cortisol)_i + \beta_{12}(Outcome)_i + \beta_{13}(Cortsiol \times Outcome)_i + r_{1i}$$

Covariates of testosterone with a potential to confound basal levels or fluctuation over the

course of this study's procedure were considered for inclusion in these models based on a similar

procedure used by Hackman, Betancourt, Brodsky, Hurt, and Farah (2012): each of the potential

control variables listed in section 2.6 were tested individually in the prediction model containing

only Outcome as a level-2 predictor. Variables for which coefficients were significant and model

fit was improved at a level of $p < .10$ were included in this model concurrently (Singer & Willett,

2003). Next, control variables were removed that were not significant at a level of $p < .05$,

starting in order of the highest p values and ending when only significant control variables were

left in the model. This same procedure was carried out for subsequent models that considered

baseline cortisol and anthropomorphism of the slot machine

Coefficients for cortisol and anthropomorphism in each model are estimated when

Outcome is coded as 0. Because of this, models with significant interaction effects were run

twice, once with the previously mentioned coding scheme (winners coded as 1 and losers coded as 0), and another time with winners coded as 0 and losers coded as 1. By recoding the outcome variable in this way, coefficients for cortisol and anthropomorphism were estimated for both winners and losers.

2.7.2 The Effects of Testosterone and Cortisol on Gambling Behaviour

Hierarchical modeling was also used to assess hypothesis 4, which predicted a positive association between testosterone levels (a predictor variable in this case) and gambling behaviour as the outcome or dependent variable. Given that my design constrained both the betting strategy and session length (precluding the measurement of persistence), slot machine play behaviour was operationalized as the number of spins (bets) during the play session (see section 2.3.8). Spin presses were binned into 3-minute intervals so that change in behaviour throughout the session could be assessed using a longitudinal growth model. As the number of spin presses in each time bin constitute non-continuous 'count' data (i.e., only positive integers), the level-1 sampling model was assumed to be Poisson distributed. When multilevel repeated measurement data are discrete, Poisson models provide an advantage over normal-distribution models of improving the estimation of variance components by reducing multicollinearity (Greenberg & Phillips, 2014; Raudenbush & Bryk, 2002). A Poisson model of level-1 data specifies the dependent variable, η, in the following way:

$$\eta_{ti} = \log(\lambda_{ti})$$

In this case, η_{ti} is the log of the spin rate, λ_{ti}, which represents the rate of play by person, i, within a 3-minute portion of the gambling session, t. There were no *a priori* assumptions about

the shape of the play behaviour growth model, so the level-1 data were visually inspected to determine whether changes in betting during the session occurred at a linear or nonlinear rate. On average, participants' play behaviour appeared to fit a nonlinear growth trajectory (detailed later in section 3.5.1). This was confirmed using a step-up procedure described by Long and Ryoo (2010), to determine the best nonlinear link function to model these data. Starting with an intercept-only model including all fixed effects of interest, model fit was assessed by calculating Akaike's information criterion (AIC; Akaike, 2011). This value was then compared to corresponding values of a linear model of these effects and a series of nonlinear models with increasing values of m, the exponent of the polynomial in the link function. The m value corresponding to the lowest AIC (indicating best fit) was chosen. This process resulted in the following level-1 quadratic model:

$$\eta_{ti} = \log(\lambda_{ti}) = \pi_{0i} + \pi_{1i}(Time\ Bin_{ti} - 1) + \pi_{2i}(Time\ Bin_{ti} - 1)^2 + e_{ti}$$

Time bins were coded according to order, with a constant of 1 subtracted (e.g., the first time bin was coded as 0, the second bin was coded 1, etc.). This way, the intercepts in the models of play behaviour represent initial status, or rate of play in the first 3-minute portion of the gambling session. Interpretation of this level-1 model is similar to models in the previous section. However, the addition of the polynomial term now allows the acceleration (or curvature) of the growth trajectory to vary across participants and be predicted by level-2 variables.

Testosterone, Outcome, and their interaction term were added as level-2 predictor variables in a stepwise fashion, first testing the association between Outcome and slot machine play, to establish whether individual play styles were associated with winning and losing. Outcome and the Testosterone $\times$ Outcome interaction term were then added in a second model to

explore the relationship between baseline testosterone and play behaviour. All level-2 predictors of slot machine play were grand-mean centered. Since hypothesis 4 makes no predictions about acceleration, or curvature, of rate of play over time, only fixed effects for intercept and slope are included. Accordingly, the level-2 equations for the final model of betting behaviour are as follows:

$$\pi_{0i} = \beta_{00} + \beta_{01}(Outcome)_i + \beta_{02}(Testosterone)_i + \beta_{03}(Outcome \times Testosterone)_i + r_{0i}$$

$$\pi_{1i} = \beta_{10} + \beta_{11}(Outcome)_i + \beta_{12}(Testosterone)_i + \beta_{13}(Outcome \times Testosterone)_i + r_{1i}$$

$$\pi_{2i} = \beta_{20} + r_{2i}$$

Estimation and testing of all hierarchical model parameters in this study were carried out using HLM 7.01 (Scientific Software International Inc., Skokie, IL) with restricted maximum likelihood estimation and robust standard errors. All other analyses were performed using SPSS 24.0 (IBM, Armonk, NY). For tests involving Poisson distributed variables, unit-specific models were reported, as these models are more appropriate for analysis of within-person change than population-average models (Szmaragd, Clarke, & Steele, 2013). For each hierarchical model, standardized residuals at each level and Mahalanobis distance were calculated to investigate whether any data points excessively influenced the models. No cases were found to impart unreasonable influence on the models, nor were any assumptions of the models violated. Furthermore, model coefficients and significance values were not unduly affected by the inclusion of control variables or the exclusion of hormone outliers. All significance tests were two-tailed ($\alpha = .05$).

Chapter 3: Results

3.1   Preliminary Analyses of Principal Model Variables for Hormone Reactivity

3.1.1   Hormone Measures

In line with previous research, baseline (T1) and post-task (T2 and T3) measurements were normally distributed for testosterone and positively skewed for cortisol. Cortisol values were normalized by applying a log (n + 1) transformation, as has been done previously (Mehta, Welker, Zilioli, & Carré, 2015; Wu, Eisenegger, Zilioli, Watson, & Clark, 2017; Zilioli & Watson, 2012). For testosterone, 4 participants had one or more values that differed by more than three standard deviations from the mean of the corresponding time point measure, and thus were considered outliers. These values, which were removed from subsequent analyses, constituted 2.7% of the data at level 1 of the hierarchical linear models for testosterone. Similarly, 3 participants had one or more cortisol value that were removed from analyses on the same basis[2] (1.5% of level-1 data).

Table 3.1 below lists descriptive statistics for testosterone and untransformed cortisol concentrations. As has been observed previously (e.g, Mehta, Welker, Zilioli, & Carré, 2015; Popma et al., 2007; Zilioli & Watson, 2012), baseline testosterone and cortisol were moderately correlated ($r(107) = .35$, $p < .001$). Time of day was not correlated with testosterone ($r(109) = -.113$, $p = .24$), but time did correlate negatively with cortisol, ($r(109) = -.318$, $p = <.001$), i.e. cortisol levels were higher earlier in the day. Correlations among hormone measurements are shown below in Table 3.2.

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[2] Because these hormone data are not missing at random, the final hierarchical linear models were tested separately with testosterone and cortisol outlier values included (Singer & Willett, 2003). Based on these tests, coefficients and *p*-values were determined to not be unduly influenced by the missing values.

	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)	135.65 (3.43)	35.99		133.97 (4.99)	34.94		137.01 (4.74)	37.05
Pre-gambling cortisol (µg/dL)*	.1665 (.007)	.07		.1651 (.012)	.08		.1677 (.009)	.07
Post-gambling cortisol, +15min (µg/dL)*	.1221 (.005)	.05		.1215 (.007)	.05		.1227 (.006)	.05
Post-gambling cortisol, +30 min (µg/dL)*	.1110 (.004)	.05		.1122 (.007)	.05		.1101 (.005)	.04

*Calculations for means, standard error of the mean (SEM), and standard deviation were based on untransformed cortisol values

Table 3.1 Descriptive statistics for untransformed hormone measurements

	I.	II.	III.	IV.	V.	VI.
I. Pre-gambling testosterone (pg/mL)	—					
II. Post-gambling testosterone, +15 min (pg/mL)	.873**	—				
III. Post-gambling testosterone, +30 min (pg/mL)	.802**	.916**	—			
IV. Pre-gambling cortisol (µg/dL)	.347**	.327**	.301	—		
V. Post-gambling cortisol, +15min (µg/dL)	.232*	.241*	.250*	.858**	—	
VI. Post-gambling cortisol, +30 min (µg/dL)	.201*	.240*	.308**	.748**	.910**	—
VII. Time of saliva sample	-.113	-.115	-.179	-.318**	-.358**	-.378**

$* p < .05; ** p < .01$

Table 3.2 Correlations among measures of testosterone, cortisol, and time

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3.1.2   Anthropomorphism Measures

Excluding one outlier, scores for generalized trait anthropomorphism (IDAQ; $M = 59.04$, $SD = 19.09$) were normally distributed. There was also substantial variability in slot machine anthropomorphization scores (SMAS; $M = 29.34$, $SD = 11.80$), which were positively skewed with no outliers. The two measures of anthropomorphism were moderately correlated ($r(111) = .46$, $p < .001$). Histograms for trait anthropomorphism and slot machine anthropomorphization are depicted in Figure 3.1 and Figure 3.2, respectively.
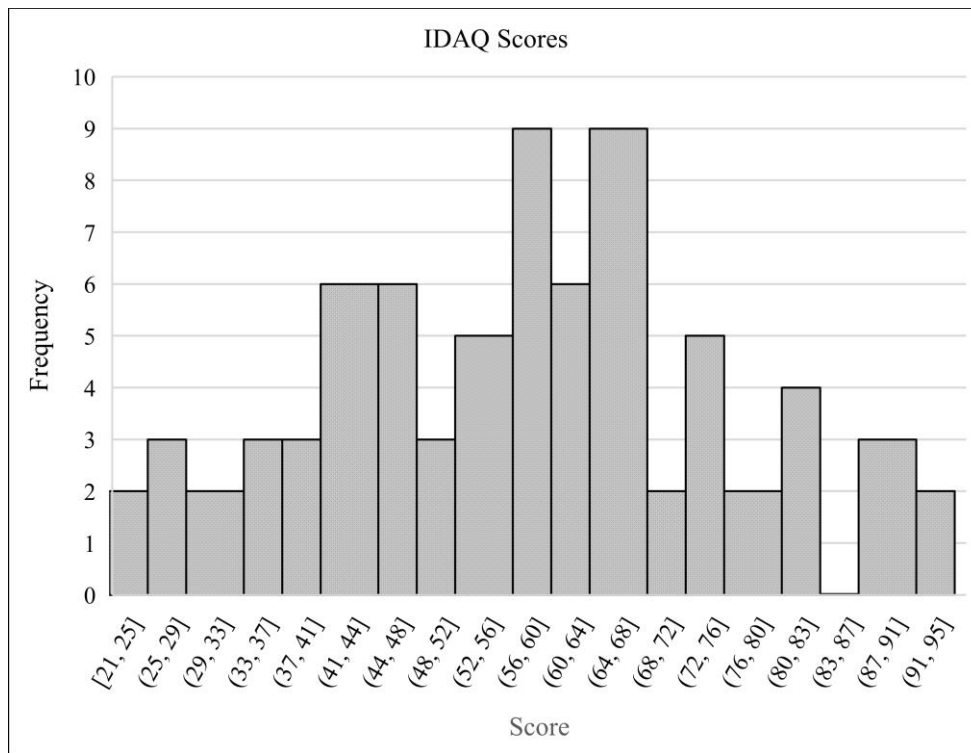
Figure 3.1 Histogram of IDAQ scores
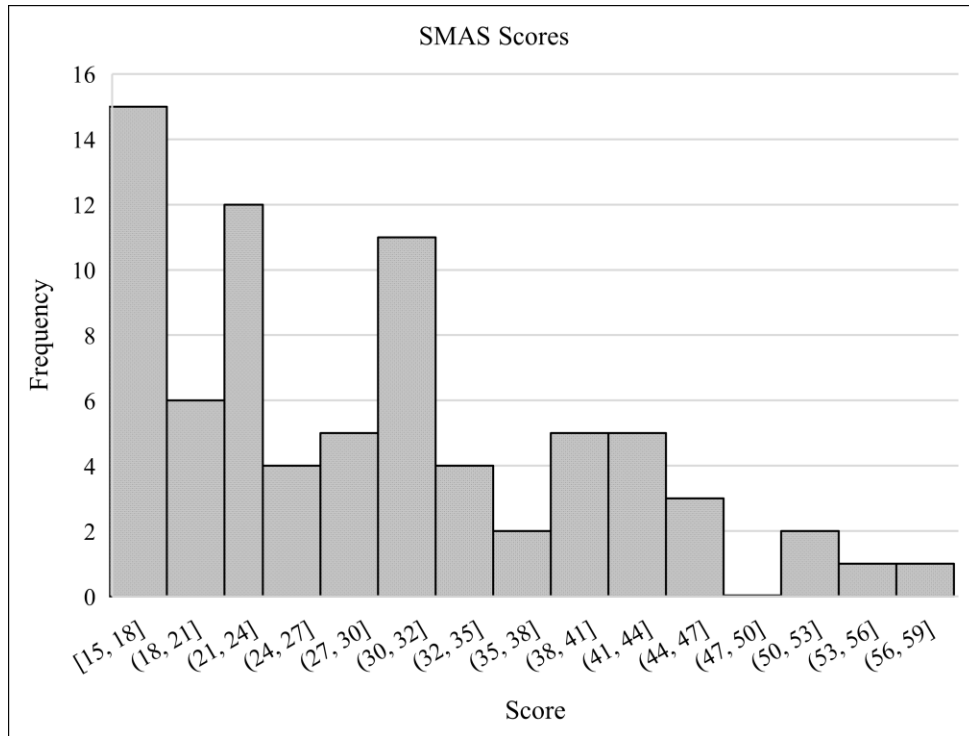
Figure 3.2 Histogram of SMAS scores

3.1.3   Wins and Losses

Net slot machine scores (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 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). Independent-samples t-tests revealed that winners and losers did not differ significantly on any bio-demographic variables (all ps > .15), nor did they differ on relevant measures of gambling behaviour, including PGSI ($t(111)$ = 1.42, p = .16), GRCS total and subscale scores (all ps > .10), or gambling competitiveness ($t(111)$ = -1.21, p = .23). Notably, winners and losers had comparable baseline levels of both testosterone

(t(109) = .27, p = .79) and cortisol (t(109) = .24, p = .81), and did not differ in their general

tendency to anthropomorphize nonhuman entities (t(111) = -.89, p = .38).

Examining measures of participants' subjective gambling experiences revealed that

winners and losers anthropomorphized the slot machine to a similar extent (t(111) = -.05, p =

.96). As expected, winning the gambling session was associated with greater post-gambling

measures of positive affect (t(111) = -2.80, p = .006) and lower measures of negative affect

(t(111) = 2.90, p = .005), on the PANAS-X. Winners also reported having significantly greater

feelings of competence (t(111) = 9.84, p = < .001) and lower feelings of tension/annoyance

(t(111) = 3.16, p = .002) during the gambling session. Winners and losers scored similarly on the

other GEQ scales reflecting gambling immersion (t(111) = -1.63, p = .11), flow (t(111) = -1.05, p

= .30), and challenge (t(111) = .79, p = .43).

Twelve participants (11 winners and 1 loser) did not gamble continuously throughout the

gambling session, either because they ran out of credits (1 participant) or because they

voluntarily elected to stop play before the experimenter returned. Thus, it is possible that these

participants experienced higher appraisals of their skill and/or play strategy as contributing

factors to their wins or losses. Because these appraisals can influence a winner-loser effect

(González-Bono et al., 2000; Salvador et al., 2017; van Anders & Watson, 2007), an additional

dummy variable was coded (0 = played full session, 1 = stopped early) for each participant, and

was added as a further covariate in the models adjusting for bio-demographic variables (see

section 2.7).

3.2   Effects of Wins and Losses on Testosterone and Cortisol

Following exclusion of outliers (section 3.1.1), there were 326 level-1 observations, comprising complete salivary testosterone data for 108 participants and partial data for a further 3 participants. Two participants had insufficient level-2 data to be included in analyses of testosterone change. For cortisol, there were a total of 334 level-1 observations.

The unconditional model for testosterone change from pre- to post-gambling produced a significant fixed effect for slope in the negative direction ($B = -.13$, $p = .01$), indicating that, on average, participants' salivary testosterone decreased during the study. The unconditional model generated significant variance estimates for both intercept ($\sigma_0^2 = 1617.23$, $p < .001$), representing variance of pre-gambling testosterone measurements, and slope ($\sigma_1^2 = .21$, $p < .001$), indicating substantial variation in both baseline testosterone levels and testosterone reactivity during the study. Consequently, there was sufficient variability for these measures to be predicted by person-level variables at level 2 in the hierarchical linear models.

Gambling outcome and the significant control variables were added to the model to test whether winners and losers of the gambling session experienced differences in testosterone reactivity during the study procedure. Results from this model are presented below in Table 3.3. Hypothesis 1 was not supported: the association between Outcome and testosterone reactivity (slope) was not significant, ($B = -.05$, $p = .60$), indicating that testosterone change from pre- to post-gambling was similar for both winners and losers. Significant variance estimates for intercept ($\sigma_0^2 = 1611.59$, $p < .001$) and slope ($\sigma_1^2 = .18$, $p < .001$) indicate that a substantial amount of variability in testosterone baselines and reactivity was not explained by Outcome and the control variables. Thus, further model specification was appropriate to explain variance in baseline testosterone measures and pre- to post-gambling testosterone changes. In the equivalent

	Effects of Outcome on Testosterone		
Fixed Effects	Estimate	SE	p
Pre-gambling testosterone, π_{0i}			
Intercept	143.04	5.34	$< .001$
Gambling outcome	1.61	7.83	.84
Sexual orientation	-30.64	9.64	.002
Testosterone reactivity, π_{1i}			
Intercept	-.24	.066	$< .001$
Gambling outcome	-.054	.10	.60
Caffeine within past 12 hours	.22	.087	.014
Sexual orientation	.53	.15	$< .001$

Random Effects	Estimate		p
Level 1			
Within-person (σ_ε^2)	88.75		—
Level 2			
Pre-gambling testosterone (σ_0^2)	1611.59		$< .001$
Testosterone reactivity (σ_1^2)	.18		$< .001$
Covariance, σ_1^0	-8.75		—

Table 3.3 Associations between gambling outcome and testosterone baseline and change
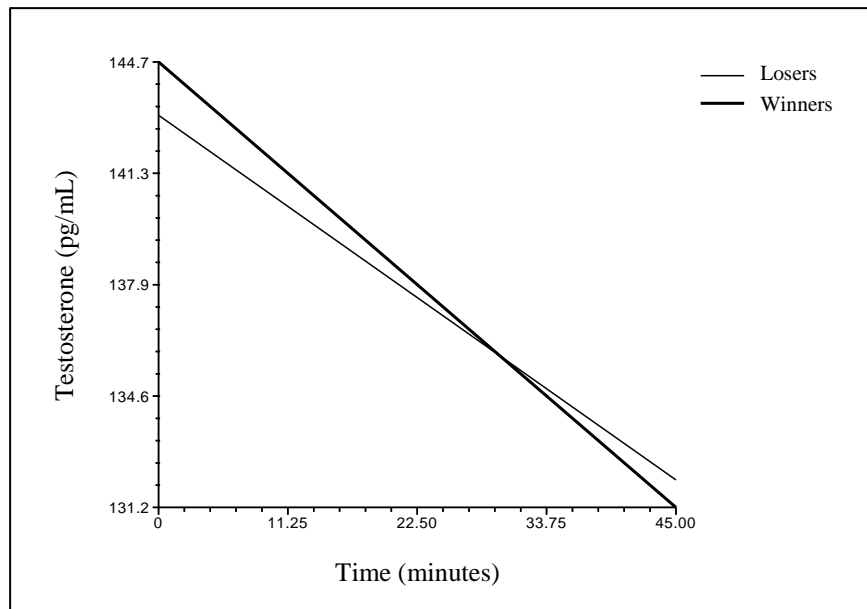

Figure 3.3 Testosterone reactivity for winners and losers

model with cortisol as the dependent variable, the unconditional model showed that cortisol also declined on average from pre- to post-gambling ($B = -.0013$, $p = < .001$). Similarly, when Outcome and significant control variables were added to the model, no associations between Outcome and either baseline cortisol ($B = .0090$, $p = .50$) or cortisol reactivity ($B = -.000059$, $p = .78$) were observed, indicating that winners and losers had similar baseline cortisol levels and experienced similar decreases over the study session (see Table 3.4).

Fixed Effects	Effects of Outcome on Cortisol		
	Estimate	SE	p
Pre-gambling cortisol, π_{0i}			
Intercept	.174239	.008400	< .001
Gambling outcome	.008957	.013370	.504
Time of day	-.000003	-.000001	< .001
Age	-.006608	.001925	< .001
Sexual orientation	-.056437	.015853	< .001
In a committed relationship	-.023938	.007315	.001
Cortisol reactivity, π_{1i}			
Intercept	-.001323	.000136	< .001
Gambling outcome	-.000059	.000216	.783
Age	.000081	.000038	.033
Sexual orientation	.000958	.000343	.006
Random Effects	Estimate		p
Level 1			
Within-person (σ_ε^2)	.00021		—
Level 2			
Pre-gambling cortisol (σ_0^2)	.00438		< .001
Cortisol reactivity (σ_1^2)	< .00001		< .001
Covariance, σ_1^0	-.00006		—

Table 3.4 Associations between gambling outcome and cortisol baseline and change
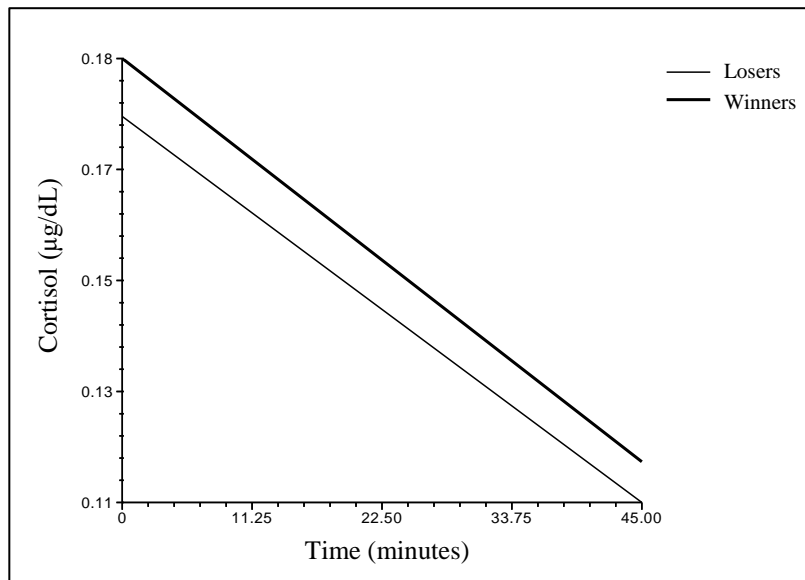
Figure 3.4 Cortisol reactivity for winners and losers

3.3   Testing Moderators of the Winner-Loser Effect

3.3.1   Cortisol

According to hypothesis 2, baseline cortisol levels should moderate (specifically,

attenuate) changes in testosterone in response to winning gambling sessions (hypothesis 1).

Thus, hypothesis 2 predicted a significant association between the Outcome $\times$ baseline cortisol

interaction term, and the slope of testosterone change. However, since hypothesis 1 was not

supported, hypothesis 2 was modified to include Cortisol and the Cortisol $\times$ Outcome interaction

terms in the next model, to determine whether wins and losses would generate divergent effects

on testosterone after controlling for baseline cortisol (see Wu et al., 2016). The results of this

model (see Table 3.5) show that both baseline cortisol and testosterone were positively

associated ($B = 162.38$, $p = .009$), but that baseline cortisol ($B = -1.01$, $p = .20$) and the Cortisol

$\times$ Outcome interaction ($B = -.11$, $p = .93$) did not significantly predict the slope of testosterone

change. Accordingly, the effect of Outcome on both testosterone baseline ($B = -.17$, $p = .99$) and

reactivity ($B = -.038$, $p = .87$) remained non-significant. In other words, the addition of cortisol in this model did not support the hypothesis that the winner-loser effect on testosterone would be moderated by cortisol.
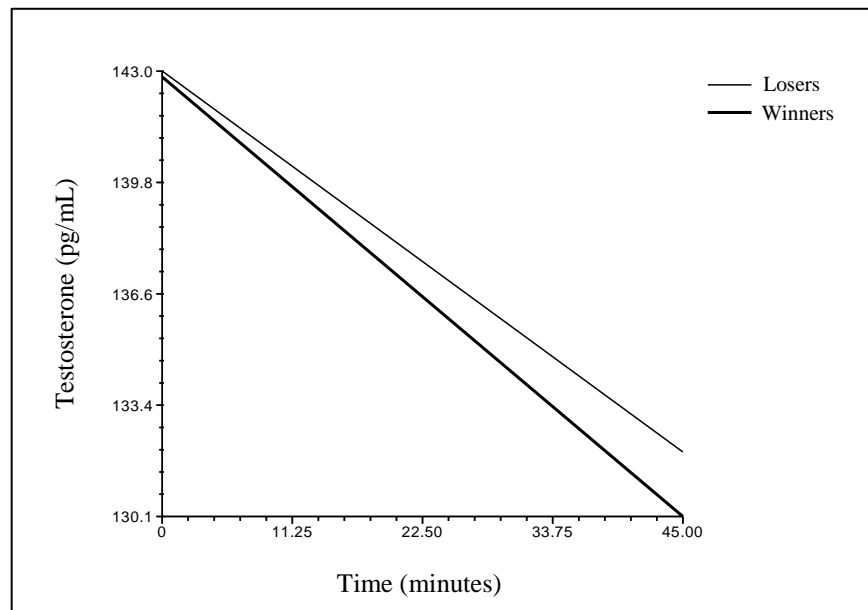
Figure 3.5 Testosterone reactivity for winners and losers, with the inclusion of Cortisol

Thus far, I have shown that testosterone levels did not change differently in gambling winners and losers (failing to support hypothesis 1), and testosterone fluctuation was not moderated by baseline cortisol, as predicted by hypothesis 2 and the dual-hormone hypothesis. In the next set of models, I turned my attention to the role of anthropomorphization of the slot machine in explaining the established variability in testosterone fluctuation.

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	Cortisol			Anthropomorphization		
Fixed Effects	Estimate	SE	p	Estimate	SE	p
Pre-gambling testosterone, π_{0i}						
Intercept	143.04	8.23	< .001	122.16	9.52	<.001
Cortisol	162.38	60.89	.009			
Anthropomorphism of the slot machine				.55	.47	.24
Gambling outcome	-.16	16.80	.99	48.69	21.50	.026
Cortisol × Gambling outcome	11.92	82.11	.89			
Slot machine anthropomorphization × Gambling outcome				-1.62	.64	.012
Sexual orientation	-20.99	10.02	.039	-29.17	9.53	.003
Testosterone reactivity, π_{1i}						
Intercept	-.25	.11	.027	-.25	.14	.085
Cortisol	-1.01	.78	.20			
Anthropomorphism of the slot machine				.001	.005	.77
Gambling outcome	-.038	.22	.87	-.05	.31	.88
Cortisol × Gambling outcome	-.11	1.17	.93			
Slot machine anthropomorphization × Gambling outcome				-.0002	.009	.98
Caffeine within past 12 hours	.22	.089	.016	.22	.084	.012
Sexual orientation	.47	.15	.003	.53	.15	< .001

Random Effects	Estimate		p	Estimate		p
Level 1						
Within-person (σ_ε^2)	88.79		—	88.74		—
Level 2						
Pre-gambling testosterone (σ_0^2)	1475.33		< .001	1545.46		< .001
Testosterone reactivity (σ_1^2)	.18		< .001	.19		< .001
Covariance, σ_1^0	-7.85		—	-8.89		—

Table 3.5 Effects of Cortisol and Anthropomorphization on Testosterone change

3.3.2 Anthropomorphization of the Slot Machine

This model incorporated the slot machine anthropomorphization score (and its interaction with gambling outcome) in place of the cortisol predictors in the previous model, to test whether testosterone reactivity was moderated by the tendency to humanize the slot machine (hypothesis 3). Testosterone reactivity was neither associated with Anthropomorphization ($B = .001$, $p = .77$) or the Anthropomorphization $\times$ Outcome interaction term ($B = -.0002$, $p = .98$) (Table 3.5). While the association between Anthropomorphization and baseline testosterone was not significant in this model ($B = .55$, $p = .24$), baseline testosterone was significantly predicted by the Anthropomorphization $\times$ Outcome interaction term ($B = -1.62$, $p = .012$). In this case, coefficient estimates were derived from Outcome values of 0, reflecting the association in the losers. To further characterize the significant Anthropomorphization $\times$ Outcome interaction, the model was re-run with winners and loser reverse coded to provide an estimate of the association between Anthropomorphization and baseline testosterone in winners. In the winners, higher baseline testosterone was associated with lower slot machine anthropomorphization ($B = -1.07$, $p = .036$). This is illustrated in Figure 3.6 by plotting values for Anthropomorphization at 1 SD above and below the grand mean (Aiken & West, 1991; Cohen, Cohen, West, & Aiken, 2003). In the equivalent model with cortisol as the dependent variable, cortisol baseline and reactivity were neither associated with Anthropomorphization (intercept $B = .00022$, $p = .76$; slope $B = .000002$, $p = .85$) or the Anthropomorphization $\times$ Outcome interaction (intercept $B = -.00077$, $p = .76$; slope $B = .000022$, $p = .21$).
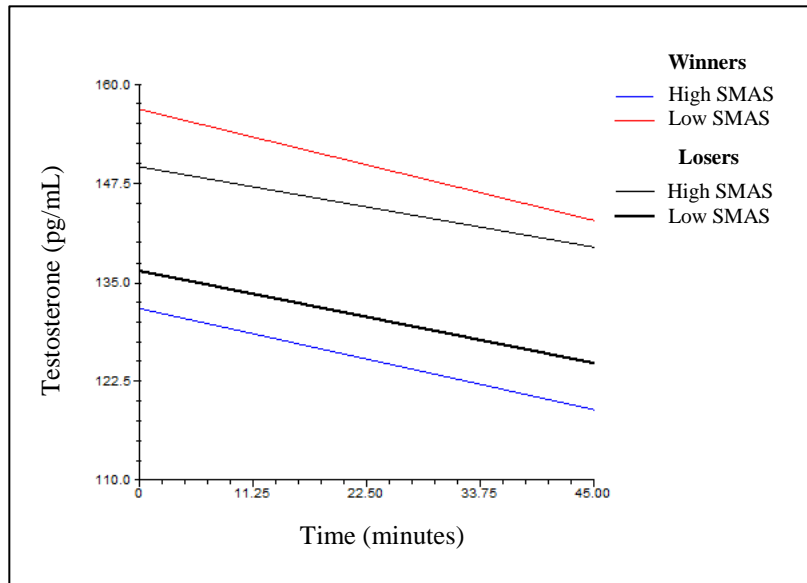
Figure 3.6 Testosterone reactivity by gambling outcome and SMAS score

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. However, this model indicated a significant interaction between Outcome and Anthropomorphization, for baseline testosterone. In the next section, I considered whether subjective appraisals of gambling experiences clarified the relationships seen in previous models, between gambling outcomes and testosterone reactivity.

3.4   Effects of Subjective Gambling Experiences on Hormone Fluctuation

As winners and losers differed substantially in their gambling experience (GEQ; feelings of competence, tension/annoyance) and post-gambling mood ratings (PANAS-X; positive and negative affect), a further set of post-hoc models were run to explore whether the GEQ and PANAS-X ratings predicted testosterone fluctuation. Each rating scale and the interaction terms with Outcome were entered in separate models. With respect to testosterone reactivity, higher

ratings of flow (B = -.16, p = .009), immersion (B = -.16, p = .002), tension (B = -.14, p = .038), and positive affect (B = -.023, p = .006) following slot machine play predicted steeper testosterone declines (Negative Affect, B = -.0062, p = .45; Challenge, B = -.087, p = .16; Competence, B = -.15, p = .10). The interaction terms between subjective experiences and gambling outcome were all non-significant (all ps > .099).

Gambling outcomes (winning) and testosterone reactivity were thus separately associated with positive affect, and positive affect has previously been found to mediate testosterone fluctuations following winning and losing outcomes (Mazur & Lamb, 1980; McCaul et al., 1992; Zilioli & Watson, 2012). Based on this prior literature, participants' mood in this study may have indirectly mediated a link between the outcome of the slot machine session and the change in testosterone levels. A supplementary analysis tested this relationship using ordinary least squares path analysis with bootstrap confidence intervals (Preacher & Hayes, 2004). Potential covariates (section 2.6) were included based on the procedure described in section 2.7, which, for this analysis, yielded a significant association between sexual orientation and testosterone fluctuation. Controlling for this variable, the outcome of the gambling session appeared to indirectly influence testosterone reactivity (based on area under the curve with respect to increase; Pruessner, Kirschbaum, Meinlschmid, & Hellhammer, 2003) through its effect on positive affect. Confirming the results from the earlier models, this mediation analysis showed that gambling wins predicted higher positive affect (a = 4.59, p = .006, 95% CI [1.38, 7.79]), and positive affect was negatively associated with testosterone change (b = -17.90, p = .011, 95% CI [-31.67, -4.12]). For the indirect effect, the confidence interval (50,000 bootstrap samples) did not capture zero (ab = -80.07, 95% CI [-201.16, -15.32]). Independent of this indirect effect, gambling outcomes were not associated with testosterone reactivity (c' =104.34, p = .40, 95% CI

[-138.87, 347.55]). Controlling for participant sexual orientation did not inordinately influence these results.

Together, these results show that, despite the absence of a clear winner-loser effect, higher positive affect (endorsed by winners) indirectly mediated steeper declines in testosterone during the study. In other words, positive affect in this study is implicated as an indirect mediator of a *reverse* winner-loser effect.

3.5   Effects of Testosterone and Cortisol on Gambling Behaviour

3.5.1   Preliminary Analyses of Behavioural Data

Due to a computer hardware malfunction, spin press behavioural data was unavailable for 20 participants. The behavioural analysis used level-1 data for 445 time bins, for 89 participants. Visual inspection of these data revealed a nonlinear trend over time, such that participants tended to increase their speed of play initially, with a subsequent plateau that could reflect fatigue or boredom (see Figure 3.7 Figure 3.8). Results from the step-up procedure for determining best model fit (see Figure 3.9) revealed that a quadratic link function had the lowest AIC value, and thus spin press behaviour was modeled quadratically over time.

The unconditional model of spin press behaviour was corrected for over-dispersion. Coefficient estimates for this model indicated that the slope ($B = .068$, $p = .35$) and acceleration ($B = -.025$, $p = .14$) terms were not significant, but the variance estimates for intercept ($\sigma_0^2 = .42$, $p < .001$), slope ($\sigma_1^2 = .52$, $p < .001$, and acceleration ($\sigma_2^2 = .12$, $p < .001$) were significant. Thus, participants maintained a stable pace of play overall, but the pace of play trajectories varied significantly and to an extent that substantiated my use of hierarchical nonlinear modeling for predicting these effects. Exploration of potential covariates of gambling behaviour (see section
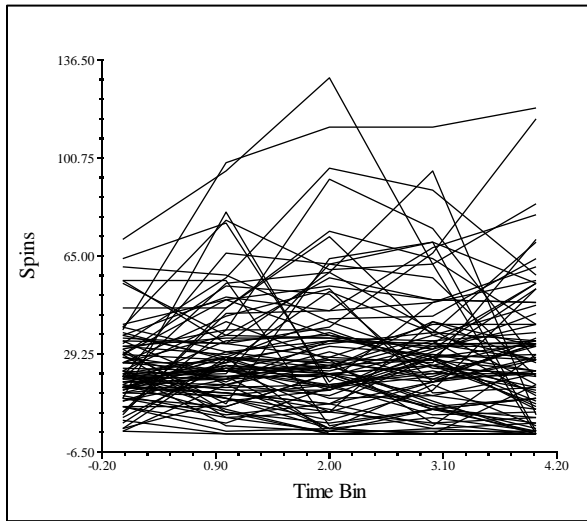
Figure 3.7 Individual play rate trajectories during the gambling session for all participants
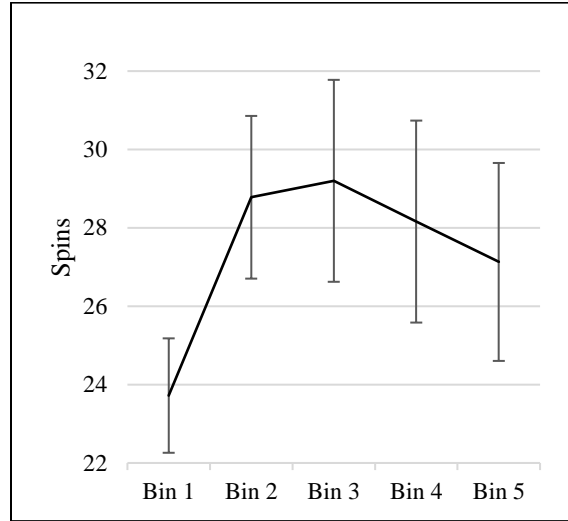

Figure 3.8 Average number of bets during each 3-minute epoch of the gambling session
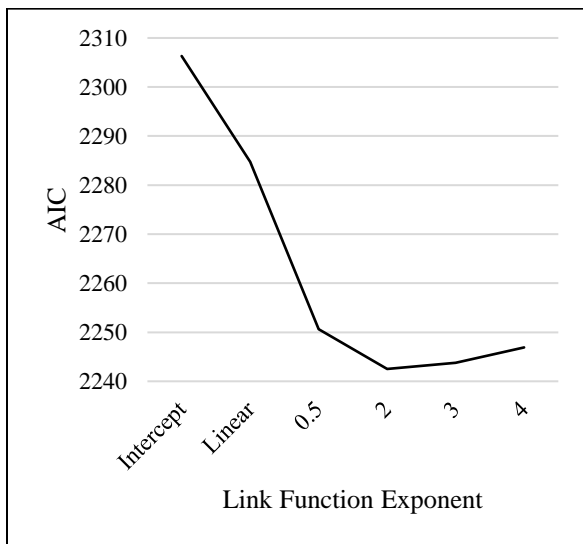

Figure 3.9 Play rate model fit depicted by AIC values for increasing polynomial degrees
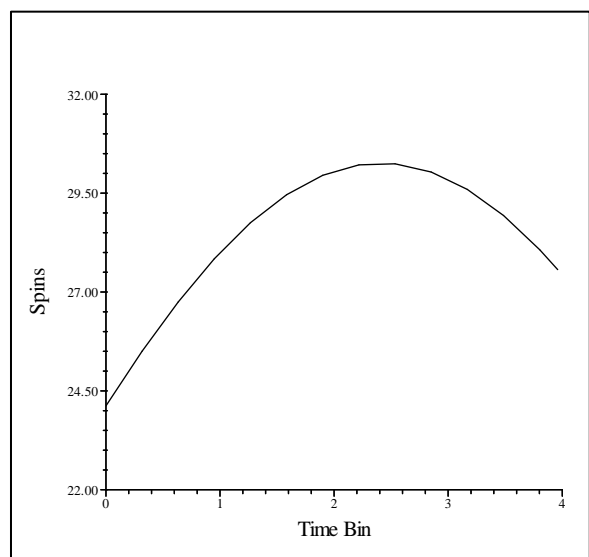

Figure 3.10 Fitted quadratic function for play rate during the gambling session

59

2.7) suggested that participants' initial rate and slope of betting in this study were not predicted

by their initial endowment amount, gambling competitiveness, endorsement of gambling-related

cognitive distortions, problem gambling severity, general tendency to anthropomorphize, and

anthropomorphization of the slot machine.

As can be seen below in Figure 3.11, winners and losers appeared to have distinct styles

of play on the slot machine. Outcome was not significantly associated with rate of play at the

start of the session ($B = -.19$, $p = .13$), but was associated with the slope of the pace of play ($B = -.17$, $p = .003$). This indicates that winners and losers began with similar play rates,
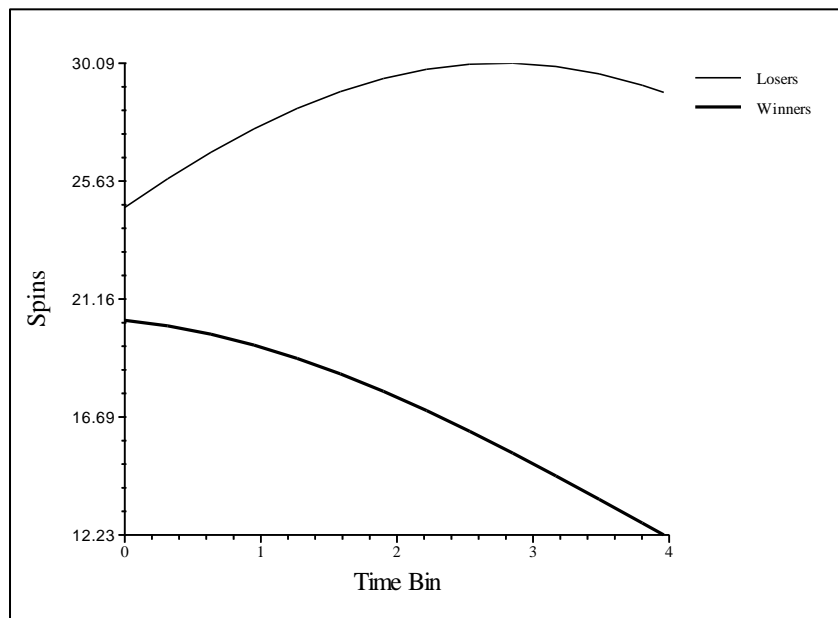

Figure 3.11 Play rate changes over time for winners and losers

then adopted different play strategies. To further elucidate this divergence in strategy, I

controlled for the effect of discontinuing gambling early (a strategy chosen by 11 participants

invariably to win the cash bonus) on slope ($B = -.64$, $p < .001$). In doing so, the relationship

between play rate slope and Outcome was no longer significant (B = -.081, p = .095), indicating that the association between play rate slope and Outcome was driven partly by participants who won altering their rate of play after winning credits above their initial endowment. This provides some evidence for the directionality of the association between Outcome and slope, that the winning and losing paradigm influenced participants' rate of play.

3.5.2   Effects of Testosterone and Cortisol on Rate of Bets Placed

Hypothesis 4 predicted a positive association between testosterone levels and slot machine play behaviour. This could be manifested in two ways: by observing basal testosterone levels to positively predict (1) higher rates of play overall, indicated partly by a positive effect on intercept of the play rate growth model, and/or (2) an increase in the rate of play throughout the gambling session, reflected by a positive effect on slope of the play rate growth model. Controlling for the influence of the winning-losing paradigm on gambling behaviour, Outcome was included in this model, along with the Outcome × Testosterone term, to test whether baseline testosterone was positively associated with rate of play, overall and in either winners or losers.

As can be seen from the hierarchical nonlinear regression model for rate of play (see Table 3.6), baseline testosterone was negatively associated with intercept of participants' betting trajectories (B = -.0035, p = .035), indicating that, because the association between testosterone

and slope was not significant ($B = .00072$, $p = .27$), higher testosterone levels were associated with a slower pace of play, overall (see Figure 3.12)[3]. Because this effect was not in

	Rate of Betting		
Fixed Effects	Estimate	SE	p
Intercept, π_{0i}			
Intercept	3.118188	.058925	< .001
Outcome	-.547505	.392162	.166
Testosterone	-.003479	.001629	.035
Outcome × Testosterone	.002601	.002589	.318
Slope, π_{1i}			
Intercept	.073848	.073120	.315
Outcome	-.179704	.220156	.417
Testosterone	.000722	.000653	.272
Outcome × Testosterone	.000038	.001539	.980
Acceleration, π_{2i}			
Intercept	-.027273	.017088	.114
Random Effects	Estimate		p
Level 1			
Within-person (σ_ε^2)	3.70491		—
Level 2			
Intercept (σ_0^2)	.17715		< .001
Slope (σ_1^2)	.27927		< .001
Acceleration (σ_2^2)	.01439		< .001
Covariance, σ_1^0	.01467		—
Covariance, σ_2^0	-.00619		—
Covariance, σ_2^1	-.05838		—

Table 3.6 Effects of Outcome and baseline testosterone on play rate during gambling

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[3] Following Mehta et al. (2015), cortisol and the testosterone × cortisol interaction were added to the previous model. A dual-hormone interaction was not supported in this model (all fixed effects ps > .098).
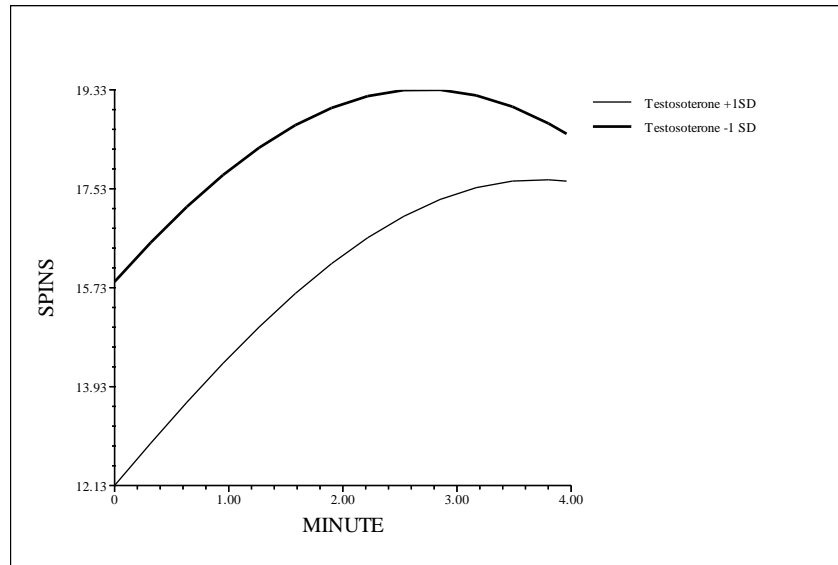
Figure 3.12 Modeled betting rate trends for baseline testosterone levels one standard deviation above and below the mean, controlling for Outcome

the predicted direction, hypothesis 4 was not supported. Outcome $\times$ Testosterone interactions were not significant for play rate intercept ($B = .0026$, $p = .32$) and slope ($B = .000038$, $p = .98$), showing that there were no associations between testosterone and play rate trajectory that were specific to either winners or losers.

Chapter 4: Discussion

This study represents an attempt to clarify the relevance of basal and fluctuating testosterone levels in slot machine gambling, as the predominant form of casino gambling and most common form of gambling among individuals with gambling problems. To this end, I examined testosterone levels in winners and losers of a 15-minute period of gambling on an authentic slot machine, to test for the winner-loser effect that is classically observed in social competitions. Two potential moderators of a winner-loser effect were also investigated. First, considering recent evidence for a dual-hormone hypothesis, salivary cortisol was measured along with testosterone, to evaluate whether winners and losers experienced divergent effects on testosterone that were dependent on cortisol status. Second, anthropomorphization of the slot machine was investigated as novel moderator of a winner-loser effect in gambling situations that involve wins and losses but without direct inter-individual competition. Lastly, I examined the association between baseline testosterone levels and slot machine player behaviour, reflected in the speed of slot machine spin presses over the session of play. The experiment also assessed various demographic and personal measures, as well as measures of gambling involvement, to both control for potential confounds of the quasi-experimental win-loss paradigm, and to clarify mediating roles of subjective gambling experiences in modulating testosterone levels of winners and losers.

The results of this study indicate a negligible effect of winning or losing outcomes following a 15-minute period of gambling, on testosterone fluctuation. Although winners and losers were comparable on measures of gambling attitudes, cognitive distortions, bio-demographic variables, and baseline hormone levels, the predicted winner-loser effect on testosterone levels was not observed. In other words, the winning or losing outcome did not

explain significant variability in participants' testosterone reactivity during the procedure. Similarly, this variability was not explained by baseline cortisol status or anthropomorphization tendencies, when those variables were added to separate hierarchical linear regression models alongside the winning/losing outcome. Thus, hypotheses 1 through 3 were not supported: a winner-loser effect was not observed, and was not moderated by cortisol levels or the tendency to humanize the slot machine. Two unexpected findings did emerge from the analyses testing these three hypotheses. First, winners and losers differed in positive and negative affect on subjective ratings (higher positive affect in winners), and higher positive affect predicted stronger *declines* in testosterone. Second, a negative association between baseline testosterone and slot machine anthropomorphization was present in winners, but not in losers.

Additionally, analysis of participants' slot machine spin behaviour revealed a negative association between baseline testosterone and the overall rate of slot machine play. In these analyses, participants appeared to adopt slower or faster styles of play in winning and losing conditions, respectively, which may have served a strategic aim to maximize their likelihood of winning the cash bonus (effectively, "If I'm ahead, I should slow down as this machine is stacked against me"). After controlling for the participants who adopted this strategy, higher testosterone appeared to predict a *slower* pace of play, indicating a safer gambling strategy in this study. Thus, although a relationship between pace of play and testosterone was found, the observed results were in the opposite direction of that predicted.

The following sections discuss some possible interpretations for the results observed in this study. I take an integrative approach to these discussions, wherein the conclusions drawn about a winner-loser in slot machine gamblers are discussed in light of competing interpretations that are supported and refuted by the data produced in this study and by the existing literature

more generally. In the same way, I also discuss the observed relationship between baseline testosterone levels and slot machine gambling behaviour. Next, the limitations of this research are presented, followed by suggestions for both mitigating these limitations and expanding this research in future studies.

4.1   Slot Machine Gambling: Evidence for a Winner-Loser Effect?

Hypothesis 1 predicted that winning versus losing during slot machine gambling would produce a classic winner-loser effect on testosterone, such that testosterone increased for winners and decreased for losers. In previous research, there is actually some flexibility in how the winner-loser effect can be expressed on testosterone changes. For example, one study (Zilioli & Watson, 2012) found that winners (on a 2-player computer Tetris competition) experienced *stable* testosterone levels, while losers experienced a testosterone decrease; this was interpreted as a classic winner-loser effect being superimposed on the natural diurnal decline of the hormone. However, neither display of divergent effects on testosterone from winning and losing were supported in this study. No significant association was found between the win/loss outcome of participants' gambling sessions, and the linear trajectories of testosterone change over three sampling periods within a 45-minute window. Rather, testosterone declined for all participants during this period, and did so equally for winners and losers. This result was observed despite several precautions based on prior boundary conditions from the literature. The ecological validity of the gambling setting was strengthened by the use of an authentic slot machine and quasi-experimental winning-losing paradigm; several potential confounds of testosterone fluctuation (e.g., subjective experience, cortisol state, etc.) were monitored and/or controlled for; a respectively large sample was taken in comparison to previous studies wherein a winner-loser

effect has been seen, and the data were modeled using a highly sensitive HLM strategy that is more appropriate for analysis of steroid hormone fluctuation than ANOVA, which has been used previously in this literature.

Clearly, the absence of any clear differences in testosterone between winners and losers may indicate that engagement in slot machine gambling is not sufficient to trigger testosterone change. This is also in line with a previous study monitoring testosterone during a poker competition (Steiner, Barchard, Meana, Hadi, & Gray, 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. For instance, the winner-loser effect has been elicited in sports fans watching matches (Bernhardt et al., 1998), and in sports players who watch personal videos of their own previous achievements (Carré & Putnam, 2010), but in both cases, these procedures involve witnessing a traditional inter-individual competition, which may generate a vicarious or imagined sense of involvement. Subjective appraisals of involvement and one's performance as having contributed to the win are known mediators of the winner-loser effect (González-Bono et al., 1999; van Anders & Watson, 2007), which could have been lacking in our design. We approached this issue via the role of anthropomorphization; the tendency to humanize the slot machine did vary among our participants, and there was further evidence of concurrent validity between the generalized trait measure (IDAQ) and the slot machine-specific state measure of anthropomorphization (SMAS). Nonetheless, anthropomorphizing the slot machine did not reliably predict testosterone reactivity, suggesting that perceived intentionality may be insufficient to engender an actual sense of competition against the machine.

Other interpretations rest on the possibility of a Type 1 error, that our design may have missed a true effect of slot machine outcomes on testosterone. First, testosterone reactivity was

modelled linearly, when in effect, the winner-loser effect's divergent testosterone trajectories for winners and losers could be nonlinear, in which case the ability of the hierarchical linear models to detect a winner-loser effect would have been impacted[4]. Second, the monetary prize awarded to winners may have been insufficient to elicit a winner-loser effect. Although participants' initial endowment was displayed by the machine in credits, so as to distance this number from the dollar amount, winners may have still recognized that the cash bonus was a fraction of their starting amount, signaling a net *loss* of money. Third, a 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, Gladue, and Joppa (1992) provide the only extant evidence that chance-based (i.e., non-competition) wins and losses can cause a winner-loser effect. One important departure between their work and the current study is the involvement of the experimenter in the win-loss paradigm: McCaul et al. had an experimenter initiate coin tosses that would cumulatively determine whether their participants would win or lose a $5 cash bonus, whereas the experimenter in the current study was only present to give instructions before the gambling session and award or withdraw the cash bonus afterward. In McCaul's study, the experimenter's presence may have substantiated a competitive experience that was not replicated in this study's solitary win-loss paradigm. Taken together, results from previous research and the current study

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[4] The winner-loser effect was also tested using nonlinear models, for which results on the effect of Outcome on testosterone reactivity corroborated the corresponding null effects produced by the linear models. Modelling the winner-loser effect nonlinearly resulted in smaller *p* values for models containing Outcome singly ($B = -.39$, $p = .26$) and Outcome with Cortisol ($B = -1.32$, $p = .13$), but as for the linear models, no terms were statistically significant.

suggest that a classic winner-loser effect may depend upon a subjective experience of competition against other people.

Some evidence in the present study pointed to a *reverse* winner-loser effect in slot machine gambling. Positive Affect (an established mediator of testosterone increases in winners of competitions; Chichinadze, Lazarashvili, Chichinadze, & Gachechiladze, 2012; Salvador & Costa, 2009) following the slot machine session was naturally higher in winners than losers, and indirectly mediated a *decrease* in testosterone. Because this mediational effect of Positive Affect was specific to winners, these results suggest that winners experienced a reverse winner-loser effect. Reverse winner-loser effects have been observed in some prior studies (e.g., G. Oliveira et al., 2013, 2014), especially in competitions in which the winning or losing outcomes are ambiguous or unexpected (i.e. surprising) (Zilioli et al., 2014). In what they call the 'Status Instability Hypothesis,' Zilioli and colleagues propose that reverse winner-loser effects—which are not easily explained by the Biosocial Model of Status—result from wins and losses that occur within unstable social hierarchies. Unstable social hierarchies are produced when competition outcomes are close or uncertain; this conveys that the present hierarchy status may be illegitimate or not clearly established, and that opportunities for status attainment are more abundant (Zilioli et al., 2014). In this case, losers of close or uncertain dominance competitions should benefit from increased persistence and status seeking behaviours, which are behaviours that are promoted by increases in testosterone. Zilioli et al. (2014), and more recently, Wu, Eisenegger, Zilioli, Watson, & Clark (2016), provide evidence that supports the Status Instability Hypothesis. Wu et al. showed that both cortisol levels and the closeness (clear versus narrow) of wins and losses of Tetris competitions, moderated change in testosterone. 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 clearly have imbued a strong sense of uncertainty about winning and losing during the gambling session in this study. Furthermore, although the number of winners and losers were close in proportion (63 losers, 50 winners), net slot machines scores had a mean in the loss range (-742.55 credits), suggesting that on average the winning outcomes occurred by a narrower margin than losses. Accordingly, a majority of participants in this study experienced either clear losses or narrow wins, which could explain why testosterone was seen to decline on average. In other words, participants in this study who should have experienced increases in testosterone according to the Status Instability Hypothesis (clear winners and narrow losers), were greatly outnumbered by participants who experienced clear losses and narrow wins (expected to decrease testosterone).

In summary, our null results for testosterone change can be reconciled with a classic winner-loser effect by drawing on a number of design factors and boundary conditions. Rather, we observed an indirect mediational relationship between Positive Affect after winning and a decrease in testosterone, in line with a reverse winner-loser effect. Based on these findings, it is possible that testosterone fluctuations caused by slot machine gambling depend jointly on whether one wins or loses, and on subjective appraisals of the closeness of the outcome.

4.2   Proposed Moderators of the Winner-Loser Effect

Both basal cortisol and anthropomorphization of the slot machine were initially proposed to moderate a winner-loser effect in this study (hypotheses 2 and 3, respectively). Since a clear winner-loser effect was not seen as per hypothesis 1, hypotheses 2 and 3 were modified to consider cortisol and anthropomorphization as additional control variables in hierarchical linear

models of the winner-loser effect. In doing so, I predicted that a winner-loser effect would emerge after controlling for the variance associated with these variables. Contrary to this prediction, neither the inclusion of cortisol or anthropomorphization helped to reveal a winner-loser effect.

Consistent with prior research, this study found that testosterone and cortisol levels were moderately correlated. Hypothalamic-pituitary-adrenal and gonadal axes, which regulate cortisol and testosterone production, respectively, function interdependently (see Johnson, Kamilaris, Chrousos, & Gold, 1992; Viau, 2002). Specifically, higher basal levels of either hormone can suppress secretion of the other into the bloodstream (Chen, Wang, Yu, Liu, & Pearce, 1997; Tilbrook, 2000; Viau & Meaney, 1996). Based on this premise, in the event that testosterone is being significantly produced in the body, a 'blunting' effect of high basal cortisol levels on testosterone should be seen. In the current study, participants' testosterone and cortisol both declined, on average, indicating low or suspended secretion of these hormones during the study. Accordingly, cortisol was not expected to moderate a winner-loser effect, and hypothesis 2 was subsequently modified to consider cortisol as a control variable. However, a winner-loser effect did not emerge after the addition of Cortisol and Cortisol $\times$ Outcome terms to the model previously used to test hypothesis 1. This further supported the notion that a classic winner-loser effect was not present in this study. Specifically, the non-significant Cortisol $\times$ Outcome term for testosterone reactivity indicated that baseline cortisol was not involved in a blunting effect on testosterone in either winners or losers.

Similar to hypothesis 2, hypothesis 3 positioned slot machine anthropomorphization scores within a hierarchical linear model of the winner-loser effect, in order to determine whether humanization of the slot machine would explain any variability in participants'

testosterone reactivity to winning or losing the gambling session. I predicted that, insofar as anthropomorphization would elicit the subjective experience of being in a competition with the machine, this variable would moderate a winner-loser effect. Contrary to this prediction, anthropomorphization was not associated with testosterone reactivity, despite evident individual differences among my participants on this dimension. One interpretation of this finding is that humanizing slot machines is not sufficient to create an actual competitive experience with the machine. As mentioned previously, subjective feelings of involvement in a competition appear to be integral to eliciting winner-loser effects, so a diminished competitive experience as reflected by a null association between anthropomorphization and testosterone reactivity, may explain why including this variable in models of testosterone change did not reveal a winner-loser effect. However, it is equally plausible that anthropomorphizing the machine conferred a feeling of competing with the machine, but that other aspects of solitary gambling (see above) prevented the discovery of any discernable associations between anthropomorphizing the machine and testosterone. As with basal cortisol, further research is necessary to establish a winner-loser effect in solitary gambling contexts, before anthropomorphism can be adequately tested as a moderator of this effect.

This study identified an outcome-moderated association between baseline testosterone and anthropomorphization of the slot machine. What the results showed was that, among winners only, higher baseline testosterone was associated with *lower* anthropomorphization of the slot machine. One tentative way to interpret this finding relates to the cash prize in the winning condition. 'Monetary priming' similar to this procedure has been shown to promote feelings of independence and a reduced need for social connection (Vohs, 2015; Vohs, Mead, & Goode, 2006), which is one of two internal motivating factors for anthropomorphizing non-human

entities (sociality motivation, Epley, Waytz, Akalis, & Cacioppo, 2008; Epley, Waytz, &

Cacioppo, 2007). If receiving a cash prize in the winning condition reduced this motivating

factor for anthropomorphizing, then individual differences in the other motivating factor,

effectance motivation (a desire to understand the behaviour of other agents), should predict

anthropomorphization of the slot machine among winners. Although effectance motivation was

not directly evaluated in this study, baseline testosterone may be negatively associated with this

factor; power motivation—a trait consistently linked with both increased testosterone levels and

multiple behavioural correlates of high testosterone (see Stanton & Schultheiss, 2009)—can

promote changes in social information processing in ways that reduce motivation to seek socially

explanatory information (Fiske & Dépret, 1996). Based on this premise, it is possible that high-

testosterone (reflecting high power motivation) winners were less inclined to pursue a social (i.e.,

anthropomorphic) explanations for why the slot machine granted wins, than low-testosterone

winners (with low power motivation). Thus, after receiving a cash prize, high-testosterone

winners would be expected to anthropomorphize the slot machine less.

4.3   Testosterone and Pace of Play

Hypothesis 4 predicted a positive relationship between baseline testosterone levels and

risky gambling behaviour, which was operationalized in this study as higher pace of play. This

follows from the premise that placing a bet on a slot machine is an inherently risky choice. Thus,

betting at an increased rate was considered a riskier strategy in this study. A preliminary analysis

of the behavioural data indicated that wins and losses were associated with different trajectories

for pace of play. Although the correlational nature of hierarchical linear models prohibits

inferences about causation, an intuitive explanation for the association between gambling

outcomes and pace of play trajectories was that the winning-losing paradigm likely influenced

gambling behaviour. This interpretation assumes that participants were motivated to win the cash

bonus. If so, their scores at any point likely influenced pace of play; because they had a finite

time to reach a net positive score to win the cash bonus, participants whose scores dropped

below the initial endowment amount could have strategically increased their pace of play (i.e., a

'nothing-to-lose' strategy). Conversely, winners who recognized the 'house edge' of the slot

machine may have strategically slowed their pace of play once they had achieved a net-positive

score.

Holding constant the influence of the winning-losing paradigm on pace of play

trajectories, higher testosterone significantly predicted an overall slower or less-risky pace of

gambling. Prevailing research has indicated—in contrast to our result—that testosterone and

risk-taking behaviours tend to be positively correlated (see, Apicella, Carré, & Dreber, 2015;

Mehta et al., 2015). However, there are several inconsistencies in this literature. Multiple studies

have found null associations between risk-taking and naturally occurring testosterone levels

(Derntl et al., 2014; Rosenblitt, Soler, Johnson, & Quadagno, 2001; van der Loos et al., 2013)

and high-normal exogenous doses (Boksem et al., 2013; Ortner et al., 2013; Zethraeus et al.,

2009). Furthermore, one study has identified clear negative associations between testosterone

and several measures of sexual risk-taking attitudes (van Anders, Goldey, Conley, Snipes, &

Patel, 2012). As van Anders et al. explain, situations wherein risk-aversive behaviours may be

beneficial to attaining higher social status may promote reversed testosterone-risk associations.

With regard to sexual risk-taking, safer sex attitudes may be positively associated with

testosterone because safer sex practices are generated not from risk aversion (i.e., consequences,

including sexually transmitted infections, are far removed from the behaviour), but from desires

to attain or display social status (e.g., buying contraceptives) (van Anders et al., 2012). A similar explanation may be relevant to the current findings. It is possible that higher-testosterone participants were more inclined to take advantage of a low-cost opportunity achieve higher status (winning the cash bonus) by using a less-risky gambling strategy. The choices to initiate or continue gambling generally connote risk-taking, however, these choices were constrained in this study to control inconsistencies in gambling experiences; participants were instructed to gamble and did not risk their own money. In other words, the win-loss paradigm may have produced a gambling scenario wherein a clear risk-averse strategy could lead to winning money, and higher testosterone promoted the use of this strategy because it was associated with status attainment.

Interestingly, the dual-hormone hypothesis has been extended to explain inconsistent findings regarding testosterone and risk-taking (Mehta et al., 2015) and other status-oriented behaviours (Mehta & Josephs, 2010). However, cortisol did not moderate the negative association between testosterone and rate of play in this study, potentially suggesting that the current finding is either spurious, or not a straightforward testosterone-risk relationship.

4.4 Further Limitations

Several limitations in this study are worthy of discussion. First, 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. On one hand, the quasi-experimental determination of wins and losses provided a realistic experience, which is known to be an important aspect of the winner-loser effect in the context of sporting competitions. Previously, a meta-analysis has revealed that sports field studies of the winner-loser effect produced larger effect sizes than laboratory studies (Geniole et al., 2017). On the other hand, the slot machine

produced random sequences of wins and losses for each participant, which may have influenced winner-loser effects on testosterone. Specifically, the closeness or ambiguity of cash bonus wins and losses could not be controlled or manipulated. Given that this dimension of subjective experience has been shown to influence the directionality of the winner-loser effect (e.g., Wu et al., 2016), a possibility in this study was that classic and reversed winner-loser effects were confounded or cancelled each other out. Variability in gambling experiences produced by the machine—specifically, frequencies and durations of both win/loss streaks and bonus features— may have resulted in inconsistent experiences among winners and losers; for example, some participants may have had gambling sessions characterized predominantly by continual losses or wins, or a mixture of wins and losses. Lastly, despite participants being equal at baseline on several important biological and demographic variables, the correlational nature of the study design and data analytic strategy, precluded inferences about causation.

The foremost aim of this study was the identification of a winner-loser effect in slot machine gamblers. Thus, priority was given to methods that support this aim. For instance, variability in gambling experiences was reduced, and salient wins and losses were produced, by implementing a simplified winning-losing paradigm that restricted participants' betting strategies and reward possibilities. One drawback to this was that the binary 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 gambling behaviour in ways that are inconsistent with authentic gambling settings. Essentially, gambling in the current study differed from typical slot machine gambling in the risk of losing one's money, and in receiving rewards or losses of variable amounts. Furthermore, betting behaviour was constrained to fixed bet amounts (i.e., 40 cents per spin), and to a maximum rate of play if participants were unfamiliar with the slot

machine 'skill stop' feature (i.e., the ability to stop the reels early). Because of these limitations in gambling experiences, caution is warranted in generalizing the current findings to other gambling settings.

Lastly, an import limitation of this study concerns the participants sampled, who were mostly novice, non-problem gamblers. Indeed, many participants were likely playing a slot machine for the first time. It is possible that this sampling characteristic was associated with different gambling attitudes and play styles, in comparison to regular gambling involvement, or to problem gambling. For example, Riva, Sacchi, and Brambilla, (2015) found that regular gamblers anthropomorphized a slot machine more than non-regular gamblers. Differences such as this may inform a higher likelihood of finding, for instance, a moderation effect of anthropomorphization of a slot machine on testosterone fluctuation, in regular gamblers.

4.5   Conclusions and Future Directions

This study was the first to investigate whether slot machine wins and losses could produce a winner-loser effect on testosterone. An examination of testosterone changes revealed that winners and losers did not differ in their testosterone levels after a 15-minute period of slot machine gambling, in contrast to what was predicted according to the winner-loser effect. Thus, this research contributes to a growing number of studies that refute this effect in situations wherein one has little influence over their wins and losses. However, this study also showed that higher positive affect indirectly mediated decreases in testosterone, which may suggest that slot machine gambling promotes *reverse* winner-loser effects. As a follow-up to this finding, a subsequent study can examine whether more rigorous control over gambling session conditions will elicit this effect clearly. For example, according to Wu et al. (2016) and Zilioli et al. (2014),

narrow or uncertain wins and losses should produce decreases and increases in testosterone, respectively. If this is true, I predict that the winning-losing paradigm used in this study should elicit testosterone decreases (increases) in winners (losers) of gambling sessions that include i) slot machine scores that consistently fluctuate above and below the bonus prize cutoff (conveying an uncertain outcome), and ii) a final score that is very near to the prize cutoff (indicating that outcome was narrow or close). Similarly, a classic winner-loser effect may be elicited by wins and losses characterized by consistent winning or losing streaks. Investigating these different winner-loser effect boundaries in gambling settings could be achieved by 'rigging' both wins and losses, and winning and losings streaks.

Another line of inquiry could investigate associations between testosterone and gambling strategies when laboratory constraints on gambling behaviour are *lessened*, rather than increased. In the present study, a negative association between baseline testosterone levels and rate of play was found after controlling for the influence of the binary winning-losing paradigm. In a future study, switching from this binary determination of winning and losing to a persistence paradigm (which allows the player to choose when to stop gambling), would allow a number of additional risk-related variables to be tested against baseline testosterone levels. This methodological strategy could help to clarify the negative association seen in this study. For example, recent research has found that individual differences in testosterone positively correlate with persistence on an unsolvable puzzle task (Welker & Carré, 2015). If this association translates to gambling, individual differences in testosterone may be implicated in riskier gambling strategies, as characterized by longer periods of play. Furthermore, by allowing participants control over their bet amounts, a similar relationship can be explored between testosterone and patterns in bet changing.

In conclusion, this thesis has sought to examine aspects of slot machine gambling that affect and can be affected by testosterone, which is a hormone supported by separate literatures in its ability to both promote risk-taking and be influenced by wins and losses. Although minimal associations were observed between gambling outcomes and hormone fluctuation, this study adds to a growing body of research that clarifies the conditions necessary for producing the winner-loser effect. Furthermore, extensions of this research may yet uncover specific gambling conditions that elicit predictable testosterone changes, which may be relevant to predicting changes in gambling strategy. Furthermore, this study revealed an association between individual differences in baseline testosterone and gambling strategies, which implicates basal testosterone as a relevant factor in gambling related decision-making. Further research is recommended to disentangle the bidirectional relationships between testosterone and slot machine gambling.

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