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Psychophysiological responses to wins, losses, and losses disguised as wins during gambling on electronic machines: from laboratory tasks to live gambling

Lisa Lole
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Psychophysiological responses to wins, losses, and losses disguised as wins during gambling on electronic machines:

From laboratory tasks to live gambling

A thesis submitted in fulfilment of the requirements for the award of the degree

DOCTOR OF PHILOSOPHY

From
UNIVERSITY OF WOLLONGONG

by
Lisa Lole, BSc (Hons)
School of Psychology
2013

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Declaration

I, Lisa Lole, declare that this thesis, submitted in fulfilment of the requirements for the award of Doctor of Philosophy in the School of Psychology, University of Wollongong, is entirely my own work unless otherwise referenced or acknowledged. This document has not been submitted for the purpose of a degree or qualification at any other academic institution.

Lisa Lole

16 December 2013
Abstract

Problem gambling is characterised by maladaptive gambling patterns, resulting in severe psychological, interpersonal, and financial problems. Electronic gaming machines (EGMs, also called a ‘poker’ or ‘slot’ machines) are one of the most harmful gambling forms and are associated with higher risk, greater severity, and faster progression of problem gambling. The physiological arousal (perceived as excitement) caused by the experience of unpredictable positive reinforcement (wins) during EGM play has been posited to be a primary motivator for normal gambling activity, and dysfunctional incentive value processing has been implicated in the development of problem gambling behaviours. Specifically, hypersensitivity to reward in problem gamblers may selectively reinforce gains, but not discourage the effect of losses.

Alternatively, problem gamblers may be hyposensitive to punishment, or hyposensitive to both reward and punishment, leading to compensatory thrill-seeking behaviours, such as gambling. Previous studies examining incentive processing in problem gambling have reported mixed results and the nature of deficit in this disorder remains unresolved. The primary aim of the current doctoral thesis was to investigate whether problem gamblers display abnormal psychophysiological responses to gambling outcomes. A variety of psychophysiological and psychometric measures were used to examine how naive gamblers, experienced regular gamblers, and problem gamblers process different magnitude wins, losses, near-wins, and losses disguised as wins.

Ambulatory equipment was used to measure electrodermal and cardiac activity in order to examine the effect different gambling outcomes have on arousal levels in a sample of healthy controls (n = 23) while they played a computer gambling task, and in samples of problem (n = 15) and non-problem gamblers (n = 15) gambling their own money on an EGM in a licensed club. Cortical event-related potentials (ERPs) were recorded from
healthy controls \((n = 17)\) while they played a computer gambling task, and analysed using a principal components analysis, to examine the effects on key indices of reward and punishment processing, namely the P300 and the feedback-related negativity (FRN). These measures were also examined to determine whether they could successfully distinguish problem \((n = 16)\) from non-problem gamblers \((n = 20)\). Results from this series of studies indicate that outcomes of differing valence (good vs. bad) and magnitude (small vs. large) can be reliably captured and quantified using psychophysiological measures, and that responses to these outcomes can successfully differentiate problem gamblers from non-problem gamblers. A major finding is that, compared to healthy controls, problem gamblers were consistently less responsive to both reward and punishment stimuli, as evidenced by both cortical and autonomic responses. These findings suggest that dysfunction in reward-processing pathways of the brain may, at least partly, explain the aberrant behaviours associated with problem gambling. The thesis contributes significantly to the theoretical conceptualisation of the psychophysiology of gambling, helps clarification among the competing mechanisms suggested to underlie this disorder, and establishes a firm foundation to inform future research including the determination if a pattern of maladaptive incentive processing could serve as a reliable biological marker for problem gambling.
Publications Constituting this Thesis

Published Manuscripts


Manuscripts Submitted for Peer Review

Statement of Verification

This statement verifies that the greater part of the work in the manuscripts named above is attributed to the candidate, Lisa Lole. This thesis was associated with a funded Australian Research Council-Linkage Grant, LP0776836. Although some experiments of the study were designed for the Grant proposal, the candidate contributed to the review and evaluation of other design aspects. The candidate took primary responsibility for all data collection and analysis, prepared the first draft of each manuscript, and prepared the papers for submission to relevant journals. Co-authors contributed to the thesis by providing guidance on the design and structure of each study, and provided editorial suggestions for every paper and for the thesis.

Lisa Lole, PhD Candidate

Craig J. Gonsalvez, Primary Supervisor

Alex Blaszczynski, Secondary Supervisor

16 December 2013
Conference Abstracts arising from this Thesis


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List of Abbreviations

Δ – Difference
µS – microSiemen
µV – microVolt
ACC – Anterior cingulate cortex
Ag/AgCl – Silver/silver-chloride
APA – American Psychiatric Association
AMS – Ambulatory Monitoring System
AUD – Australian dollars
BAS – Behavioural Activation System
BIS – Behavioural Inhibition System
CPGI – Canadian Problem Gambling Index
DSM – Diagnostic and Statistical Manual of Mental Disorders
EGM(s) – Electronic gaming machine(s)
ERN – Error-related negativity
ERP – Event-related potential
fMRI – functional magnetic resonance imaging
FRN – Feedback-related negativity
FRP – Feedback-related positivity
FRR – Feedback-related response
HC – Healthy control
HR – Heart rate
I₇ – Impulsiveness questionnaire
List of Abbreviations (continued)

IBI – Inter-beat interval
IGT – Iowa Gambling Task
LDW – Losses disguised as wins
LPC – Late positive complex
OFC – Orbitofrontal cortex
OT1 – Outcome Time 1
OT2 – Outcome Time 2
PCA – Principal components analysis
PG(s) – Problem gambler(s)
PGSI – Problem Gambling Severity Index
SCL – Skin conductance level
SCR – Skin conductance response
SF – Spatial factor
TF – Temporal factor
VMPFC – Ventromedial prefrontal cortex
Overview of the Empirical Studies in this Thesis

The current thesis is comprised of four studies in which psychophysiological reactions to gambling outcomes were examined. Two of these studies are in the autonomic nervous system domain, and the other two investigated central nervous system reactivity to manipulations of outcome valence and magnitude.

The first study reported within this thesis (Study A/Chapter Four) sought to establish whether psychophysiological responses following outcomes commonly encountered during electronic gaming machine (EGM) gambling could be reliably captured and quantified. Autonomic nervous system activity of naive gamblers \((n = 23)\) was examined for gambling outcomes, including wins, losses, and outcomes similar to losses disguised as wins (LDWs, where the amount returned is less than the amount bet), near-wins (these outcomes are similar to losses disguised as wins because the symbols ‘nearly’ match up to create a winning combination; however, these outcomes are not associated with monetary return or exciting auditory stimuli), following decisions to bet high or low during play on a computer-simulated EGM task.

Electrodermal activity, but not heart rate (HR), was found to successfully differentiate the stimulus valence (good/reward vs. bad/punishment) and the magnitude (small vs. large) of reward. Wins elicited greater skin conductance responses (SCRs) than losses and near-wins, and larger wins produced greater SCRs than smaller wins. Thus, we were able to support the contention that the motivational significance of gambling events is reflected in psychophysiological responses of healthy controls within a controlled laboratory setting.

Unlike Study A, Study B was a field study and examined phasic electrodermal activity of problem gamblers in response to wins and losses, as well as responses to actual LDWs, while they gambled on EGMs in licensed gaming venues (Study
B/Chapter Five). Electrodermal activity following reward was found to differentiate problem gamblers \((n = 15)\) from non-problem gamblers \((n = 15)\). For non-problem gamblers, wins elicited greater SCRs than losses; however, problem gamblers demonstrated significantly reduced SCRs following wins. LDWs did not produce SCRs different to losses for either problem or non-problem gamblers.

Study C (Chapter Six) sought to clarify the latent nature of two event-related potential (ERP) components previously shown to index incentive processing, the feedback-related negativity (FRN) and the P300. A spatiotemporal principal components analysis (PCA) was used to determine whether these components could reliably differentiate between EGM outcomes (wins, losses, and near-wins). Data recorded while healthy naive gamblers \((n = 17)\) played a computer EGM task revealed win outcomes elicit a frontally maximal feedback-related positivity (FRP) (that was also sensitive to reward magnitude), whereas losses elicit a feedback-related negativity (FRN) at the same topography and latency. This finding implies that the neural system/s that generate these feedback-related brain responses are differentially activated by positively and negatively valenced stimuli. The results also revealed that the P300 component observed in previous research (that was greater for wins compared to losses) was likely to be a P3b subcomponent. Near-wins were found to elicit significantly smaller FRN amplitudes than losses (no difference in P3b amplitude was found), suggesting that, while these outcomes are not perceived to be rewarding, they may be less averse than losses.

The final study (Study D/Chapter Seven) sought to determine whether abnormal ERP responses to reward and punishment stimuli are able to differentiate problem gamblers, and whether these objective responses correlate with self-reported levels of impulsivity, attraction to appetitive stimuli, and avoidance of aversive stimuli. Problem
(n = 16) and non-problem gamblers (n = 20) played a computer EGM task, and the latent correlates of incentive processing observed in Study C were examined. Problem gamblers exhibited both attenuated FRN and FRP amplitudes following wins and losses, respectively, whereas P3b amplitudes did not differentiate problem gamblers from non-problem gamblers for valence manipulations. Problem gamblers also tended to display anomalous reward magnitude processing, with similar P3b amplitudes following large and small wins (although this result was only approaching significance and therefore, requires further verification). The neural responses of problem gamblers to near-wins did not differ from those of non-problem gamblers. Individual differences in attraction to appetitive and aversive stimuli, as assessed by self-report questionnaires, were found to be potentially suitable indicators of underlying neural processes.

In summary, the psychophysiological measures employed in the empirical chapters of the current thesis were found to be reliable indices of incentive processing for EGM gambling stimuli. While winning appeared to be exciting and motivationally significant for non-problem gamblers, problem gamblers were found to be less responsive to both win and loss outcomes. Although the studies presented in this thesis could not determine the direction of causality for this hyposensitivity to reward and punishment/non-reward stimuli (i.e., whether it is a reflection of an inherent dysfunction in cortical reward processing pathways, or the result of repeated exposure to gambling activity, or other factors), these results have important implications for conceptualisations of the nature of deficit in disordered gambling, and may explain why affected individuals gamble for longer periods of time and with larger amounts of money despite repeated losses. This pattern of maladaptive incentive processing presents a potential marker for addiction and provides a foundation for future research into this complex disorder.
CHAPTER ONE – The Phenomenology of Gambling Behaviours

1.1. Description of Gambling and Disordered Gambling

Gambling is defined as an intentional act of placing an item of value on an unpredictable outcome, one that is to some extent determined by chance (Bolen & Boyd, 1968; Wildman, 1997). Gambling is a popular pastime for many Australians and most adults have gambled at some stage in their lifetime (Delfabbro, 2012); it is estimated that 70% of the adult population have participated in some form of gambling in the last year, with around 17% doing so on a regular basis (i.e., at least once a week) (Davidson & Rodgers, 2010; Productivity Commission, 2010). For the majority of these individuals, gambling provides a form of relatively harmless entertainment, with no significant negative consequences. Unfortunately, it is estimated that around 0.7% of the Australian adult population have developed a harmful addiction to gambling activity, and a further 1.7% of the population are at risk for developing an addiction (Productivity Commission, 2010). Moreover, prevalence rates are greatly elevated among regular gamblers compared to the general population (estimates range from 15-50% of regular gamblers) (Centre for Gambling Research, 2004; Productivity Commission, 2010), suggesting that the large amount of harm that is caused by this disorder is experienced more acutely by a subsection of the general population. Alarmingy, youth estimates of this disorder are much higher, with around 3% of young Australians experiencing gambling related problems (Delfabbro, Lahn, & Grabosky, 2005; O’Neil, Whetton, & Duerrwald, 2003).

Disordered gambling is defined in the Diagnostic and Statistical Manual of Mental Disorders (5th ed.; DSM-5; American Psychiatric Association, APA, 2013) as a persistent, recurrent, and problematic pattern of gambling behaviour that disrupts
personal, familial, and/or vocational pursuits (see Raylu & Oei, 2002 for a comprehensive overview of the harms associated with disordered gambling), and is not better accounted for by another psychiatric disorder (particularly mania). A number of comorbid mental disorders are more likely to be experienced by problem gamblers compared to the general population, including personality disorders, anxiety disorders, and substance use disorder (see Shaffer & Korn, 2002 for a review). For every individual afflicted with this disorder, it is estimated that another five to ten people are negatively affected as a consequence of their problematic behaviours (Productivity Commission, 2010). While quantifying the costs on the general quality of life associated with this disorder is difficult, from an economic perspective, problem gambling is conservatively estimated to cost the Australian economy around AUD $4.7 billion annually (Productivity Commission, 2010).

Disordered gambling was previously termed pathological gambling and classified in the DSM (e.g., DSM-III-TR, APA, 1987; DSM-IV-TR, APA, 2000) as an impulse control disorder. Since then, evidence supporting this disorder as an impulse control disorder appeared in the literature, due to the seemingly impulsive behaviours demonstrated by afflicted individuals, such as gambling despite negative consequences and a previously expressed desire to refrain from gambling. Recently, this disorder was reclassified as an addictive disorder (DSM-5, APA, 2013) due to the observation of many similarities shared with substance use disorder, including the experience of withdrawal symptoms, cravings, tolerance, repeated self-destructive behaviours, response to treatment medications, comparable neural activation to addiction-based stimuli (as indicated by neuroimaging research), similar neurotransmitter dysregulation, as well as a high comorbidity rate between the two disorders (see Blaszczynski, Walker, Sharpe, & Nower, 2008; Wareham & Potenza, 2010).
The psychological and economic costs that cascade down from the affected individual, to their family and the wider community, represent a major public health concern. The vast majority of gamblers who seek help for their addiction do so after serious adverse consequences have occurred (for example, many people seeking treatment have reported past attempts at suicide; Ledgerwood, Steinberg, Wu, & Potenza, 2005; Petry & Kiluk, 2002). Moreover, despite major problems eventuating, it is estimated that only 7-29% of problem gamblers have ever sought professional help (Suurvali, Cordingley, Hodgins, & Cunningham, 2009), often doing so only in response to pressure from concerned family members and not in recognition of the severe problems caused by their gambling (Ladoucer, 2002; Raylu & Oei, 2007; see also Ledgerwood, Arfken, Wiedemann, Bates, Holmes, & Jones, 2013). For these reasons, the prevention, early identification, and treatment of disordered gambling is vital to the success of any initiative seeking to ease the burden experienced by affected individuals, their families, and the wider community as a result of this disorder.

1.2. Quantifying and Classifying Problematic Gambling Behaviours.
A number of different terms have been used to describe individuals exhibiting maladaptive gambling behaviours. These include the term given by the DSM-5 (APA, 2013), disordered gamblers; the term used in previous editions of the DSM (e.g., DSM-IV, APA, 1994; DSM-IV-TR, APA, 2000), pathological gamblers; compulsive gamblers, which is commonly used in treatment programs, such as Gamblers Anonymous; and problem gamblers, which has sometimes been used to describe conditions of a less severe nature (Walker & Dickerson, 1996).

The Problem Gambling Severity Inventory (PGSI) of the Canadian Problem Gambling Index (CPGI) (Ferris & Wynne, 2001, Appendix A) was used in the current
dissertation to group participants based on their level of risk and the severity of gambling-related problems. The PGSI is comprised of nine questionnaire items that assess an individual’s ability to control their gambling behaviours, and the frequency (i.e., ‘never,’ ‘sometimes,’ ‘most of the time,’ or ‘almost always’) they experienced health-related, financial, and/or psychological problems in the previous twelve months as a result of their gambling activity. (The CPGI also includes an additional 20 items that relate to social aspects of gambling behaviour. These may serve as a screening tool to indicate an increased risk for problem gambling; however, only the nine items that form the PGSI used to estimate diagnostic status were completed by participants in the current body of research).

Individuals scoring above eight on the PGSI are classified as problem gamblers. Individuals classified as healthy controls or non-problem gamblers in the empirical chapters of this dissertation were those participants who did not meet this criterion (i.e., who scored less than eight on the PGSI). Problem gambling, as defined by the CPGI, is conceptually equivalent to the diagnostic criteria for pathological gambling outlined in the DSM-IV-TR (APA, 2000). It is a more conservative measure of gambling prevalence than the South Oaks Gambling Screen (Lesieur & Blume, 1987; Stevens & Young, 2008), but less conservative than the DSM-IV criteria (Ferris & Wynne, 2001), and has been shown to have adequate reliability and validity parameters (Ferris & Wynne, 2001). The CPGI was chosen for use in the current thesis due to the fact that it was specifically designed and clinically validated for use in the general population, rather than solely for clinical treatment populations (as was the development of DSM criteria). This was particularly important in the current body of research, which included participants who were drawn from a broad cross-section of the community and varied in their exposure to gambling activities, including novice undergraduate students with no
particular interest in gambling, experienced non-problem gamblers, and individuals with reportedly severe and debilitating gambling problems. An advantage of using the self-report CPGI over clinical diagnosis of problem gambling by a health professional is that it provides a reliable means of identifying problem gamblers in a quick, confidential, and anonymous manner (thus, avoiding the problem of symptom under-reporting commonly associated with socially undesirable behaviours, e.g., Sudman, 2001).

Consistent with threshold scores on the CPGI, the terms problem gambling and problem gamblers will be used henceforth in this dissertation to refer to relatively severe manifestations of the disorder and will be preferred over terms such as compulsive and pathological gamblers.

1.3. **Gambling on Electronic Gaming Machines.**

There are many forms of gambling, including instant scratch-its (‘scratchies’), lotteries, track gambling, sports betting, bingo, EGMs, Keno, and casino-style gambling (e.g., card games, such as Black Jack and Poker, dice games, such as Craps, and table games, such as Roulette), as well as online gambling, and ‘quasi-gambling’ activities, such as investing in the stock market. The most common and problematic type is arguably gambling on EGMs (also known as poker machines or ‘pokies’ in Australia, ‘fruit machines’ in the U.K., or ‘slot machines’ in North America). Electronic gaming machines are often thought to be an electronic equivalent of the older mechanical spinning-reel slot machine versions. The reels on traditional mechanical slot machines would actually physically spin and wins could occur depending on whether certain symbol combinations appeared when the reels stopped. On the original Liberty Bell version, these machines had three spinning reels, with ten symbols on each (Fey, 1975), meaning only 1000 combinations were possible, and that the probability of winning the
jackpot on each spin (i.e., where all symbols matched) was one in 1000 (or 0.001). Wins on modern EGMs are also determined by certain combinations of winning symbols, with both earlier and modern machine types operating on a random variable ratio schedule of reinforcement; however, this is where the similarities between these two gambling mediums end. Outcomes on technologically-sophisticated, computer-operated EGMs are determined by random number generation, and the number of symbols on the ‘reels’ of modern EGMs are not restricted by physical parameters, meaning that there can be up to 256 different symbols available on each reel. As a result, the odds of having the best-paying symbol line up in each of the columns and winning the major prize on these machines are usually no better than one in 10 million (or 0.0000001); thus, there are many more loss combinations possible than winning ones. Moreover, despite faulty gambling-related cognitions and superstitions, these machines are non-strategic (although, players may control the amount bet and the number of lines played on each spin).

While certain types of casino gambling and wagering present an opportunity for large amounts of money to be placed on individuals bets, EGMs allow bets to be placed in rapid succession where available credits (funds) and fatigue are the only limiting factors, with current legislation in New South Wales, Australia, limiting wagers to be placed at a maximum of one bet every 3.5 seconds. However, the ‘downtime’ between bets is often filled with exciting auditory and visual stimuli, and accumulating credits when non-loss outcomes occur. Current legislation requires an individual EGM to theoretically return 85-92% of the amount played on it over an infinite number of trials (see Department of Families, Housing, Community Services and Indigenous Affairs, 2012). If this amount is returned in a given gambling session in which $1 is played on each individual spin, up to $120 can be lost every hour. Some machines allow players to
bet AUD $10 per spin, meaning that much larger amounts of money can be lost in a very short period of time. (The actual amount that can be spent on each individual machine depends on a range of factors, such as the speed of play, and the number of lines and credits wagered; Livingstone & Woolley, 2008; see also Responsible Gambling Council, 2006). EGMs are estimated to account for over half of all expenditure on gambling activity (Productivity Commission, 2010). On average, it is estimated that each regular EGM player (those playing at least once a week) gambles AUD $7,000-8,000 each year on this form of gambling (Productivity Commission, 2010).

The relationship between EGMs and problem gambling behaviours is convincing and well-established (Productivity Commission, 2010). However, it remains somewhat unclear as to the extent the design of these machines (e.g., Dixon, Harrigan, Sandhu, Collins, & Fugelsang, 2010), their wide availability (Lund, 2009; Productivity Commission, 2010; Responsible Gambling Council, 2006; Volberg, 2000), or other complex environmental factors, contributes to the harm caused by this form of gambling (Abbott, 2006, 2007; Abbott, Volberg, Bellringer, & Reith, 2004; Shaffer, LaBrie, & LaPlante, 2004; see Storer, Abbott, & Stubbs, 2009 for a thorough discussion on this point). The majority (75-84%) of individuals presenting for treatment for problem gambling, as well as individuals considered to be at a high risk for developing this disorder, report EGMs as their preferred gambling medium (Abbott, 2006; Productivity Commission, 2010). It is estimated that approximately 15% of regular EGM gamblers (those who gamble at least once weekly) meet the diagnostic criteria for problem gambling, and an additional 15% of regular EGM players are at moderate risk of developing this disorder. Furthermore, the majority of EGM revenue purportedly comes from those afflicted by this disorder; it is estimated that problem gamblers contribute to
around 22-60% of the total expenditure on EGM gambling, which, in 2010, was around AUD $2.6 billion (Productivity Commission, 2010).

1.3.1. **Electronic gaming machines: Winning, losing, and almost winning.**

Since the majority of problem gamblers report experiencing problems with EGM gambling, it is of value to examine whether certain machine design features contribute to the great appeal of this gambling medium. Gamblers grossly underestimate the amount of money lost during EGM play; research has shown that, on average, only 3% of the actual amount lost is recalled and/or reported (Productivity Commission, 2010), and that EGM gambling is associated with erroneous estimations of total expenditure (Blaszczynski, Ladouceur, Goulet, & Savard, 2008). Apart from the fact that EGMs do not generally issue receipts for the amount of money spent on them (although, some machines do provide player information through the use of loyalty cards, or through selecting the session tracking functions available on certain machines), another factor likely to contribute to this under-approximation of expenditure are outcomes often encountered during EGM play, ‘losses disguised as wins’ (LDWs, Dixon et al., 2010). These are outcomes in which the amount returned is less than the amount bet; for example, when a 50 cent wager is made and only 30 cents is returned, resulting in a net loss of 20 cents. When wins occur on EGMs, auditory jingles and flashing symbols (which highlight the winning combination) are presented, and the machine gradually accumulates credits won on that particular spin. Losses, in contrast, are associated with a state of relative quiet, with no flashing visual or auditory feedback. Despite the fact that LDWs, which are a salient design feature of EGMs, are technically losses, they are associated with visual and auditory stimuli similar to what follows wins; thus, these outcomes are believed to deceive players into believing that they are winning when they are not and to encourage sustained gambling behaviours (Dixon et al., 2010).
Previous autonomic research by Dixon et al. (2010) found that LDWs elicit similar skin conductance responses (SCRs) to wins in naive gamblers during laboratory-based EGM gambling. Near-wins are different from LDWs and are outcomes in which nearly all the symbols required for a win are presented but another mismatching symbol prevents the win from being obtained. Near-wins do not yield any credits and have also been examined in previous research. Neuroimaging research has shown that these outcomes recruit reward-related brain circuitry in naive gamblers during gambling in laboratory conditions (Clark, Lawrence, Astley-Jones, & Gray, 2009).

Electrophysiological research has shown near-wins elicit greater P300 amplitudes (Qi, Ding, Song, & Yang, 2011) and smaller feedback-related negativity (FRN) amplitudes than losses (Luo, Wang, & Qu, 2011), suggesting these outcomes are more rewarding and less punishing than ‘full-loss’ outcomes, despite no credits being returned. While it has been previously suggested that LDWs contribute to the addictive potential of EGMs (Dixon et al., 2010; Livingstone & Woolley, 2008), the responses of problem gamblers to LDWs has not yet been examined. The current thesis examined the physiological reactions of problem gamblers, regular non-problem gamblers, and naive gamblers to LDWs and near-win outcomes in order to investigate the role these outcomes play in the development and maintenance of normal and problem gambling behaviours; specifically, it investigated whether individuals are primarily motivated by the monetary incentives that occur following these outcomes (i.e., by the small but tangible ‘increase’ in credits), their associative value (i.e., by the attendant visual and auditory stimuli similar to what normally accompanies wins), or whether they are actually responding to the significance of nearly winning (i.e., to the symbols ‘almost’ matching up). In order to test these possibilities, near-wins were presented to participants in Studies A (Chapter Four), C (Chapter Six), and D (Chapter Seven).
These outcomes were not associated with any credits returned (to investigate whether it is the monetary gain, or the significance of nearly winning, that drives the physiological responses to these outcomes) or with any auditory jingles or flashy visual stimuli. Study B (Chapter Five) examined the responses of both problem and non-problem gamblers to true LDWs during actual EGM gambling in licensed gaming venues.
CHAPTER TWO – The Nature of Deficit in Problem Gambling

2.1. Theoretical Explanations of Normal and Problem Gambling Behaviours

Several theories attempting to explain the factors that motivate people to gamble have been posited in the current literature, the principles derived from which are often used in the treatment of problem gambling. Wildman (1997) provides a summary of a number of such accounts, including those that claim individuals gamble in order to achieve wealth (Snyder, 1975), to participate in recreational play (Herman, 1976; Kusyszyn, 1990; Smith & Abt, 1984), to aid in social interactions (Rosecrance, 1988), to resolve some sort of psychodynamic conflict (e.g., Freud, 1928), in response to genetic predispositions that affect the propensity to seek the excitement caused by gambling (Boyd, 1976), or due to the attraction to unpredictable intermittent reinforcement schedules (Knapp, 1976).

Other theories that have a focus on behavioural (e.g., Brown, 1986; McConaghy, 1980; Zuckerman, 1979) and cognitive-behavioural (e.g., Blaszczynski & Nower, 2002; Sharpe, 2002; Sharpe & Tarrier, 1993) concepts implicate autonomic arousal, perceived as the excitement associated with gambling activity, as the fundamental motivator for gambling behaviours. According to such models, problem gamblers are either hypersensitive to reward (e.g., Sharpe & Tarrier, 1993), or are generally hypoaroused and use gambling activity to increase arousal levels (e.g., Brown, 1986; Jacobs, 1986).

More recently, sophisticated neurobiological accounts that focus on incentive value processing have been proposed, which have the potential to explain both normal and problem gambling behaviours (e.g., Blum, Sheridan, Wood, Braverman, Chen, Cull, & Comings, 1996; Blum et al., 2000; Damasio, 1994; Holroyd & Coles, 2002). Such accounts implicate the abnormal evaluation of, and/or the learning about, environmental stimuli as key to understanding the aberrant behaviours exhibited by
problem gamblers. The *reward deficiency syndrome* hypothesis (Blum et al., 1996; Blum et al., 2000) posits that individuals with impulsive, addictive, and compulsive disorders (including problem gamblers and individuals with substance use disorder) are hyposensitive to reward due to reduced functioning of dopamine D₂ receptors in the mesolimbic reward system of the brain. During normal gambling, wins are hypothesised to induce a cascade of neurotransmitter activity that results in the release of dopamine from the nucleus accumbens. This dopamine is normally released into the synapse, where it stimulates several dopamine receptors (D₁ to D₅), resulting in feelings of pleasure associated with experiencing reward; however, when there is some form of dysfunction in this series of events (particularly in dopamine transmission, cf. Blanco, Orensanz-Munoz, Blanco-Jerez, & Saiz-Ruiz, 1996), perhaps caused by certain genetic variants, the affected individual will be less likely to experience feelings of well-being. These individuals are thought to engage in thrill-seeking behaviour (such as trying to obtain larger wins) to compensate and achieve a threshold of normal functioning.

The *somatic marker hypothesis* (Damasio, 1994) posits that the tendency for certain individuals to choose short-term gratification over long-term advantages, despite otherwise normal functioning and intelligence, is due to deficit in the ventromedial prefrontal cortex (VMPFC) (Damasio, 1994) and/or the orbitofrontal prefrontal cortex (Lodge, 2011). Accordingly, these individuals are unable to associate appropriate emotional reactions (termed *somatic markers*) to the physiological responses that occur following both positively and negatively valenced events, and therefore, cannot generate and use such markers for effective and efficient decision making. The difficulties experienced by problem gamblers may be the result of a deficit in the VMPFC, making them less adept at processing the incentive value of both reward (win) and punishment (loss) stimuli. Thus, the pleasure a non-addicted person feels toward wins would not be
experienced as strongly by problem gamblers, and losses would not be experienced to be as displeasing.

Although not originally intended to explain addictive or impulsive behaviours, the assumptions of the reinforcement learning theory (Holroyd & Coles, 2002) also have the potential to elucidate the nature of deficit in problem gambling through examination of the feedback-related negativity (FRN) event-related potential (ERP) component. Similar to the error-related negativity (ERN) that is elicited following errors on reaction time tasks (Falkenstein, Hohnsbein, Hoorman, & Blanke, 1991; Gehring, Goss, Coles, & Donchin, 1993; Miltner, Braun, & Coles, 1997), this ERP component is believed to index activity of the mesolimbic-dopaminergic system. Activation in this cortical pathway is believed to be associated with the evaluation of events according to previous learning experiences. The FRN is believed to reflect the disinhibition in the anterior cingulate cortex (ACC) caused by decreased dopamine transmission in the basal ganglia following the evaluation of environmental stimuli as ‘bad’ or ‘worse-than-expected.’ Various studies have found that the ACC is closely involved with the generation of the ERN and FRN ERP components (Bellbaum & Daum, 2008; Donamayor, Marco-Pallares, Heldmann, Schoenfeld, & Munte, 2011; Luu, Tucker, Derryberry, Reed, & Poulsen, 2000; Miltner et al., 1997; Nieuwenhuis, Slagter, von Geusau, Helenfeld, & Holroyd, 2005; Potts, Martin, Kamp, & Donchin, 2011; Yu & Zhou, 2009; Zhou, Yu, & Zhou, 2010). Activity of the ACC is hypothesised to be reflected in greater FRN amplitudes following negative compared to positive outcomes (although there has recent contention regarding the nature of this component; see Section 2.2.2. and Chapter Six). Involvement of the ACC in decisions to continue to gamble following the experience of non-reward has received some support from neuroimaging research. Campbell-Meiklejohn, Woolrich, Passingham, and Rogers
examined neural activity using functional magnetic resonance imaging (fMRI) while healthy non-gamblers chose to ‘chase’ losses or to prevent further losses by ceasing gambling activity. Participants were advised that they were eligible to win a cash prize if they had the most credits left on a laboratory-based computer task compared to all participants at the completion of the experiment. On each trial, participants incurred one of five different magnitude losses to their pool of allocated credits. Participants were able to choose to accept the loss and continue to play, or they could choose a gamble option, where, if they were successful, they would have the originally deducted amount returned, but if they lost, they would incur a penalty of double the credits lost. Greater activation in the ACC was associated with the decision to quit gambling and to not recompense losses. Unfortunately, neural activity of problem gamblers was not examined in that study, so it remains unclear whether dysfunction of the ACC or other structures in the mesolimbic-dopaminergic system are associated with problematic gambling behaviours, particularly repeated gambling despite repeated and often severe losses. Moreover, the task used in that study did not resemble true gambling activity, with no opportunity to wager on an outcome or to experience wins (only reimbursement of deducted credits). Nevertheless, this study provides valuable insight into the role the ACC plays in the evaluation of negative outcomes, and subsequent effects on future decision making. The FRN component has been found to be a reliable indicator of incentive value; thus, abnormal FRN responses following loss outcomes may be used to indicate an inability to appropriately process and judge the value of environmental stimuli. This is particularly relevant for the study of problem gambling and warrants further investigation.
2.2. Are Problem Gamblers Abnormally Sensitive to Reward and/or Punishment?

Problem gamblers have been shown to exhibit a number of abnormalities in neural processing, including disrupted sensory gating (Stojanov, Karayanidis, Johnston, Bailey, Carr, & Schall, 2003) and impairment in various executive functions (see Goudriaan, Oosterlaan, de Beurs, & Van den Brink, 2004 for a review); however, as predicted by several theoretical positions outlined above, abnormal incentive processing may better account for the maladaptive behaviours displayed by these individuals (it should be noted, that research has not confirmed whether such impairments predate gambling problems). In order to test this conjecture, physiological indices of incentive processing in response to reward and punishment stimuli need to be examined. Since problem gamblers have been shown to underreport past gambling problems (Abbott, 2001), an advantage of using objective measures rather than self-report measures, is that they are less susceptible to deliberate and unintentional distortions.

Due to the many similarities shared between problem gambling and substance use disorder (see Section 1.1.), the extensive research that has been conducted on individuals with substance use disorder may provide valuable insight into the nature of deficit in problem gamblers. Substance use disorder is increasingly being understood in terms of anomalous incentive processing. Individuals with substance use disorder have demonstrated decreased P300 (Goldstein et al., 2008; Kamarajan et al., 2010; Porjesz, Begleiter, Bihari, & Kissin, 1987) and FRN amplitudes following reward and non-reward outcomes (Fein & Chang, 2008; Kamarajan et al., 2010). These individuals have also exhibited increased activation in cortical reward pathways following the presentation of addiction-based cues, but reduced activation following the experience of non addiction-based reward and punishment (see reviews by Ditchter, Damiano, &
Allen, 2012 and Volkow, Fowler, Wang, & Swanson, 2004). Anomalous assessment of reward magnitude has also been shown to characterise substance use disorder, with afflicted individuals attributing equal value to large and small magnitude rewards (Goldstein, Tomasi, Alia-Klein, Cottone, Zhang, Telang, & Volkow, 2007). It remains unclear whether such a pattern of dysregulated incentive processing also underlies problem gambling. Research investigating incentive value processing in problem gamblers using neuroimaging or psychophysiological methods is relatively scarce, and preliminary results are by no means consistent. Corroborating research on substance use disorder, neuroimaging research suggests that problem gamblers are hyposensitive to both the experience of reward and punishment (de Ruiter, Veltman, Goudriaan, Oosterlaan, Sjoerds, & van den Brink, 2009; Reuter, Raedler, Rose, Hand, Glascher, & Buchel, 2005; cf. Miedl, Fehr, Meyer, & Herrmann, 2010; van Holst, Veltman, Büchel, van den Brink, & Goudriaan, 2012). Autonomic nervous system research examining heart rate (HR) suggests that problem gamblers are hyposensitive to reward (Goudriaan, Oosterlaan, de Beurs, & Van den Brink, 2006). Electrophysiological research using ERPs implies that these individuals are hypersensitive to reward (Hewig, Kretschmer, Trippe, Hecht, Coles, Holroyd, & Miltner, 2010; Oberg, Christie, & Tata, 2011). Autonomic research examining electrodermal activity has found no difference in the skin conductance responses (SCRs) of problem gamblers and healthy controls following outcomes of varying incentive value (Goudriaan et al., 2006). A number of factors may contribute to these discrepant findings. One such factor is the type of paradigm employed in the different studies; for example, some studies have used stimuli that present clear instances of reward and punishment/non-reward (e.g., de Ruiter et al., 2009; Goudriaan et al., 2006; Oberg et al., 2011; Reuter et al., 2005), whereas others have used tasks that may primarily consider the anticipation rather than the actual
experience of reward (Hewig et al., 2010; Miedl et al., 2010; van Holst et al., 2012; see Section 2.3. for a further discussion of this point). The methods used to quantify physiological responses to stimuli may have also contributed to these mixed findings. For example, the study by Goudriaan et al. (2006) reported an increase in HR after wins and decrease following losses for the healthy control group, but a decrease after both win and loss outcomes for problem gamblers. However, HR responses were calculated by subtracting the third inter-beat interval (IBI) value after the outcome from the IBI value during the response to that outcome, not by considering the difference from a pre-stimulus baseline. It is also possible that the perceived value of the experimental stimuli may have contributed to the lack of a difference in SCRs between wins and losses for either healthy controls or problem gamblers found in that study. Thus, it is of value to determine whether problem gamblers respond differently to non-problem gamblers to the actual experience of wins and losses during realistic EGM gambling scenarios (something that is particularly relevant considering the potential for harm associated with this form of gambling), including when they gamble with their own money in licensed gaming venues.

2.2.1. Autonomic indices of reward and punishment processing.

As mentioned above (Sections 1.1. and 2.1.), gambling is associated with (at least some) feelings of excitement. This excitement is believed to be reflected in activity of the autonomic branch of the peripheral nervous system, particularly in electrodermal activity which reflects activation of eccrine sweat glands following physical activity, stressful situations, or cognitive-emotional processing (see Dawson et al., 2000). Functioning of this system has been shown to reflect cortical arousal (Barry, 1996; Barry, Clarke, McCarthy, Selikowitz, Rushby, & Ploskova, 2004). Autonomic
responses are generally thought to rely largely on subcortical activity, but several higher-order cerebral structures have also been implicated in the monitoring and regulation of autonomic function (Augustine, 1985; Augustine, 1996; Devinsky, Morrell, & Vogt, 1995; Jänig, 1995; Jänig & McLachlan, 1992). Neuroimaging research has shown that the regions of the brain involved in generating electrodermal activity, such as the orbitofrontal, cingulate, and insular cortices (Cechetto & Saper, 1990; Fredrikson et al., 1998; Nagai, Critchley, Featherstone, Trimble, & Dolan, 2004; Williams et al. 2000), the hypothalamus and brainstem (Critchley, Melmed, Featherstone, Mathias, & Dolan, 2001, 2002; Nagai et al., 2004), and the amygdala (Asahina, Suzuki, Mori, Kanesaka, & Hattori, 2003), are also associated with emotional and motivational behaviour (Critchley et al., 2002; Damasio, 1994); thus, autonomic responses are believed to reflect the salience of stimuli. Activity in areas believed to be closely implicated in decision making processes, such as the ventromedial prefrontal cortex and medial temporal lobe, has also shown to be positively correlated with electrodermal activity (Critchley et al., 2000; Nagai et al., 2004; Williams et al. 2000). Other event-related fMRI research has revealed abnormal functioning of these brain areas in subjects identified as problem gamblers (Potenza, Leung, Blumberg, Peterson, Fulbright, Lacadie et al., 2003), making an examination of the autonomic indices of incentive processing in these individuals particularly germane.

The majority of previous research using autonomic indices of arousal to examine problem gambling has employed tonic methods, from which the effects of differently valenced stimuli cannot be directly determined (see Section 4.3.2.). Phasic methods are better able to indicate the influence of individual win and loss events for both problem and non-problem gamblers; however, largely due to technological limitations, studies
using such measures in the current literature are scarce and have not been conducted outside of sanitised laboratory conditions.

2.2.2. Event-related potentials as indicators of reward and punishment processing.

Unlike substance use disorders, problem gambling is characterised by maladaptive behaviours and cognitions in response to immediate visual feedback (and perhaps, to a lesser extent, associated auditory stimuli). The functional and superior temporal information gained from recording cortical ERPs is well suited to the investigation of whether problem gambling is characterised by abnormal reward and punishment processing. Two separate ERP components in particular, the feedback-related negativity (FRN) and the P300, have demonstrated potential as reliable indicators of incentive processing.

2.2.2.1. The feedback-related negativity.

The FRN is an ERP component, traditionally conceptualised as a negative deflection occurring 250-350 ms that is greater following unfavourable compared to favourable feedback (Falkenstein, Hohnsbein, Hoormann, & Blanke, 1990, Gehring & Willoughby, 2002a; Miltner et al., 1997). This component is believed to reflect the monitoring and evaluation of ongoing performance; specifically, when there is a mismatch between internal and external representations (Holroyd & Coles, 2002). Neutral feedback elicits similar FRN responses as negative feedback, suggesting that this component reflects a classification process concerned with whether outcomes achieve goals or not (Holroyd, Hajcak, & Larsen, 2006). The amplitude of the FRN is sensitive to violations of expectancy, with greater responses elicited following unexpected negative feedback.
compared to expected negative feedback (Bismark, Hajcak, Whitworth, & Allen, 2013; Holroyd & Krigolsen, 2007; Wu & Zhou, 2009; cf. Castellar, Kuhn, Fias, & Notebaert, 2010; Hajcak, Holroyd, Moser, & Simons, 2005); such amplitude increases are associated with the magnitude of deviation from the actual and the expected outcome (Bellbaum & Daum, 2008). Greater FRN amplitudes have been observed when higher rewards were possible, further highlighting the influence of expectancy on this ERP component (Bellbaum, Polezzi, & Daum, 2010). The FRN has generally not been found to be sensitive to outcome magnitude (i.e., large vs. small), indicating that this component reflects a binary evaluation of outcomes (Gu, Huang, & Luo, 2010; Hajcak, Moser, Holroyd, & Simons, 2006; Sato, Yasuda, Ohira, Miyawaki, Nishikawa, Kumano, & Kuboki, 2003; Toyomaki & Murohashi, 2005; Yeung & Sanfey, 2004; Yu & Zhou, 2006a; cf. Donamayor et al., 2011; San Martin, Manes, Hurtado, Isla, & Ibanez, 2010; Yu & Zhou, 2009), although, some research has demonstrated that this component is sensitive to the magnitude of losses but not wins (Donamayor et al., 2011).

As mentioned above (Section 2.1.), the FRN was traditionally thought to be characterised by greater negative deflections to unfavourable compared to favourable outcomes (Falkenstein et al., 1990; Gehring & Willoughby, 2002). More recent research suggests that this component is better conceived as indicative of greater activation to reward, and constitutes larger amplitude positive deflections (a feedback-related positivity, FRP) following beneficial outcomes at the same topography and latency as the traditionally conceptualised FRN (Foti, Weinberg, Dien, & Hajcak, 2011; Holroyd, Nieuwenhuis, Yeung, & Cohen, 2003; Holroyd, Pakzad-Vaezi, & Krigolson, 2008; San Martin et al., 2010). Because most research on the FRN has used a difference waveform technique to quantify the FRN in response to experimental variables, the effects of
beneficial and unfavourable outcomes cannot be directly determined (see Sections 6.3 and 7.3).

2.2.2.2. The P300.

Commonly examined using the oddball paradigm (Donchin, Ritter, & McCallum, 1978; Pritchard, 1981), the P300 is a parietally-maximal (Johnson, 1993) positive deflection that peaks 250-500 ms (although this latency range may vary, depending on task requirements and instructions) and is elicited following the presentation of infrequent targets that are interspersed among frequent non-target stimuli. Although the neural generators of this component are somewhat unclear, it is generally believed that the temporal-parietal junction (Halgren, Squires, Wilson, Rohrbaugh, Babb, & Crandall, 1980; Johnson, 1988; McCarthy et al., 1989; Nieuwenhuis, Slagter, von Geusau, Heslenfeld, & Holroyd, 2005) is involved in producing the P300 ERP component; however, the wide topographical distribution of the P300 suggests that this cortical region is highly interconnected with other brain regions, or rather, that there are actually numerous generators of this component (Duncan, 2003; Nieuwenhuis et al., 2005; Pineda et al., 1989). The P300 has been shown to index various cognitive and attention processes, including, the subjective motivational significance of stimuli (Begleiter, Porjesz, Chou, & Aunon, 1983; Johnston, 1979; Sutton, Tueting, & Hammer, 1978), stimulus uncertainty (Sutton et al., 1965), subjective probability (Duncan-Johnson & Donchin, 1977; Tueting, Sutton, & Zubin, 1970), unexpected events (Sutton et al., 1978), confidence of a decision, and orienting response processes (Donchin, 1981). It is generally accepted that this ERP component reflects context updating and memory processes according to cognitive mental models (Donchin, 1981), although alternative accounts have been proffered (Gonsalvez, Barry, Rushby, & Polich, 2007; Mecklinger
Several researchers have demonstrated that the traditionally conceptualised P300 is actually a complex made up of several subcomponents (Ford, Roth, & Kopell, 1976; Friedman, Vaughan, & Erlenmeyer-Kimling, 1981; Squires, Donchin, Herning, & McCarthy, 1977; Squires, Squires, & Hillyard, 1975), which are characterised by different topographies, latencies, and responses to different experimental manipulations, including the P3b, P3a/novelty P3, no-go P3, and Slow wave components. These subcomponents are believed to form part of a larger inhibitory system that serves to control attention (reflected by the P3a) and facilitate memory processes (reflected by the P3b) (see Polich, 2007).

P300 amplitude has been shown to be dependent on an individual’s available attentional resources, with smaller P300 amplitudes and longer latencies elicited when processing requirements are more difficult. This has been demonstrated using dual-performance tasks, where the subject is required to perform a primary task while simultaneously counting target stimuli in a secondary oddball task. Results from such experiments have shown that, as the difficulty of the primary task increases, the amplitude of the P300 to the oddball in the secondary task decreases (Israel, Chesney, Wickens, & Donchin, 1980; Kramer, Wickens, & Donchin, 1985; Wickens, Kramer, Vanasse, & Donchin, 1983). The allocation of attentional resources is, in turn, believed to be dependent on state and trait levels of arousal (Kahneman, 1973; Kok, 1990; Pribram & McGuinness, 1975). A robust relationship between P300 amplitudes and alpha band spectral power further suggests that this ERP component is closely associated with arousal, attention, and memory processes (Basar et al., 1989; Intriligator & Polich, 1995; Jasiukaitis & Hakerem, 1988; Polich, 1997; Pritchard et al., 1985). Moreover, although the relationship between personality and the P300 is somewhat
unclear, individuals with lower levels of arousal, linked to individual differences in personality including extraversion/introversion, sensation-seeking, and impulsivity, have been found to elicit attenuated P300 amplitudes compared to individuals with higher arousal levels (Gurrera, O’Donnell, Nestor, Gainski, & McCarley, 2001; Stelmack & Houlihan, 1994; cf. Brocke, 2004; Brocke, Tasche, & Beauducel, 1997; De Pascalis, 2004; Stenberg, 1992). These findings may be caused by neurotransmitter dysfunction, reflected in less effective attentional resource capabilities (Hill et al., 1998, 1999; Polich & Criado, 2006).

The P300 has been shown to be a reliable indicator of incentive value processing in gambling research, with greater P300 responses observed following win compared to loss outcomes (Bellbaum & Daum, 2008; Bellbaum et al., 2010; Hajcak, Moser, Holroyd, & Simons, 2007; San Martin et al., 2010; Toyomaki & Murohashi, 2005; Wu & Zhou, 2009; Yeung, Holroyd, & Cohen, 2005; Zhou et al., 2010), even when the probability of occurrence is controlled for, and greater responses following larger magnitude compared to smaller magnitude outcomes (Homberg, Grunewald, & Grunewald-Zuberbier, 1981); however, it remains unclear as to what subcomponent is driving these differences. Since several theories postulate problem gambling to be the result of deficits in arousal, it is worthwhile examining whether problem gamblers elicit attenuated P300 amplitudes following gambling outcomes compared to healthy controls.

2.2.2.3. **Using principal components analysis to analyse event-related potentials.**

Despite the excellent temporal resolution of ERPs, the electrical activity generated by one cortical structure may be observed at various scalp topographies, causing componential overlap (Dien, 2012). Principal components analysis (PCA) overcomes
this problem by statistically separating individual ERP components from overlapping raw data. It involves extracting linear combinations of data points that fulfil certain criteria, differentiating consistent patterns of electrocortical activity (Dien & Frishkoff, 2005). PCA has been used to successfully parse the P300 from the positive-going Novelty P3 and Slow Wave subcomponents, which overlap in time at similar parietal topographies (Dien, Spencer, & Donchin, 2003; Spencer, Dien, & Donchin, 2001). Temporal PCA allows the reduction of data across time points (over all subjects, trial types, and recording sites), whereas spatial PCA allows such analysis across electrode sites (across all time points, participants, and trial types). The application of a PCA allows more time efficient and objective analysis of ERP data, without the confounding influences of overlapping components.

The application of a PCA to examine the FRN has the potential to elucidate the nature of this component; specifically, whether it is best conceptualised as a positive deflection to positive feedback, or as a negative deflection to negative feedback. To date, only one preliminary study has employed this analysis method to examine reward and punishment processing in healthy controls during play on a two-option guessing task (Foti et al., 2011). Research examining the latent nature of this ERP component in response to EGM gambling outcomes, particularly in problem gamblers, is lacking. Moreover, it is worthwhile determining which P300 subcomponent is driving the pattern of responses observed in previous research, in order to determine whether problem gamblers have deficits in attention or memory updating processing (e.g., Polich, 2007).
2.3. **Are Problem Gamblers Addicted to the Possibility, but not the Actual Experience of Reward?**

Previous neuroimaging (Miedl et al., 2010; van Holst et al., 2012) and electrophysiological (Hewig et al., 2010) research reporting a hypersensitivity to reward in problem gamblers has generally employed high risk Black Jack tasks. While such research provides valuable information, the nature of this gambling task may have influenced the results obtained (Oberg et al., 2011), and therefore definite conclusions regarding the nature of reward responsiveness in problem gambling cannot be made with certainty. In such paradigms, participants are required to compete with a computer opponent on a gambling card game where the aim is to reach a score of twenty-one. On each trial, the participant is dealt two cards that usually do not summate to the winning score, and thus, they are required to make a decision on whether to draw as additional card (i.e., to ‘hit’ or not). If the participant decides to hit and they do not exceed the twenty-one score limit, this is called a ‘no-bust’; however, if they hit and the card causes them to exceed the maximum score, this is called a ‘bust,’ and the participant loses that trial. While it is reasonable to assume that ‘bust’ outcomes are appropriately considered to be punishment/non-reward/loss outcomes, ‘no-bust’ outcomes merely indicate that the participant’s choice to draw an additional card did not result in a score exceeding the twenty-one score threshold; thus, the possibility of a future win remains (unless the computer opponent had a winning card combination) and they do not present clear instances of reward attainment. It is possible that problem gamblers are more excited by the prospect, rather than the actual experience of reward, and is reflected in the hypersensitive responses to no-bust outcomes observed in previous research (see Crockford, Goodyear, Edwards, Quickfall, & el-Guebaly, 2005; Volkow et al., 2004).
Thus, the question remains: does the previously reported reward hypersensitivity of problem gamblers apply to the actual experience of reward during EGM gambling?

2.4. Are People with Certain Personality Traits More Attracted to Gambling?
It has been previously suggested that people with certain dispositions and personality traits may be more inclined to participate in gambling research, and also more likely to develop problematic patterns of gambling behaviours. The current thesis sought to determine whether individual differences in reward and punishment sensitivity and in impulsive tendencies are related to and can predict the psychophysiological reactions to outcomes of varying incentive value. Such an investigation is of great pragmatic value to clinical settings. Among other potential uses, if psychophysiological measures prove to be reliable indicators of incentive processing then personality variables related to these measures may be capable of contributing to the assessment of underlying physiological processes. If problem gamblers demonstrate abnormal physiological reactions to gambling outcomes then such self-report psychological inventories may be used to indicate underlying deficits in incentive processing. Moreover, if future research proves that such psychophysiological responses preceed the development of problem gambling, then these inventories may potentially used as screening tools to help in the early detection and intervention of individuals at risk of developing severe problematic gambling behaviours.

2.4.1. Individual differences in sensitivity to reward and punishment.
It has been postulated that certain individuals may be more attracted to gambling activities due to inherently different propensities to seek out appetitive stimuli and/or to avoid punishing stimuli (Gray, 1991; Zuckerman, 1979). For example, if problem
gamblers continue to gamble despite negative consequences due to an abnormal attraction to reward, these individuals are likely to score higher on psychological inventories that assess reward sensitivity, such as the BAS subscale on the Behavioural Inhibition System/Behavioural Activation System (BIS/BAS) scale (Carver & White, 1994, Appendix B).

The BIS and the BAS were originally proposed to explain how neurobiological factors are reflected in an individual’s personality (Carver & White, 1994; Fowles, 1980; Gray, 1975, 1990); specifically, why some individuals are motivated to seek appetitive stimuli, whereas others appear to be more motivated by avoidance of averse stimuli. The BIS is believed to govern the avoidance of threatening stimuli by stopping ongoing behaviour, and is particularly sensitive to punishment or non-reward stimuli. High BIS scores are believed to be linked with states of enhanced attention, arousal, and vigilance, and very high BIS scores are believed to be associated with anxiety disorders (Fowles, 1988; Quay, 1988); very low BIS scores, on the other hand, are believed to correspond with primary psychopathy (Newman, MacCoon, Vaughn, & Sadeh, 2005).

The BAS is believed to be involved in the active approach of goals, and is believed to be sensitive to reward stimuli and the avoidance of punishment/non-reward stimuli. High BAS scores are believed to correspond with goal-driven behaviour, and are associated with impulsivity disorders (Wallace, Newman, & Bachorowski, 1991), attention-deficit/hyperactivity disorder (Mitchell & Nelson-Gray, 2006), and secondary psychopathy (Newman et al., 2005).

Psychophysiological responses to stimuli of varying incentive value may, at least in part, be determined by individual differences in personality (Dikman & Allen, 2000). Thus, it is worthwhile investigating whether individual differences in reward and punishment sensitivity are related to psychophysiological responses to gambling
outcomes. If self-reported individual differences in personality traits and ratings of subjective excitement during gambling are found to show a relationship with psychophysiological indices of incentive processing, then questionnaires that assess such variables may be used as indicators of underlying physiological deficits. Consequently, such tools, which are less invasive and less expensive to administer than physiological recordings, may be successfully implemented as screening instruments for individuals at risk of problem gambling. Previous research has shown that BIS/BAS scores are associated with psychophysiological reactions in response to reward and punishment stimuli (Balconi & Crivelli, 2010; Boksem, Tops, Kostermans, & De Cremer, 2008; Boksem, Tops, Wester, Meijman, & Lorist, 2006; De Pascalis, Varriale, & D'Antuono, 2010). BIS scores have been found to positively correlate with a putative neural index of conflict processing, the N200 ERP component, on no-go trials (on which inhibition of a prepotent response is required) during a Go/NoGo paradigm, and higher BAS scores has been shown to be associated with greater resting frontal left hemisphere activation (Amodio, Master, Yee, & Taylor, 2007). A study by Balconi and Crivelli (2010) examined the relationship between the FRN and P300 amplitudes and scores on the BIS/BAS. In that study, participants performed a two-choice decision-making computer task in which they were required to indicate the position of a target stimulus using button press, and feedback was given on the correctness of their response. On 50% of trials participants were given false feedback in which they were advised that they had indicated an incorrect choice when, in fact, their selection was correct. Higher FRN amplitudes were observed for individuals with higher BIS scores following both correct and incorrect feedback, and greater P300 amplitudes were found for individuals with higher BAS scores following false feedback.
The relationship between self-reported sensitivity to reward and punishment stimuli and psychophysiological indices of incentive value processing in problem gamblers has been scarcely studied (Goudriaan et al., 2006), particularly using cortical measures. The relatively few studies that have investigated self-reported sensitivity to reward and punishment in problem gamblers have reported heightened reward and punishment sensitivity in these individuals (Goudriaan et al., 2006; Loxton, Nguyen, Casey, & Dawe, 2008).

2.4.2. **Individual differences in impulsivity.**

Impulsivity is a complex, multi-dimensional personality construct. While there are various definitions and components associated with impulsivity, this personality trait is generally associated with an inclination towards acting with less forethought or consideration of consequences, perhaps due to an inability to suppress reward driven responses, often in a way that is not appropriate to the current situation compared to other individuals who have equal opportunities and/or knowledge (Aron, 2007; Barratt, 1959; Dickman, 1993; Eysenck, Pearson, Easting, & Allsopp, 1985; Patton, Stanford, & Barratt, 1995; Whiteside & Lynam, 2001). According to Eysenck and Eysenck (1978, 1980), there are two types of impulsivity; venturesomeness and impulsiveness. Venturesomeness is associated with sensation-seeking and risk-taking behaviour that is previously considered, but engaged in nevertheless, and is believed to be closely related to extraversion. Impulsiveness is associated with doing or saying things on the spur of the moment without prior planning or consideration, and is believed to be related to psychoticism.

Previous research has shown that problem gamblers have greater impulsive tendencies compared to non-problem gamblers (see van Holst, van den Brink, Veltman,
& Goudriaan, 2010 for a review), manifested in the tendency to seek immediate
gratification at the expense of greater long-term reward, despite the experience of
negative consequences, and/or a previously expressed desire to refrain from gambling.
Such a pattern of behaviour may be considered to be related to impulsiveness type of
impulsivity described by Eysenck & Eysenck (1978, 1980); thus, it is this type of
impulsivity that will be focussed upon in this dissertation. Specifically, in order to
determine whether an abnormally strong attraction to the experience of reward, or a
reduced sensitivity to punishment underlies the tendency to engage in impulsive
behaviours, the relationship between impulsivity and psychophysiological indicators of
incentive value processing was examined.
3.1. Aims of the Current Thesis

The primary aim of the current body of research was to investigate whether problem gamblers exhibit abnormal psychophysiological reactions to wins and losses that occur during EGM play, something that is of major significance for conceptualisations of this disorder. For example, a hypersensitive response to reward may indicate that problem gamblers are highly motivated to achieve the intermittent wins that occur during EGM gambling despite repeated losses. A hyposensitive response to reward may indicate that these individuals need to experience larger magnitude wins to achieve the same level of excitement as non-problem gamblers feel toward smaller wins, whereas a hyposensitive response to losses would suggest that the frequently occurring and often severe losses encountered during EGM gambling are not perceived by problem gamblers to be as aversive as what is perceived by non-problem gamblers. The identification of the nature of deficit in problem gambling has significant clinical implications with the potential to aid in diagnosis, clarify mechanisms, and to guide interventions by focussing on a specific response deficit.

Before examining the psychophysiological reactions of problem gamblers, we proceeded to determine whether reliable indicators of reward and punishment processing could be identified in healthy controls (Studies A and C). Novel data approaches with significant methodological improvements were used in the current body of research to comprehensively examine both peripheral (Studies A and B) and central (Studies C and D) nervous system functioning. The specific methodological advances achieved by each study will be outlined in the relevant empirical chapters (Chapters Four to Seven). Briefly, ERP-style averaging techniques (in which multiple epochs of the same outcome type were averaged together to improve the signal to noise
ratio) were applied to autonomic (electrodermal and heart rate activity) measures in order to investigate the effect different EGM outcomes have on psychophysiological responses. Reactions to large and small magnitude wins, losses, and near-wins, were examined at the exact time they occurred in order to replicate and extend the findings of previous research (Study A/Chapter Four). Following verification that autonomic measures can reliably index incentive value processing in controlled laboratory settings, the responses of problem gamblers to gambling outcomes while they gambled with their own money in actual club settings were examined in order to determine whether such responses could differentiate them from non-problem gamblers (Study B/Chapter Five). These data were analysed using LedaLab (Benedek & Kaernbach, 2010), a program that separates overlapping SCRs in order to get a more accurate representation of reactions to individual gambling stimuli. The application of a spatiotemporal PCA to ERP components previously shown to be sensitive to outcome valence (i.e., positive/good vs. negative/bad), allowed the determination of the actual nature of these responses (e.g., which conceptualisation of the feedback-related response is correct [Study C/Chapter Six]), and whether these latent ERP component responses were able to reliably differentiate the responses of problem gamblers from non-problem gamblers (Study D/Chapter Seven).

In addition to verifying whether problem gamblers were abnormally motivated by reward and/or punishment/non-reward, the current body of work addressed a number of key research questions in order to help advance the understanding of this disorder. These included:

- **Can problem gamblers successfully differentiate outcomes of varying magnitude?** As mentioned above (Section 2.2.), individuals
with substance use disorders demonstrate anomalous processing of the value of large and small magnitude rewards (Goldstein et al., 2007). In order to examine whether abnormal processing of reward magnitude also applies to problem gamblers, the psychophysiological effects of experiencing large and small wins was examined in Studies A (Chapter Four), B (Chapter Five), and C (Chapter Seven). The computer tasks used allowed participants to select the amount bet on each trial, meaning that the effect of experiencing different magnitude losses could also be indirectly examined (i.e., on non-reward trials following decisions to bet low or to bet high). These are important contributions to the field of gambling research with important implications for the understanding of the nature of deficit in this disorder. For example, if problem gamblers are found to be sensitive to large but not small wins, this may explain why these individuals gamble with larger amounts of money and for longer periods of time to obtain less frequent, large magnitude rewards and experience the same level of excitement non-problem gamblers feel toward smaller wins.

- **Do losses disguised as wins (LDWs) elicit similar psychophysiological responses to wins?** As previously outlined (Section 1.3.1.), it has been suggested that LDWs contribute to the addictive nature of EGMs (Dixon et al., 2010; Livingstone & Woolley, 2008). It is important to examine the role that these outcomes have on the attraction and continuation of play on EGMs; specifically, *do they make them more addictive?* The current thesis investigated whether these outcomes types are inherently
significant compared to losses for non-problem gamblers (Studies A and C), and whether problem gamblers find near-wins and/or LDWs more appetitive compared to individuals without gambling problems (Studies B and D).

- **Can self-report questionnaires indicate underlying neural deficiencies in incentive processing?** The current thesis examined whether self-reported differences in personality variables, such as impulsivity (assessed using the Impulsiveness questionnaire, Eysenck et al., 1985, Appendix C) and reward and punishment sensitivity (measured using the BIS/BAS scales, Carver & White, 1994) correlate with, and can predict objective psychophysiological responses to gambling outcomes.

- **Which theoretical accounts of problem gambling have empirical support?** The examination of whether abnormal physiological reactions to reward and/or punishment differentiate individuals with this disorder will allow an investigation of the theoretical conceptualisations of gambling behaviours, which may help guide therapeutic interventions. It may also help advance the understanding of the underlying mechanisms that contribute to the maladaptive behavioural patterns observed in afflicted individuals.

The specific focus on EGM gambling in the studies reported within this thesis is of significance. Data were recorded in ecologically-valid gambling scenarios, either
while participants played a realistic computer simulated EGM task (Studies A, C, and D), or while they gambled with their own money on actual EGMs within licensed club venues (Study B). The examination of how outcomes encountered during EGM gambling are responded to is an important contribution of this research, considering the potential for harm associated with this form of gambling. This focus will afford a better understanding of how problem gamblers respond to the stimuli that form the basis of their disorder, rather than other quasi-gambling laboratory experiments that may be far removed from the actual experience of gambling.
CHAPTER FOUR – STUDY A

Electrodermal Activity Reliably Captures Physiological Differences between Wins and Losses during Gambling on Electronic Machines

4.1. Preamble


Chapter Four contains the first empirical paper of this thesis. The study examined whether outcomes commonly encountered during EGM play can be reliably captured and quantified using psychophysiological indices of autonomic nervous system activity. The study demonstrated that electrodermal activity but not heart rate was found to be sensitive to manipulations outcome valence and magnitude while a sample of naive gamblers played a computer EGM task in a laboratory setting. The findings provide evidence that the methodology used to quantify psychophysiological responses may be successfully used to examine the responses to actual gambling activity in licensed gaming venues. Aspects of this paper were presented at the 19th Annual Conference of the Australasian Society for Psychophysiology, Newcastle, Australia, 28-30 November, 2009 (Appendix D).
4.2. **Abstract**

Differential patterns of physiological arousal to win and loss events during gambling is central to psychological conceptualisations of gambling behaviours but is poorly researched. We recorded HR and SCRs to wins and losses while 23 healthy participants played for small incentives on a computer-simulated EGM task. Wins produced large SCRs whereas losses did not, and large wins produced larger SCRs than small wins. Electrodermal measures also correlated with reward responsiveness on a personality measure and with ratings of excitement experienced while gambling. HR evidenced a slight deceleration before event outcomes and the rebound HR was larger after wins than after losses. This study demonstrates that physiological changes to gambling events can be reliably captured, and that these changes are sensitive to differential outcomes. These findings establish a foundation for future research in field settings.
4.3. **Introduction**

The fascinating and challenging aspect about the psychology of gambling behaviours is that, despite the fact that most people lose, gambling continues to attract wide community participation. It is noteworthy that most psychological theories (e.g., Blaszczynski & Nower, 2002; Brown, 1986, 1987; Sharpe, 2002; Sharpe & Tarrier, 1993) consider physiological arousal, or the “buzz-factor” associated with gambling, to be a critical determinant in the development and maintenance of gambling behaviours both within the community at large and for the 0.5-1% of the population who become problem gamblers (APA, 2000; Productivity Commission, 2010). For instance, Brown (1986) claims that the underlying motivator involved in the development and maintenance of gambling (including problem gambling) behaviours is the excitement caused by gambling rather than actual monetary gains. However, the empirical literature on gambling is over-represented by studies examining demographic, personality, social, cultural, and psychopathological factors associated with gambling, with a reciprocal neglect of psychophysiological activity during gambling. Further, although several theories posit that gambling increases arousal and suggest that gamblers may be hypoaroused (e.g., Brown, 1986), such theories often lack clarity, fail to specify mechanisms that may underlie and mediate these behaviours, and are often sufficiently malleable to fit contrary findings. For instance, both decreased reactivity (Goudriaan et al., 2006) and increased reactivity (e.g., Sharpe, Tarrier, Schotte & Spence, 1995) during gambling and gambling related activities are described as being consistent with the hypoarousal theory of gambling. The former findings are taken as evidence that gamblers are hypoaroused, and the latter are interpreted as evidence for compensatory mechanisms to address a hypoarousal problem. The current study focuses on electrodermal and cardiac activity in a laboratory based task using a sample of healthy
participants, so the literature governing pathological gambling will not be discussed in
detail. However, key themes and lacunae that emerge from a critical review of the
literature will be highlighted.

4.3.1. **Laboratory vs. field studies.**

An obvious dilemma confronting the gambling research domain is the competing
emphasis between laboratory versus field research. The empirical results concerning
gambling psychophysiology have yielded valuable information in some domains, but
have lacked robustness and consistency in others. Several studies have reported higher
levels of physiological activity (e.g., heart rate or cortisol) among problem/frequent
gamblers in the field (e.g., Carroll & Huxley, 1994; Krueger, Schedlowski, & Meyer,
2005; Meyer, Hauffa, Schedlowski, Pawlak, Stadler, & Exton, 2000) or in the
laboratory (e.g., Leary & Dickerson, 1985; Roby & Lumley, 1995; Sharpe et al., 1995).
Other studies have reported no group differences in field studies (e.g., Coulombe,
Ladouceur, Desharnais, & Jobin, 1992; Coventry & Constable, 1999; Coventry &
Norman, 1997; Griffiths, 1993) or in laboratory conditions (e.g., Diskin & Hodgins,
2003). On the one hand, playing for credits or a small incentive within a sanitised
laboratory set-up may appear a pale resemblance of real world gambling where large
amounts of money can be lost and won in rapid succession. On the other hand, although
field studies have ecological validity, they are expensive to run, and are difficult to
conduct because of limited access to patrons and gaming venues, or because adherence
to ethics principles (appropriately so) greatly restrict the types of variables that can be
manipulated in field settings (Dixon & Schreiber, 2004; Gainsbury & Blaszczynski,
2010). Importantly, even if these constraints could be overcome, a range of factors
within field settings pose major challenges for accurate and reliable recording of
physiological data (Gainsbury & Blaszczynski, 2010; Wilkes, Gonsalvez, & Blaszczynski, 2010). The typical glitz and glamour of club environs, accompanied by loud music, auditory jingles, and public announcements are likely to affect physiological measures but are difficult to control. Further, other factors systematically influence psychophysiological measurement including physical activity and movement, social interactions, and the fairly common use of substances such as alcohol, nicotine, and caffeine within gambling venues. Thus, despite some advantages of conducting ambulatory studies, which examine physiological reactions in actual gambling environments, there are still a large number of unresolved issues that have to be first demonstrated within the laboratory.

4.3.2. **Neglect of research on phasic activity.**

The vast majority of past physiological research in gambling has examined tonic changes. Typically, averaged levels (e.g., HR, or skin conductance level, SCL) during episodes of gambling lasting several minutes are compared to baseline measures. This has occurred across dependent measures, including for cardiac activity (Anderson & Brown, 1984; Coventry & Hudson, 2001; Krueger et al., 2005; Ladouceur, Sevigny, Blaszczynski, O’Connor, & Lavoie, 2003; Meyer et al., 2000; Meyer et al., 2004), electrodermal activity (Diskin & Hodgins, 2003; Roby & Lumley, 1995; Sharpe, 2004; Sharpe et al., 1995), and other measures, such as cortisol levels (Krueger et al., 2005; Meyer et al., 2000). The focus on tonic measures of arousal is problematic. Because tonic levels are determined by averaging over relatively long periods of gambling activity, the number and nature of win and loss events during the gambling period are unpredictable, but are nevertheless likely to influence results. Tonic measures are also sensitive to a range of other confounding influences previously mentioned, including
social interactions and the consumption of caffeine, nicotine, and alcohol, all common
behaviours during gambling activities. Further, it is somewhat difficult to interpret tonic
differences observed between groups. This is because different behaviours and practices
among gamblers, such as betting larger amounts for longer periods of time
(Blaszczynski, Sharpe, & Walker, 2001) and also the diverse outcomes from such
betting, may account for physiological changes observed, rather than the independent
variable manipulated (e.g., type of gambling activity or group differences such as
problem gamblers vs. non-problem gamblers). It appears obvious that to make
meaningful progress, a careful examination of phasic responses, particularly changes
associated with win and loss events within gambling activities, has to be undertaken;
however, research on phasic activity has been largely neglected. This neglect is
understandable because the rigorous laboratory protocols required to isolate individual
responses (e.g., skin conductance responses, SCRs), to prevent the influence of
uncontrolled variables affecting results, and to enable the measurement of subtle
changes, have limited applicability to gambling in the natural environment. Four recent
studies have attempted to examine phasic responses to win and loss events as they occur
in real time. Moodie and Finnigan (2005) monitored HR during actual gambling on
electronic gaming machines (EGMs) in the field and reported HR increases immediately
following win events, but the authors did not report results for losses. Goudriaan et al.
(2006) found that both problem gamblers and non-problem gamblers experienced a
decrease in HR after a loss, whereas non-problem gamblers exhibited an increase in HR
and problem gamblers a slight decrease following wins during play on the IGT. Wilkes,
Gonsalvez, and Blaszczynski (2009, 2010) examined SCRs and HR responses to win
and loss events among healthy participants as they played a commercial EGM. They
reported skin conductance (but not HR) changes to wins when subjects played for small
incentives (entertainment vouchers), but no changes in HR or skin conductance in response to losses. A salient point emerging from this limited but important body of research is that physiological changes to gambling events are sufficiently large and robust to be reliably captured on a second-by-second basis. It is notable that some studies (e.g., Wilkes et al., 2009, 2010) have successfully adopted methods not typically used for skin conductance and HR activity (e.g., the averaging technique routinely used to compute event-related brain potentials) to capture overlapping and ‘noisy’ SCRs in the field and to estimate their magnitude.

It is also of note, that amidst the equivocal and sometimes contrary findings to emerge from gambling research involving autonomic measures, one consistent finding is that winning raises physiological activity. This result is observed in field studies (Coventry & Hudson, 2001; Dickerson, Hinchy, England, Fabre, & Cunningham, 1992; Moodie & Finnigan, 2005) and in laboratory studies (Coventry & Norman, 1997; Wilkes et al., 2009, 2010). Also, this result is consistently seen for both tonic measures (Anderson & Brown, 1984; Coventry & Hudson, 2001; Diskin & Hodgins, 2003; Krueger et al., 2005; Ladouceur et al., 2003; Meyer et al., 2000, 2004; Roby & Lumley, 1995) and for phasic activity when the effects of win and loss events have been isolated (Moodie & Finnigan, 2005; Wilkes et al., 2009; 2010).

4.3.3. **Neglect of research on electrodermal activity.**

Electrodermal activity is considered the ‘gold standard’ when measuring physiological arousal because of its reliability as an index of arousal and its sensitivity to arousal-related changes (Barry, 1996; 2006), yet most of the research on gambling has focussed on HR (Anderson & Brown, 1984; Coulombe et al., 1992; Coventry & Constable, 1999; Coventry & Hudson, 2001; Coventry & Norman, 1997; 1998; Dickerson et al., 1992;
Griffiths, 1993; Ladouceur et al., 2003; Leary & Dickerson, 1985; Meyer et al., 2004; Moodie & Finnigan, 2005). Whilst there have been some reports on tonic measures of electrodermal activity (e.g., Diskin & Hodgins, 2003; Roby & Lumley, 1995; Sharpe et al., 1995), the neglect of phasic activity is glaring, especially within a theoretical context where arousal mechanisms are considered central to gambling theory. The relevance of phasic electrodermal work is further accentuated by recent research demonstrating that SCRs, but not HR, was sensitive to win events during EGM play (Wilkes et al., 2009; 2010). Although the studies of Wilkes et al. (2009; 2010) make a valuable contribution, it is unclear whether the observed changes to win events are produced by the outcome (win/loss), by stimulus novelty (win events are infrequent), and/or the elaborate visual and auditory stimuli that accompanied win events. There is a considerable body of work that demonstrates that stimulus novelty and attention-drawing cues produce SCRs (see Barry, 1996; Ben-Shakhar, 1994). Investigations that can isolate phasic changes associated with outcome (win/loss) from changes evoked by other stimulus and sensory factors are warranted.

4.3.4. Integration with neurobiological theory and research.

Better integration between the work on autonomic measures and advances in neurobiological theory is required. Two key areas are of relevance. First, recent pioneering research from the domain of ERPs has important theoretical and clinical implications for our understanding of mechanisms that may underlie reinforcement theory in general, and the operation of reinforcement contingencies within gambling tasks in particular (e.g., Gehring & Willoughby, 2002a; Holroyd & Coles, 2002). Central to this development is a body of ERP work that convincingly demonstrates that stimuli signalling negative and/or worse-than-expected outcomes evoke a negative
potential that peaks about 250 ms post-stimulus (Gehring & Willoughby, 2002a; Miltner et al., 1997) and is maximal at medial-frontal scalp sites. On the other hand, correct responses and wins during play on simulated gambling tasks generate a waveform of much smaller magnitude. The component(s) have been called error-related negativity (ERN; Holroyd & Coles, 2002), medial-frontal negativity (MFN; e.g., Gehring & Willoughby, 2002a), or feedback negativity (e.g., Hajcak et al., 2006; 2007). The relevance of this ERP component to gambling is that research has shown that losses (but not gains) in laboratory-based gambling tasks evoke a similar waveform. Although there is debate about whether the components generated by errors (the ERN) and correctly predicted negative feedback (the FRN) are the same component (see Gehring & Willoughby, 2002a, 2002b; Holroyd, Coles, & Nieuwenhuis, 2002), what is salient is that the ERP component, linked to generators in or near the anterior cingulate cortex (ACC), may reflect activity associated with reward mechanisms in the brain. For instance, the reinforcement learning theory proposes that, “ERN is generated when a negative reinforcement learning signal is conveyed to the anterior cingulate cortex via the mesencephalic dopamine system and that this signal is used by the anterior cingulate cortex to modify performance on the task at hand” (Holroyd & Coles, 2002, p. 679). Although the bulk of the empirical ERP work has been conducted on healthy participants, consistent results have emerged. In addition to being sensitive to direction of outcome (loss > wins) even when probability of occurrence is controlled (Gehring & Willoughby, 2002b; Masaki, Takeuchi, Gehring, Takasawa, & Yamazaki, 2006; Miltner et al., 1997), the FRN has also been shown to be sensitive to the magnitude of the loss (small wins > large wins; large losses > small losses; Gehring & Willoughby, 2002b; Masaki et al., 2006); and loss frequency (infrequent > frequent; Holroyd et al., 2003). The context of the outcome has also been shown to influence the component, with the
same outcome producing different amplitudes depending on whether the chosen alternative was the better or worse of the two options (Holroyd, Larsen, & Cohen, 2004). A recent study that examined ERPs among problem gamblers in a computer-simulated black-jack game, reports that problem gamblers have a hypersensitive response to rewards rather than an insensitivity to negative events (Hewig et al., 2010).

Also of relevance is reinforcement sensitivity theory (Gray, 1991) that implicates two neurological brain mechanisms in motivating individuals to seek out activities that provide reward and punishment. According to this model, punishment and punishment cues activate the Behavioural Inhibition System (BIS) whereas reward and its associated cues activate the Behavioural Activation System (BAS), promoting approach behaviours toward conditioned appetitive stimuli and hedonism. Depending on the relative levels of BIS and BAS inputs, a central arousal system will either facilitate or inhibit motor behaviour. Although Gray’s theory was not designed to explain gambling behaviours, its distinction between reward and punishment mechanisms underpins Damasio’s (1994, 1996) somatic marker hypothesis and predictions about the mechanisms that may underlie gambling behaviours. Win events will activate the BAS and loss events the BIS. A hypersensitivity to reward, low punishment sensitivity, or a dominance of immediate outcomes, together with insensitivity to positive and negative future outcomes, may promote the development and maintenance of gambling behaviours. A recent empirical study claims some support for Damasio’s predictions (Goudriaan et al., 2006).
4.3.5. **The relationship between physiological and psychopsychological measures of arousal.**

There is a substantive body of work on the relationship between personality and gambling behaviours. Specifically, there is strong evidence for the relationship between impulsivity and problem gambling. For instance, compared to non-problem gambler groups, problem gamblers show higher levels of impulsivity (Nordin & Nylander, 2007; Steel & Blaszczynski, 1998), and impulsivity has been found to mediate the severity of symptoms in problem gamblers (Steel & Blaszczynski, 1998; Vitaro, Arseneault, & Tremblay, 1997; 1999). Further insight may be gained by examining the relationship between impulsivity and physiological reactions to individual win and loss events.

Several ERP researchers have attempted to determine whether physiological measures are related to behavioural measures, including risk-taking, during gambling tasks (see van Holst et al., 2010 for a review). It is of value to determine whether higher levels of electrodermal and cardiac activity to win and loss events are associated with reward and punishment sensitivity, respectively, and whether, when, and which physiological measures of arousal actually translate to subjective reports of excitement. There have also been attempts among researchers to better understand the relationship between impulsivity and gambling behaviour.

4.3.6. **The current study.**

The aim of the current study was to systematically examine the physiological activity of healthy participants associated with win and loss events during play on a computer-simulated gambling task. The study also included measures of impulsivity, reward and punishment sensitivity, and obtained subjective measures of arousal during gambling to examine their relationship with psychophysiological measures.
Because EGMs dispense a schedule of rapid, discrete rewards and losses, and because EGMs are linked with increased risk for gambling problems (Blaszczynski et al., 2001; Delfabbro & Le Couteur, 2005), the current study designed and used a computer-simulated EGM task. The use of a simulated task had several advantages: (1) Wins and losses could be presented without attendant visual and auditory cues; (2) An accurate and reliable coding of events was possible, which is an improvement from previous experiments that relied on a manual coding of win and loss events (Wilkes et al., 2009; 2010); (3) Real-time play on EGMs allows a quick succession of betting and event outcomes (e.g., two events can occur within a 3 second time frame). Consequently, the physiological activity of the event of interest is affected by the physiological remnants of immediately preceding events including the placement of bets. The computerised program allowed a customised inter-event delay and therefore, a better separation of event-related responses; (4) Finally, the computerised task enabled the manipulation, and therefore, the systematic examination of different bet and win sizes on physiological measures.

We predicted that: (1) Wins, but not loss, events would produce significant SCRs and HR changes. (2) Larger wins would produce larger SCRs and HR changes than smaller wins, (3) Larger bet sizes will produce larger SCRs and HR changes than smaller bets, and (4) Objective measures (SCRs and HR activity) would be related to subjective reports of excitement during gambling activities.

4.4. Method

4.4.1. Participants.

Twenty-three first year psychology undergraduate students (4 male, 19 female) with age ranging from 18 to 59 years ($M = 19.70; SD = 3.76$) from the University of Wollongong
volunteered to participate in the study in exchange for research participation course credits.

4.4.2. Materials.

4.4.2.1. Physiological recording equipment.

The Ambulatory Monitoring System (AMS, version 5fs; Groot, de Geus, & de Vries, 1998) was used to record HR and skin conductance. HR was measured by two active Ag/AgCl electrodes positioned on the middle of the sternum and between the ninth and tenth ribs on the left hand side. HR was recorded as an inter-beat interval (IBI) which was then reconverted to mean HR for statistical analyses and representation in figures. Electrodermal activity was recorded through two sintered Ag/AgCl electrodes, each one cm in diameter, filled with an inert 0.05 M sodium chloride electrolyte ointment, and placed on the volar surface of the medial phalanx of the third and fourth digits of the non-dominant hand. Skin conductance was recorded with a constant voltage of 0.5 V, and sampled at 100 ms intervals.

4.4.2.2. Psychological inventories.

The current study is part of a larger program of research that involves the ambulatory recording of physiological activity among community and problem gamblers in actual gambling venues. Accordingly, data from a battery of questionnaires was also of interest. The following questionnaires to assess gambling behaviour and personality were administered.

Measure of the severity of gambling behaviours. The PGSI of the Canadian Problem Gambling Index (CPGI; Ferris & Wynne, 2001) assesses gambling behaviours and gambling severity (scored as follows: non-problem: 0; low risk: 1-2; moderate risk:
3-7; problem: 8 or above). The mean score of participants in this study on the PGSI was 1, with scores ranging from 0 to 3. The CPGI has adequate internal consistency (α = .84), test-retest reliability (α = .78), and validity (Ferris & Wynne, 2001).

**Measure of impulsivity.** The I7 Impulsiveness questionnaire (Eysenck & Eysenck, 1978; Eysenck et al., 1985) assesses the level of three different types of impulsivity, including impulsiveness (associated with the personality trait of psychoticism, and related behaviours, such as unintentionally engaging in behaviours without properly considering the associated risk or consequences), venturesomeness (related to the construct of extraversion, and associated with conscious decisions to engage in sensation- and novelty-seeking behaviour), and empathy (which has been found to relate to neuroticism). Only the 19-item impulsiveness subscale was completed by participants in the current study. Scores on this subscale can range from 1 to 19; on average, males score 8.76, $SD = 4.31$, and females score 8.17, $SD = 4.44$ (Eysenck et al., 1985). This inventory has been shown to have good reliability (Eysenck et al., 1985) and concurrent validity (Caci et al., 1998; Dickman, 1990).

**BIS/BAS scales (Carver & White, 1994).** The BIS/BAS scale has 24 items and is designed to assess the personality characteristics of behavioural activation and behavioural inhibition based on Gray’s (1991) theory. The scales have reasonable reliability (BIS: $α = .74$; BAS Reward Responsiveness: $α = .73$; BAS Drive: $α = .76$ and BAS Fun-seeking: $α = .66$) and convergent and discriminate validity (Carver & White, 1994).

**Gambling experience questionnaire.** A 12-item questionnaire was designed by the researchers to have participants rate their subjective experience (e.g., nine-point Likert scale level of excitement; 1 = very excited, 5 = moderately excited, 9 = not at all excited) before, during, and after their involvement in the experiment (Appendix E).
4.4.2.3. **Computer gambling task.**

A computer-simulated gambling task was designed using Presentation® software (Version 13.0, Neurobehavioral Systems, www.neurobs.com) to mimic common games played on real EGMs. A matrix comprising 5 columns × 3 rows of fruit pictures (8 different pictures were used) were displayed on the screen, as was information on the amount bet on each trial, how many credits left to play with, and the amount won (if anything) on each trial (Appendix F). The overall probability of occurrence of the three main outcomes (Wins, Near-wins, and Losses) was matched with observed outcomes from a real EGM (as determined by Wilkes et al., 2010). In effect, the program, although random, was designed to produce an overall distribution that comprised 20% Wins, 60% Losses, and 20% Near-wins. Among Win trials, three different types were identified: Small wins (three identical symbols in an uninterrupted sequence within a row), Medium wins (four in a row), and Large wins (five in a row). Two loss types were differentiated: Near-wins (three or more identical symbols within a row, but with a different symbol inserted between them) and Losses (fewer than three identical symbols within a row). From a monetary perspective, both Loss types resulted in identical outcomes, namely, the loss of the amount bet. A pool of 400 trials to mimic the above probabilities was predetermined by the researchers, with the computer program randomly picking three trials (rows) to present for each outcome. Statistically, the program was designed to ensure a 95% payout, that is, on average, 95% of the total amount bet would be returned to participants, although payouts would vary significantly around this mean for each participant. Participants could select ‘Bet 1’, ‘Bet 5,’ or ‘Bet 10’ options to wager one, five, or ten credits, respectively, on each trial. Win multipliers were set by the researchers as follows: the amount bet was multiplied by five for Small wins; by ten for Medium wins; and by twenty for Large wins. The program was
designed to automatically mark bet placement, Bet size (1, 5, or 10 credits), and type of Outcome (i.e., Large win, Medium win, Small win, Near-win, and Loss). The interval between the point when a bet was placed and an outcome displayed was set at 2 seconds. The interval between the time an outcome was displayed to the time the participant was able to make their next bet (signalled by the Bet-buttons turning from red to green) was set at 4 seconds, in order to reduce the occurrence of overlapping physiological activity associated with contiguous events. To ensure that visual and auditory cues did not influence physiological activity, no sounds or visual features were associated with any of the events.

4.4.3. **Procedure.**

The study’s protocol was approved by the University of Wollongong Human Research Ethics Committee. After providing informed consent (Appendices G and H), participants completed the CPGI, I7, and BIS/BAS scales. Participants who reported they smoking or consuming alcohol, illicit drugs, or medication in the two hours prior to testing were excluded from the experiment. The gambling task consisted of 300 trials, administered in two equal blocks of 15 minutes and separated by a 5 minute rest interval. Participants were free to choose one of three bet options on each trial (1, 5, or 10 credits). Each participant started the session with a pre-allocation of 5000 credits (valued at AUD $50). They were informed that they were able to win one movie voucher (valued at AUD $11.70) if they accumulated above 6000 or more credits, and two vouchers if they accumulated 7000 or more credits. At the end of each of the block, total credits were determined to ascertain winners (no adjustment was made to the credits won/lost during the half-time break). In effect, although the computer task simulated gambling to a certain extent, for ethical reasons participants did not gamble
with or lose their own money. Participants completed the gambling experience questionnaire immediately after the gambling task.

4.4.4. **Data extraction and analysis.**

The physiological data were collapsed into averages to chart a second-by-second record of ongoing physiological activity. These data were segmented into 21 second epochs comprising a 5 second baseline before the ‘bet’ was placed and a 15 second post-bet segment. This data analytic strategy is readily applied to field research and has been used previously to good effect (Wilkes et al., 2009; 2010). Its adoption in the current research also enables the comparison of results across studies. For the purposes of statistical analyses, three data points were computed to represent the physiological activity associated with the events of interest, including the Bet (B), derived from the activity averaged 1 to 2 seconds post-bet, *Outcome Time 1* (OT1; derived from the average activity 1 to 2 seconds post-outcome), and *Outcome Time 2* (OT2; average activity derived from 3 to 5 seconds post-outcome). These values were computed separately for each of the five different outcomes (Win and Loss combinations) before subtracting corresponding baseline values (1-2 seconds prior to bet). Because no more than a few participants experienced a *Large win* or used the bet-low option (1 credit) these data were excluded from statistical analysis. Tonic SCL for pre-, during, and post-play periods were derived by averaging the activity within 2 minute segments during the 5 minute breaks that were given to participants.

4.5. **Results**

The raw skin conductance data for the 21 second epoch are presented in Figure 1. The differential values (Δ, with baseline values subtracted) were subjected to two separate 4
Event (Intermediate win, Small win, Near-win, Loss) × 3 Time (Bet, OT1, OT2) × 2 Bet size (Intermediate, High) repeated measures analysis of variance (ANOVA) separately for HR and skin conductance data. For the Event factor, three planned contrasts were conducted comparing (i) Small wins and Intermediate wins, (ii) Wins (Small and Intermediate combined) and Losses, and, (iii) Near-wins and Losses. For the Time factor, two planned contrasts were performed: (i) Bet versus Outcome (Time 1 and Time 2 combined), and (ii) Outcome Time 1 versus Outcome Time 2 to determine if physiological changes to certain events persisted over a longer period.

4.5.1. **The effects of gambling on skin conductance activity.**

Skin conductance results are presented in Figure 2. A significant effect of Event was found with planned contrasts revealing a significant difference between Wins (Small and Intermediate combined) and Losses, \( F(1, 22) = 12.82, p = .002, \eta_p^2 = .37 \).

![Figure 1. SCL over time as a function of event Outcome (n = 23). Note, the scale of the y-axis does not start from zero.](image)
Figure 2. ∆SCL (baseline) at Bet, at Outcome Time 1 (OT1), and at Outcome Time 2 (OT2; n = 23).

Planned contrasts revealed larger SCRs at Outcome (Time 1 and Time 2) versus Bet, $F(1, 22) = 17.22, p < .001, \eta^2_p = .44$, and larger amplitudes at Outcome Time 1 compared with Outcome Time 2, $F(1, 22) = 7.13, p = .014, \eta^2_p = .25$. The Event × Time interaction was also significant, $F(1, 22) = 7.86, p < .001, \eta^2_p = .26$ (Figure 2). Taken together, the results indicate comparable SCRs for Wins and Losses at Bet, following which Wins, but not Losses, produced SCRs, $F(1, 22) = 15.91, p = .001, \eta^2_p = .35$. No significant difference was found between Small and Intermediate wins nor between the two loss types (Near-wins and Losses). The graphic representation (Figure 1) also indicates large SCRs associated with Large wins, although these data derive only from a subgroup of participants (n = 8) who experienced a Large win. A separate analysis conducted for this group tested the prediction that Large Wins would produce larger SCRs (1-tailed test). The results indicated support for the hypothesis, with significant
effects for Event, $F(1,7) = 5.92, p = .04, \eta^2_p = .46$, and for Time, $F(1,7) = 10.71, p = .01, \eta^2_p = .61$. None of the main or interaction effects for Bet size was significant.

4.5.2. The effects of gambling on heart activity.

Figure 3. Heart rate (HR) over time as a function of event outcomes ($n = 23$).

The 21 second epoch for HR (absolute values) are presented in Figure 3. The effect of Event on HR was not significant. The effect of Time was significant, $F(1, 22) = 5.14, p = .010, \eta^2_p = .19$, with larger HR increases observed at Outcome (Time 1 and Time 2 combined) compared to Bet, $F(1, 22) = 5.77, p = .025, \eta^2_p = .21$, but not between Outcome Time 1 and Outcome Time 2, $F(1, 22) = 1.97, p = .174$. The interaction between Event and Time (Bet vs. Outcome OT1, OT2) was also significant: Win events (Small & Intermediate) produced significant HR increases compared to Bet Time ($M = .80), F(1, 22) = 9.80, p = .005, \eta^2_p = .19$, whereas Losses did not ($M = .030). The results showed no significant main effect of Bet Size on $\Delta$HR.
4.5.3. **The relationship between physiological and personality data.**

For $\Delta$SCL, Wins correlated positively and Losses correlated negatively with the BAS Reward-responsiveness subscale; for Wins, $r(21) = .64, p = .001$; for Losses, $r(21) = -.41, p = .049$. Losses also correlated negatively with the BAS Drive subscale, $r(21) = -.49, p = .016$. No relationship between SCL changes and impulsivity ($I_7$ scores) was found. For $\Delta$HR, none of the correlations were significant.

4.5.4. **The relationship between individual differences in personality and objective measures of arousal.**

We examined the concordance between subjective reports of excitement and physiological measures. Both phasic changes (baseline-to-peak SCRs and HR) and tonic levels were examined. A significant correlation was observed between subjective reports of excitement during the play segment and baseline SC levels, $r(21) = .43, p = .041$. The other correlations were not significant, $\Delta$SCL for Wins, $r(21) = .03, p = .170$; for Losses, $r(21) = -.19, p = .373$; for the tonic data at pre-play, $\Delta$SCL: $r(21) = -.05, p = .835$; at post-play, $r(21) = .33, p = .122$. None of the correlations between HR and reports of subjective excitement were significant, $r(21) = -.07, p = .747$; for Wins, $r(21) = -.31, p = .148$; for Losses, $r(21) = .27, p = .219$; or for tonic HR data pre-play, $\Delta$HR, $r(21) = -.03, p = .900$; or post-play, $r(21) = -.19, p = .931$.

4.6. **Discussion**

The current study contributes to the existing literature in a number of ways. Before we progressed to conduct ambulatory studies in real time in actual gambling venues, it was essential to determine whether psychophysiological changes to win and loss events could be reliably captured and whether these changes could be attributed with certainty.
to differential outcomes (i.e., win vs. loss). The current study accomplished both of these purposes. First, we demonstrated that, consistent with previous studies (Wilkes et al., 2009; 2010), Win events produced reliable SCRs, whereas Losses did not. Second, because equally novel Near-wins did not produce the same effects observed for Wins, our results cannot be explained by the effects of stimulus novelty that is known to influence electrodermal activity in certain conditions (e.g., Ben-Shakhar, 1994). Further, because these results were observed when no visual or auditory cues accompanied the events, and when the events were separated by extended inter-event intervals, the larger SCRs observed to win events can be convincingly attributed to the outcome (Win/Loss) of the trial. Because SCR latencies are long (1-4 s post-event is typical), it is feasible that SCR peaks observed 2 to 5 s post-outcome represent the cumulative or interactional effects of bet and outcome. However, because the bet-event was common to all five outcomes (Losses, Near-wins, and the three Win types), the differential effects observed between Win and Loss outcomes may not be attributed solely to the Bet, but represent, at least in part, an outcome-related effect. This is demonstrated clearly in Figure 1, where the Bet/Loss and Bet/Near-win combinations provoke no observable SCR, whereas each of the three Bet/Win combinations triggers large SCRs. These results are supportive of Hypothesis 1 and, together with previous results, offer reliable and robust support that electodermal activity is sensitive to the reward characteristics of the EGM outcomes. It must be acknowledged that results from this study and the data from Wilkes et al. (2009; 2010) apply to healthy participants under conditions when incentives are small (a movie voucher) and are received at the end of the experiment. In actual gambling practice, monetary gains and losses are large and accrued on a trial-by-trial basis. Nevertheless, our results have important theoretical and clinical applications, because it can be reasonably assumed that in real gambling conditions, the physiological
patterns observed in our study will be matched and probably exceeded. The
determination that changes in electrodermal activity are indeed associated with win/loss
events is important on several counts. Psychological theories of gambling have
hypothesised that hypersensitivity to wins and/or a hyposensitivity to losses may be
responsible for the maintenance of gambling behaviours. Our results have provided the
preliminary but essential platform to actually test these hypotheses among problem
gamblers in field studies. Second, there is interesting ERP research that suggests that the
magnitude of the FRN is sensitive to reward reinforcement in laboratory simulated
gambling tasks (Gehring & Willoughby, 2002b; Masaki et al, 2006; San Martin et al.,
2010; Yeung & Sanfey, 2004). Any attempt to fully understand neurobiological
mechanisms that may cause and sustain gambling behaviour among healthy controls
and among problem gamblers will need to clarify whether and how central and
autonomic indices of reward-reinforcers interact with each other. Within this context,
the results of the current study emphasise the need to extend such research to
ambulatory recordings during real gambling activities in commercial clubs and casinos.
Such research is already underway.

We failed to find significant SCR differences between win sizes (Small and
Intermediate wins). However, a separate analysis based on the subgroup ($n = 8$) who
experienced both Large and Small Wins indicates that Large Wins produce larger SCRs
than Small Wins, further validating the assumption that SCRs are sensitive not only to
win/loss differences but to also win-magnitude effects. The higher baseline associated
with Large Wins (see Figure 1) was due to an outlier within the small group ($n = 8$), and
should not affect the results, given that SCRs were measured relative to baseline. Larger
SCRs for large wins as compared with small wins were also reported by Wilkes et al.
(2010). We failed to find significant SCR differences between the two loss types, Near-
wins and Losses. It has been suggested that Near-wins (sometimes incorrectly called near misses in the literature) may be considered a type of reinforcement (Reid, 1986). These outcomes have been predicted to produce physiological arousal similar to wins (Reid, 1986), and are hypothesised to encourage continuation of playing (Griffiths, 1999), and to be responsible for other gambling behaviours such as response latencies and win expectancies (Dixon & Schreiber, 2004). Few studies have actually investigated whether Near-wins are accompanied by higher arousal. Wilkes et al. (2010) demonstrated that, compared to losses, “losses disguised as wins” generated larger SCRs but had no effect on HR. Although our results do not corroborate the results of these studies, it should be pointed out that the Near-wins in our study differed from losses disguised as wins on two counts. Near-wins did not yield a return in our study (and therefore, were identical to a loss in terms of credit-value) whereas losses disguised as wins yielded a small return (less than amount bet) in Wilkes et al. study. Secondly, Wilkes used a commercial EGM where losses disguised as wins are accompanied by ‘positive’ visual and auditory cues that may have influenced the electrodermal activity.

4.6.1. **Temporal course of the physiological responses.**

An examination of the temporal course of electrodermal and HR reactivity appears important for several reasons. As compared to the temporal course of skin conductance changes sustained over an 8 second period of EGM play (Wilkes et al., 2009; 2010), the computer-simulated task in the current study evidenced initial SCL increases (OT1; 3-5 seconds) that were followed by a more rapid decrease at OT2 (5-7 seconds). In both studies, participants played for similarly small incentives. This difference is likely to be due to the longer inter-event intervals in the computer-simulated task of the current study (4 seconds vs. 2 seconds in the Wilkes et al., 2009; 2010 study), and/or the
influence of auditory jingles and flashy visual displays during EGM play. EGMs have been described as the crack-cocaine of gambling activities (Korn & Schaffer, 1999) because of their addictive power to create and maintain problem gambling behaviours. The current research has clarified that Win events, in the absence of the attention-grabbing cues, still evoke larger SCRs than Loss events. However, it is possible that the temporally dense array of win-and-loss events, together with their accompanying sensory cues contribute to the sustenance of arousal and therefore their “buzz” potential. In fact, previous researchers have speculated that a longer temporal separation of events may reduce the addictive potential of EGMs and whether such changes will indeed affect dysfunctional gambling behaviours (Dickerson et al., 1992). Research that compares the effects of dense versus dispersed blocks of events on subjective and objectives indices of arousal within a within-subjects experimental design may help clarify this hypothesis.

4.6.2. Skin conductance vs. heart activity measures.

Unlike SCL, heart rate has proved to be a fairly insensitive measure of arousal, and no significant win-loss HR differences were detected in previous experiments where healthy participants used commercial EGMs (Wilkes et al., 2009; 2010). The computer-simulation task adopted in the current study had the advantage of being able to disentangle effects of bet and outcome and to separate overlap between rapidly occurring events. The HR results were interesting in that a slight but noticeable HR deceleration immediately before the outcome (at Bet) was followed by a rebound increase after the outcome (See Figure 3). Relative to HR at Bet, Wins generated a small but significant increase whereas Losses did not. A similar pattern of HR deceleration followed by a rebound has been reported for shooting sports and is likely to
reflect a state of vigilance (see Barry, 2006; Tremayne & Barry, 2001). As compared with SCL, HR appears more variable and “noisy” (Figure 3), and it is possible that the many rapid events associated with gambling on EGMs (bet-outcome in rapid succession) embed true HR differences in “noise.” Thus the longer outcome-to-subsequent-bet interval (4 seconds) introduced in the current study may have helped these subtle differences to emerge. In any case our results concerning HR should be replicated and should be investigated further in real-life gambling. Previous studies have shown tonic increases of HR during gambling in laboratory (Anderson & Brown, 1984; Ladouceur et al., 2003) and field settings (Coventry & Hudson, 2001; Krueger et al., 2005; Meyer et al., 2000; Meyer et al., 2004), and it is possible that phasic changes would be observed when actual money is won and lost.

4.6.3. The relationship between psychophysiological reactivity and personality measures.

As far as we are aware, this study was the first to examine the extent to which personality variables account for within subject variation in individual physiological responses to win and loss events. We tested the interesting possibility that physiological reactivity to wins, as evidenced from HR and SCL changes, might be correlated with the personality trait of reward sensitivity, and that reactivity to losses might be related to punishment-sensitivity. We found evidence that SCL reactivity to wins was related to reward responsiveness and drive (subscales of the BAS). Given the relatively small sample, these results warrant replication. We also found evidence of a fairly strong negative correlation between electrodermal changes to Win and Loss events. Specifically, a greater sensitivity to wins (larger SCRs) was associated with the opposite effect for Losses (drop in SCL relative to baseline). The theoretical implication is that,
at least in a psychophysiological sense, there was no support for the position that reward sensitivity is a dimension independent of punishment sensitivity. The decrease of SCL immediately after Loss-events is an interesting observation that requires further corroboration and clarification in terms of the mechanisms that could underpin such a response. If it reflects a manifestation of an inhibitory mechanism, its measurement among problem gamblers will become relevant and warrant further investigation. We also tested the potential association between trait impulsivity and electrodermal and cardiac reactivity, but found no significant association. Our prediction that participants with higher impulsivity scores would exhibit higher HR and SCL in response to win events was not upheld.

4.6.4. **Subjective vs. objective measures of arousal.**

Our study is also one of few gambling studies that report subjective and objective indices of arousal. Higher electrodermal activity during the gambling session was associated with higher ratings of excitement during play. However, participant ratings of the extent to which they reacted specifically to Win and Loss events during the gambling task bore no relationship with their physiological reactivity to these events. Thus, subjective ratings of overall excitement (for the gambling session overall) appeared to be consonant with tonic measures of electrodermal activity during the session, but subjects were oblivious to their physiological reactivity to specific events (e.g., Wins and Losses). The study raises the interesting possibility that higher tonic levels, rather than phasic changes, may be the key determinant of subjective states of excitement. These findings have important implications because they imply that gambling activities that achieve larger and more sustained tonic levels of arousal have a greater potential to be perceived as exciting and therefore, probably more addictive.
Within this perspective, the specific allure of EGMs could be explained by their frequent dispensation of a range of small wins that raise SCL during play giving participants a good dose of the “buzz” regardless of whether they emerge from the session an overall winner or loser. We are unaware of similar findings reported elsewhere and hence, these observations need further clarification and corroboration.

4.6.5. **Physiological effects of different bet sizes.**

It was important to examine the physiological effects that accompany betting behaviour, particularly effects associated with bet size. Problem gamblers are known to indiscriminately bet larger amounts when gambling on EGMs (Blaszczynski et al., 2001), and such behaviour would be consistent with the hypoarousal theory of problem gambling if it could be shown that larger bets evoke larger physiological responses. The current study did not yield a significant finding for *Bet size*, although our manipulation of *bet-size* was compromised because data from the bet-small option had to be excluded because too few participants chose this option.

4.6.6. **Limitations and future research.**

Compliance with ethics guidelines prevents exposing participants to actual gambling in the laboratory. Although participants could gain an overall incentive at the end of the session, they did not risk losses, they were not allowed to keep trial-to-trial gains, and were prevented from winning large amounts of money. These constraints could have served to reduce effects in one or both measures examined. The gender bias in our sample could also constrain interpretation and generalisation of effects. Gender has been shown to influence underlying motivations to gamble (Potenza, Steinbery, McLaughlin, Wu, Rounsaville, & O’Malley, 2001), age of onset, symptom progression, and preferred
gambling medium (Blanco, Hasin, Petry, Stinson, & Grant, 2006). A limited number of studies have investigated the impact of gender differences on psychophysiological measures. These studies have reported that gender does not differentially affect physiological reactions (e.g., Coventry & Hudson, 2001). However, it should be pointed out that age ranges and the large bias in favour of women in our sample are representative of age and gender ratios in undergraduate psychology student populations, but not representative of these variables within populations of recreational or problem gamblers.

Despite these limitations, laboratory based studies were an essential prerequisite that sets the foundation for further real-life investigations. Such studies contribute significantly in a number of ways. They confirm that: i) reliable and robust physiological changes occur to wins, but not to losses, even when the participant is not allowed to keep/lose trial-to-trial wins/losses, (ii) skin conductance is a better index of these changes than HR, at least in laboratory conditions, (iii) a method of time-locked, second-by-second averaging can reliably capture these changes despite the possibility that they are embedded in the noise associated with trial-to-trial expectations, physical and muscle movements, variations in terms of bet size, and overlapping physiological responses associated with several events in sequence, (iv) electrodermal reactivity to wins and losses is correlated with personality characteristics of reward-sensitivity, and that (v) the overall tide of high arousal during gambling (rather than phasic rise and fall of electrodermal activity associated to specific wins) may be the key determinant of subjective states of enjoyment. The systematic examination of HR and skin conductance while participants gamble money on EGM in commercial venues is currently underway to build upon the progress established in the current study.
CHAPTER FIVE – STUDY B

Problem Gamblers are Hyposensitive to Wins: An Analysis of Skin Conductance Responses during Actual Gambling on Electronic Gaming Machines

5.1. Preamble


Chapter Five contains the second empirical paper of this thesis. This paper aimed to determine whether the differential skin conductance responses observed in Study A could also be captured while gamblers played with their own money on EGMs in licensed gambling venues, and to determine whether the responses of problem gamblers differ from those of non-problem gamblers. Heart rate was not a reliable indicator of valence and magnitude manipulations in Study A (Chapter Four) so was not explored in this study. Aspects of this paper were presented at the 22nd Annual Conference for the Australasian Society for Psychophysiology, Sydney, Australia, 28-30 November, 2012 (Appendix I), and at the 51st Annual Meeting of the Society for Psychophysiological Research, Boston, U.S.A., 14-18 September, 2011 (Appendix J).
5.2 Abstract

Physiological arousal is purportedly a key determinant in the development and maintenance of gambling behaviours, with problem gambling conceptualised in terms of abnormal autonomic responses. Theoretical conceptualisations of problem gambling are discordant regarding the nature of deficits in this disorder; some accounts posit that problem gamblers are hypersensitive to reward, and others that they are hyposensitive to reward and/or punishment. Previous research examining phasic electrodermal responses in gamblers has been limited to laboratory settings, and reactions to real gaming situations need to be examined. Skin conductance responses (SCRs) to Losses, Wins, and Losses disguised as wins (LDWs) were recorded from 15 problem gamblers (PGs) and 15 non-problem gamblers (NPGs) while they wagered their own money during electronic gaming machine play. PGs demonstrated significantly reduced SCRs to reward. SCRs to losses and LDWs did not differ for either PGs or NPGs. This hyposensitivity to wins may reflect abnormalities in incentive processing, and may represent a potential biological marker for problem gambling.
5.3. Introduction

Problem gambling (also known as disordered or pathological gambling) has been reclassified recently as an addictive disorder in the DSM-5 (cf. DSM-IV-TR) (APA, 2013) due to its high co-morbidity and many shared similarities with substance use disorders (Blaszczynski et al., 2008). It is characterised by continued harmful patterns of gambling activity despite severe personal and interpersonal consequences, and is associated with high rates of depression and suicide (Raylu & Oei, 2002).

Several theories attempting to explain problem gambling behaviours highlight abnormal psychophysiological reactions to reward and/or punishment as a major determinant in the development and maintenance of this disorder (e.g., Blaszczynski & Nower, 2002; Blum et al., 1996; Blum et al., 2000; Damasio, 1994; Goldstein & Volkow, 2002; Holroyd & Coles, 2002; Sharpe & Tarrier, 1993). Specifically, such theories propose that characteristic behaviours of problem gambling stem from either a hypersensitivity to reward, or a hyposensitivity to reward and/or to punishment. Behavioural (e.g., Brown, 1986; McConaghy, 1980; Zuckerman, 1979) and cognitive-behavioural (e.g., Blaszczynski & Nower, 2002; Sharpe, 2002) models of gambling behaviour implicate autonomic arousal, perceived as the excitement associated with gambling, as fundamentally appealing and a (possibly the) major reinforcer for the gambler. Sharpe and Tarrier (1993) posit that problem gamblers cognitively appraise rewarding outcomes as more significant due to conditioning that occurred during previous encounters with gambling activity, and they should therefore demonstrate greater increases in physiological arousal following positively valenced (i.e., win) outcomes. The state of optimal functioning theory (McConaghy, 1980) postulates that problem gamblers are chronically hypoaroused and engage in harmful gambling behaviours in order to achieve a normal level of functioning. Biological hedonism
models (e.g., Zuckerman, 1979) propose that individual differences in personality mediate the propensity to seek out reward or to avoid punishment. Further empirical research on how problem gamblers respond to positively and negatively valenced stimuli is required to ascertain which of these conceptualisations of problem gamblers are correct, and to ultimately determine the nature of deficit in this disorder.

Electrodermal activity has proven to be a reliable indicator of autonomic and cortical arousal (Barry, 1996; Barry et al., 2004; Bouscein, 1992; Lykken & Venables, 1971; Raskin, 1973); however, most gambling studies have examined changes in heart rate (HR) as the primary index of arousal (e.g., Anderson & Brown, 1984; Coventry & Hudson, 2001; Krueger et al., 2005; Ladouceur et al., 2003; Meyer et al., 2000; Meyer et al., 2004), a variable shown to be a better indicator of vigilance and task performance (Barry, 2006; Tremayne & Barry, 2001).

To determine whether problem gamblers are abnormally sensitive to outcomes of varying incentive value, the responses that accompany instances of reward and punishment must be examined; however, the majority of previous autonomic research has examined the effects of gambling on tonic arousal levels over extended periods of time, either comparing levels before, during, and after play (e.g., Carroll & Huxley, 1994; Coulombe et al., 1992; Griffiths, 1993; Meyer et al., 2000; Meyer et al., 2004), or over long periods of gambling (e.g., Coventry & Norman, 1997; Dickerson et al., 1992; Sharpe, 2004). This type of tonic research has generally found that gambling activity increases arousal, particularly when winning (Coventry & Constable, 1999; Coventry & Hudson, 2001; Sharpe, 2004) and during gambling in naturalistic settings (Anderson & Brown, 1984; Diskin, Hodgins, & Skitch, 2003) for both problem and non-problem gamblers. Although such research has provided valuable insights into the arousing nature of gambling activities, several problems with this approach are apparent. For
example, because tonic levels are recorded over relatively long periods of gambling activity, the effect of individual win and loss events cannot be accurately determined, but are nevertheless likely to influence arousal. Furthermore, it remains unclear whether the increases in arousal during gambling reported in previous tonic research were due to the actuality of winning, or the excitement caused by gambling activities in general (and, by extension, the mere possibility of winning). Individual differences on tonic measures are more susceptible to a range of confounding influences, including social interactions, the consumption of legal (e.g., caffeine, nicotine, alcohol) and illegal drugs, physical movements, and the context in which the outcomes are experienced (e.g., the amount wagered, the presence of features on electronic gaming machines, i.e., bonus free spins/games, second screen games, scatters, or substitutes that generally result in larger amounts of money/credits being returned to players). Comparisons between groups are also difficult due to different behaviours and practices of problem gamblers, such as wagering larger amounts of money, consuming greater amounts of alcohol and cigarettes, and/or spending greater amounts of time gambling compared to non-problem gamblers (Blaszczynski et al., 2001).

In order to overcome these problems, and to allow an investigation of the theoretical conceptualisations of problem gambling, the current study sought to examine the phasic skin conductance responses (SCRs) immediately following individual win and loss outcomes in an ecologically-valid setting. Although scarce, previous studies that have taken this phasic approach have demonstrated that the reactions to gambling outcomes are sufficiently robust to be reliably captured and quantified, and generally report greater responses following wins compared to losses (Dixon et al., 2010; Goudriaan et al., 2006; Lole, Gonsalvez, Blaszczynski, & Clarke, 2012; Wilkes et al., 2009). Even fewer studies have examined these reactions in problem gamblers.
Goudriaan et al. (2006) reported that, while healthy controls displayed decreased HR after losses and increased HR following wins, problem gamblers demonstrated a decrease in HR following both win and loss outcomes during play on the Iowa Gambling Task (Bechara, Damasio, Damasio, & Anderson, 1994). SCRs following wins or losses were not found to differ for either problem gamblers or healthy controls. The authors of that study interpreted these findings as indicative of a reward hyposensitivity in problem gambling. However, these results require further validation, as studying gambling behaviour in artificial laboratory environments can engender a number of problems (Anderson & Brown, 1984). By definition, gambling involves placing a wager on an unpredictable outcome, in which the result of the gamble reflects an element of chance (Bolen & Boyd, 1968). For ethical reasons, participants in laboratory-based studies are usually not permitted to gamble with their own money, and there is no (or, at best, limited) potential to win large amounts of money compared to when gambling in a casino or club setting. Similarly, if participants need to reach a certain threshold of credits amounts before they receive a reward, their level of excitement may be quite low if they know that they are unlikely to reach this threshold. Alternatively, if they believe that they can only catch up by taking risks they would not normally take, they may become unrealistically excited.

Another important factor for studies where participants wager freely assigned credits relates to the nature of negative outcomes. Specifically, participants may perceive loss outcomes as merely non-rewarding, rather than punishing, since they are not actually losing their own money. Thus, motivations and the extent to which the task resembles real gambling activity are likely to influence responses (Anderson & Brown, 1984). In order to develop an ecologically-valid account of problem gamblers’ responses to reward and punishment, the current study investigated the physiological
reactions that occur during actual gambling activity on electronic gaming machines (EGMs, also known as ‘poker’ or ‘slot’ machines) in licensed gaming venues when participants wagered their own money. EGM gambling is of particular clinical significance to this population, as a high proportion of individuals seeking treatment for gambling report addiction to this gambling medium (Dowling, Smith, & Thomas, 2005, 2001), and this form of gambling is associated with a faster progression of addiction (Breen & Zimmerman, 2002), as well as more severe symptoms (Petry, 2003).

In addition to wins and losses, losses disguised as wins (LDWs), outcomes in which the amount returned is less than that wagered, are a key outcome experienced during EGM gambling. These outcomes are accompanied by visual and auditory feedback similar to that triggered by wins (in contrast, flashing visual or auditory feedback are absent in response to a loss), and are estimated to constitute up to 18% of all outcomes (often outnumbering wins) (Dixon et al., 2010). Novice gamblers have been shown to display similar physiological reactions to LDWs as they do to true wins, suggesting that these outcomes are a design feature of EGMs that contribute to continued play despite overall loss of money (Dixon et al., 2010; see also Clark et al., 2009; Luo et al., 2011; Qi et al., 2011).

5.3.1. The current study.

The current study sought to investigate the immediate physiological responses of problem gamblers to EGM outcomes experienced while they gambled with their own money in an actual club environment, and whether these responses differ from the responses of experienced non-problem gamblers. Following the findings of previous literature (Dixon et al., 2010; Goudriaan et al., 2006; Lole et al., 2012; Wilkes et al., 2009), we expected greater SCRs following wins compared to losses for non-problem
gamblers. Because theoretical conceptualisations regarding the significance of reward in problem gambling are conflicting (e.g., some support the notion of reward hyposensitivity, whereas others suggest a hypersensitive response to reward in these individuals), the current study investigated which account of the nature of deficit in this disorder is supported, by examining the psychophysiological responses of these individuals that occur during actual gambling activity. Based on the assumption that wins are more motivationally significant than losses, and that LDWs are perceived to be like wins (Dixon et al., 2010), we predicted that wins and LDWs would elicit greater SCRs than loss outcomes for experienced non-problem gamblers. Moreover, because these outcomes have been suggested to contribute to the development and maintenance of problem gambling behaviours on EGMs (Clark et al., 2009; Dixon et al., 2010), we predicted that problem gamblers would be more responsive to LDWs than non-problem gamblers.

5.4. Method

5.4.1. Participants.

The current study is part of a larger ongoing program of research examining reactions to stimuli that occur during real EGM play between problem and non-problem gamblers recruited from licensed gaming venues. Signs inviting patrons to participate in the study were posted in the venue (Appendix K). Individuals wishing to participate approached the researcher seated at a small table near the gaming area.

Data were recorded from 34 non-problem gamblers (NPGs), and 22 problem gamblers (PGs). Of these participants, 11 NPGs and 7 PGs experienced a ‘feature’ during the study. Preliminary analyses showed that experiencing a feature during the course of EGM play increases tonic arousal levels over an extended period of time.
These design aspects of EGM gambling involve the player receiving free reel spins following the attainment of a particular combination of symbols, and occur over a time period lasting up to several minutes. The win outcomes experienced within a free reel spin feature are not comparable to normal wins since they are not associated with betting activity. The non-win trials that occur within features cannot be considered true loss outcomes since the player’s own credits are not actually used and lost (hence, free spin). Thus, participants who experienced features were excluded from the current analyses; their data will be analysed reported elsewhere once a sufficient sample is obtained. Eight of the individuals classified as NPGs experienced fewer than five epochs for at least one outcome type, and were also excluded from the final analysis. Finally, data from participants who reported consuming more than two standard drinks on the day of recording were not included in the final dataset. Accordingly, 15 problem gamblers (10 male, 5 female; $M_{\text{age}} = 34.17$ years, $SD = 13.40$; age range 18-70 years) and 15 non-problem gamblers (9 male, 6 female; $M_{\text{age}} = 40.18$ years, $SD = 20.64$; age range 18-70 years) were included in the current analyses. All participants were of Caucasian European or Asian heritage.

5.4.2. Materials.

Recording equipment. The Ambulatory Monitoring System (model AMS5fs; Groot et al., 1998) was used to record electrodermal activity. Two sintered silver/silver-chloride (Ag/AgCl) electrodes (outer diameter: 1.5 cm, inner diameter: 0.8 cm) were filled with an inert 0.05 M NaCl electrolyte cream, and placed on the volar surface of the medial phalanx of the third and fourth digits of the non-dominant hand, that were cleaned using 70% isopropyl alcohol wipes. Skin conductance was recorded at a constant voltage of 0.5 V, and sampled at 10 Hz (0.1 s intervals).
**Measure of gambling behaviour.** The Problem Gambling Severity Inventory (PGSI) of the Canadian Problem Gambling Index (CPGI; Ferris & Wynne, 2001) was designed to measure general population prevalence rates for problem gambling. Participants are required to answer nine questions that assess their ability to control their gambling behaviours (e.g., ‘Have you bet more than you could really afford to lose?’), and the frequency (i.e., ‘never,’ ‘sometimes,’ ‘most of the time,’ or ‘almost always’) they experienced health-related, financial, and/or psychological problems (e.g., ‘Has gambling caused you any health problems, including stress or anxiety?’) in the previous twelve months as a result of their gambling activity. This measure was used in the current study to categorise individuals as ‘problem’ gamblers (score of 8 or higher, to a total maximum score of 27) or ‘non-problem’ gamblers (score below 8). This cut-off score has been shown to reliably identify the gambler’s diagnostic status based on DSM-IV criteria and clinical assessment interviews (Ferris & Wynne, 2001). Since it provides a means of identifying problem gamblers in a quick, confidential, and anonymous manner (thus, avoiding the problem of symptom under-reporting commonly associated with socially undesirable behaviours, e.g., Sudman, 2001), this quantitative self-report measure was chosen for use in the current study.

5.4.3. **Procedure.**

After providing informed consent (Appendices L and M), participants were fitted with the recording equipment. Apart from being asked to keep movement to a minimum, participants were instructed to play on an EGM of their choice as they typically would for as long as they desired. During play, the researcher stood behind them and, when each event (win, loss, LDW, start of a feature) occurred, discreetly pressed a remote event marker (four different buttons on a small oval pad, size of a car key) that inserted
a mark at the appropriate place on the physiological recording. When wins and LDWs occur on EGMs, the machine gradually accumulates credits for that particular spin (separate auditory stimuli are also presented for as long as the credits ‘climb’); losses were identified by the researcher immediately after they occurred, whereas wins and near-wins could only be recorded as such once the credits stopped accumulating and it could be determined whether the amount returned exceeded the threshold of the amount bet (therefore, the physiological effects associated with experiencing these outcomes would be likely to start earlier than when the event was actually finally defined and marked by the researcher; see Figure 4B).

Ethics guidelines allowed the researcher to record but not to promote gambling in any manner (e.g., by setting a uniform start total or bet amount). Thus, participants were in total control of the amount of time and money spent during the session, and the amount bet on each trial (i.e., the amount wagered was not held constant). Participants determined completion of the session of play, and accordingly, advised the researcher of their intent to discontinue gambling or the testing phase. Upon completion of play, participants completed the PGSI and were given a bistro voucher (valued at AUD $40) for use within the gaming venue in appreciation of their time. Written informed consent was obtained from all participants prior to their involvement in the study; they were advised that participation was entirely voluntary, and that they could withdraw from the study at any time (Appendices L and M). The University of Wollongong Human Research Ethics Committee approved the research protocol.

5.4.4. **Data extraction and analysis.**

The raw electrodermal data were epoched offline in order to isolate each individual outcome from the continuous data trace. These epochs included a 2 s period pre-event
and a 9 s period post-event. The time of occurrence for each outcome was adjusted by 1 s to compensate for the delay in the researcher’s reaction time, as estimated by a separate computer program. A similar procedure and correction has been employed in previous studies (Wilkes et al., 2009).

Since EGMs allow a rapid succession of bet placement (every 3 to 6 s), SCRs from consecutive events frequently overlapped. Traditional data extraction methods, which examine trough to peak differences, have been shown to underestimate SCR amplitudes in paradigms with short inter-stimulus intervals due to distortion caused by the recovery slope of preceding responses (Boucsein, 1992). In order to overcome this problem, the data were analysed using LedaLab software (version 2.10; Benedek & Kaernbach, 2010). Based on the assumption that sudomotor nerve bursts (which underlie skin conductance responses) are characterised by a distinct and compact period of activity, and that theoretically, the activity of these nerves cannot be negative, this program uses biexponential algorithms to decompose overlapping SCRs in a four-step process that is repeated a number of times (in this case, three times) to ensure the data are optimised and to increase the goodness of fit of the model. The discrete decomposition analysis performed on each individual trial calculates the amount of electrodermal activity caused by tonic skin conductance levels, the sudomotor nerve (i.e., the driver of the SCR), and the remainder signal (i.e., deviations from the standard SCR shape, proposed to be caused by pore opening). SCR amplitude and area under the curve measures are then derived from the single, non-overlapped response (derived by convolution of each impulse using an algorithm that estimates the underlying sudomotor nerve activity based on the shape of the SCR, and adding the remainder activity related to subsequent pore opening processes, if these data are available), and the original skin conductance (SC) data are reconstructed by adding the tonic component. The separation
of SCRs from tonic skin conductance level (SCL) also eliminates the need to adjust for decreasing baseline levels by de-trending individual epochs. This program was set to calculate the sum of all amplitudes, and the total area under the curve, for any response over 0.01 µS in the 1 to 3 s following each stimulus occurrence.

Once the overlapping SCRs had been processed, each win and each LDW incidence was time matched with the previous loss outcome. This matching procedure was performed in order to equate for falling tonic skin conductance levels over the course of the experiment. Including only the losses that occur at comparable points in time as wins and LDWs avoids the problem of falling levels and gives a more accurate representation of the responses to these outcomes. Although the number of epochs varied between individuals, an equal number of win and loss epochs, and an equal number of LDW and loss epochs were included in the analyses (as mentioned above, each participant experienced at least five epochs of each outcome type). The amplitude and area under the curve data for each epoch were averaged together based on outcome type (win, loss, LDW) for each participant. Because a different number of win and LDW outcomes (as well as time-matched loss outcomes) were experienced by each participant, the data for these outcomes were subjected to two separate 2 Group (PG, NPG) × 2 Outcome mixed-design analysis of variance (ANOVA). An independent samples t-test was performed to assess whether the tonic skin conductance levels, as assessed by the LedaLab program, differed between problem and non-problem gambler groups.
5.5. Results

5.5.1. Group characteristics and behavioural data.

The mean PGSI scores for the PG and NPG groups were 15.0 (SD = 4.4) and 1.2 (SD = 1.4), respectively. Scores for participants in the NPG group ranged from 0 to 3 (47% scored 0, 6% scored 1, 20% scored 2, and 27% scored 3), and scores for the PG group ranged from 8 to 24, out of a possible 27. Independent samples t-tests revealed that the PG and NPG groups did not significantly differ in the age (p = .288) or sex (p = .716) of participants. Loss outcomes were experienced most frequently (67.2% of trials), followed by LDWs (17.7% of trials), and wins (15.1% of trials). On average, each participant experienced 78 losses (SD = 49; range = 26-209), 20 LDWs (SD = 48; range = 7-54), and 17 wins (SD = 48; range = 5-51) within the testing session.

5.5.2. Physiological data.

Although tonic skin conductance levels of PGs (M = 6.05 µS, SE = 3.83) appeared to be slightly higher than NPGs (M = 5.24 µS, SE = 3.42), this difference was not significant (p = .544). Compared to losses, wins elicited significantly greater area under the curve (M_{win} = 5.05 µS, SE = 1.18; M_{loss} = 3.04 µS, SE = 1.34), F(1,28) = 4.77, p = .037, \eta^2_p = .10, but the difference for SCR amplitudes (M_{win} = .26 µS, SE = .08; M_{loss} = .17 µS, SE = .04) failed to reach significance (p = .057). No main effect of Group was found for either amplitude (p = .428) or area measures (p = .498). A significant Group × Outcome interaction revealed that, in the NPG compared to the PG group, wins elicited greater area under the curve, F(1,28) = 5.22, p = .030, \eta^2_p = .15, and SCR amplitudes, F(1,28) = 5.63, p = .025, \eta^2_p = .14; but minimal differences following losses for both groups (Figure 4). The mean SCR values for the Group and Group × Outcome interaction following wins and losses can be seen in Table 1.
Figure 4. Mean skin conductance response (SCR) following wins and time-matched losses for the problem gambler (PG; n = 15) and non-problem gambler (NPG; n = 15) groups. A) Raw grand average SCR waveforms for the time matched epochs for each outcome type. B) Driver data, as calculated by LedaLab for each outcome type (note the different scales for panels A and B); the vertical dashed line, labelled x, indicates the response likely caused by the accumulation of credits before a threshold of recognition for a win, whereas line y most likely indicates the peak response elicited by processing the significance of the actual win.
Table 1. Mean (and standard deviation) skin conductance response (SCR) values (µS) of the problem gambler (PG; n =15) and non-PG groups (NPG; n =15) for amplitude and area under the curve measures following wins, losses disguised as wins (LDWs), and losses

<table>
<thead>
<tr>
<th></th>
<th>PG (M, SD)</th>
<th>NPG (M, SD)</th>
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<tbody>
<tr>
<td><strong>Group Main Effect</strong></td>
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<td><strong>Win vs. Loss comparison</strong></td>
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<tr>
<td>Amplitude</td>
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<td>Area</td>
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<td>.23 (.04)</td>
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<td>Area</td>
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<td>3.98 (.79)</td>
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<td><strong>Outcome × Group Interaction</strong></td>
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<tr>
<td><strong>Win vs. Loss comparison</strong></td>
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<tr>
<td>Amplitude</td>
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<tr>
<td>Wins</td>
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<tr>
<td>Amplitude</td>
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</tbody>
</table>
Electrodermal activity following LDW outcomes was not significantly different from the activity following losses, in terms of SCR amplitude ($M_{LDW} = .23 \text{ µS, } SE = .04$; $M_{loss} = .22 \text{ µS, } SE = .05; p = .328$), or area under the curve ($M_{LDW} = 3.98 \text{ µS, } SE = .86$; $M_{loss} = 4.42 \text{ µS, } SE = 1.02; p = .234$). No main effect of Group was found for either amplitude ($p = .229$), or area measures ($p = .189$). The Group × Outcome interaction for the loss vs. LDW comparison was not significant for SCR amplitude ($p = .189$) or area under the curve ($p = .269$) (Figure 5). The mean SCR values for the Group and Group × Outcome interaction following LDWs and losses can be seen in Table 1.
Figure 5. Mean skin conductance response (SCR) following losses disguised as wins (LDWs) and time-matched losses for the problem gambler (PG; n = 15) and non-problem gambler (NPG; n = 15) groups. A) Waveforms representing the raw grand average SCR for the time-matched epochs for each outcome type. B) Driver data, as calculated by LedaLab for each outcome type (note the different scale for panels A and B).
5.6. Discussion

5.6.1. Physiological effects of wins vs. losses.

Problem gamblers demonstrated attenuated SCRs to win outcomes, suggesting a hyposensitive response to rewarding stimuli in affected individuals. This finding corroborates previous research using HR as an indicator of arousal (Goudriaan et al., 2006), and previous neuroimaging research showing evidence for reduced cortical activity in reward-related brain circuitry of problem gamblers after the experience of reward (de Ruiter et al., 2009; Reuter et al., 2005; cf. Miedl et al., 2010; van Holst et al., 2012). Consistent with previous research (Dixon et al., 2010; Lole et al., 2012; Sharpe, 2004; Wilkes et al., 2009), wins induced larger SCRs than losses in individuals familiar with gambling, but who do not report gambling-related problems, highlighting the motivational significance of rewards in EGM gambling. It is unlikely that the attenuated SCR following losses observed in the current study are due to the frequent occurrence of these outcomes, as reduced responses were also observed following less frequent LDWs. Moreover, the main focus of the current study was to examine the between-group differences in responding during actual gambling activity, which would not be affected by the frequency of occurrence of outcomes (i.e., problem gamblers and non-problem gamblers would be expected to experience the same proportion of win and loss outcomes).

The finding of reduced reward sensitivity of problem gamblers corroborates theoretical interpretations given by several neurobiological accounts that implicate impaired reward processing as the basis of problem gambling behaviours (Blum et al., 2000; Damasio, 1994), and has important ramifications for conceptualisations of the nature of the deficit in this disorder. The apparent hyposensitivity to reward in problem gamblers may be caused by malfunctioning in the cortical regions associated with
incentive value processing, such as the mesolimbic-dopaminergic reward system (de Ruiter et al., 2009; Holroyd & Coles, 2002; Volkow et al., 2004), or areas of the brain associated with generating appropriate emotional responses to these outcomes (Damasio, 1994). Such deficits may at least partly explain the maladaptive behaviours displayed by problem gamblers, including increased reward-seeking behaviour (Blum et al., 2000) and/or sub-optimal decision making (Damasio, 1994).

The hyposensitive response to reward does not provide support for arousal-based models of problem gambling that predict problem gamblers evaluate wins as more significant, and will thus, show greater physiological reactions to these outcomes (e.g., Sharpe & Tarrier, 1993). Conversely, this pattern of responding may corroborate other arousal-based theories that posit problem gamblers are hypoaroused and use gambling to achieve an optimal state of functioning (e.g., Brown, 1986; Jacobs, 1986; cf. Cocco, Sharpe, & Blaszczynski, 1992). Taken with our finding that the tonic arousal levels did not differ between problem and non-problem gamblers on the day of gambling, this reward hyposensitivity could be an aspect of a general state of hypoarousal in this disorder. However, to confirm this, lower tonic baseline measures will need to be demonstrated among PGs on non-gambling days.

The precise mechanisms underlying the attenuated response to reward exhibited by problem gamblers could not be determined in the current study and should be the focus of future research. It could be argued that PGs demonstrate attenuated SCRs to wins due to the effects of repeated exposure to gambling activity rather than an inherent hyposensitivity to reward. Specifically, because PGs gamble more frequently, they may have become more accustomed to wins compared to non-PGs and do not perceive them to be as salient as they once did. Further research on the lifetime trajectory of these responses is required in order to determine the extent that overexposure to gambling
activity and genetic predispositions contribute to the development of problematic gambling behaviours. Nevertheless, the attenuated SCRs to rewarding outcomes observed in the current study may be used as a marker for deficit in this disorder if further research verifies it as robust.

5.6.2. **Physiological effects of losses disguised as wins.**

The current study was the first to examine the psychophysiological reactions to losses disguised as wins (LDWs) during actual gambling activity in problem and non-problem gamblers. These outcomes were not found to elicit electrodermal responses that were significantly different to losses in either group. This finding is in contrast with results previously reported by Dixon et al. (2010), who found that SCRs and HR responses to LDW outcomes were comparable with those following wins, which were both significantly different from losses. This discrepancy is possibly due to the fact that their study examined responses only in novice undergraduate gamblers in a laboratory setting, and not experienced gamblers who may have become accustomed to such events. Such an interpretation is interesting, as it suggests that occurrences of LDWs may be important to the development, but not to the maintenance of gambling behaviours, and that this problem gambling may have a distinct lifetime trajectory. Specifically, when people gamble for the first few times they may be more excited by both wins and LDWs, but as time progresses, they may only be excited by true and/or larger wins. As mentioned above, further research is required into the influence of repeated exposure to gambling activity on the developmental trajectory of this disorder; in particular, whether PGs have habituated to wins and losses disguised as wins (albeit at differential rates, i.e., responses to LDWs may be habituated to more quickly than wins).
5.6.3. **Limitations and future directions.**

Unfortunately, precise estimates of the amount of previous exposure to gambling activity that the participants of this study had were not explicitly quantified. In order to minimise disruption to patrons and business within the gaming venue in which the data were collected, only the PGSI was administered, and not the full Canadian Problem Gambling Index. The latter would have given a clearer indication of the experience participants had with gambling. Nevertheless, since nearly half of the NPG group scored 2 or 3 on the PGSI it is assumed that participants in the current study were more familiar with gambling activity than college students who merely participate in gambling research in return for course credits, or other small reward (e.g., participants in the Dixon et al., 2010 study scored either 0 or 1 on this measure).

Since the recording device used in the current study allowed only four different event markers to be inserted into the physiological data record, an examination of psychophysiological responses following different sized bets or different magnitude outcomes could not be conducted. Because Small and Large win types were averaged together, the results are likely to represent responses to the more frequently experienced small wins. As mentioned above, it is possible that, while problem gamblers appear to be hyposensitive to small wins that occur on EGMs, they may be more responsive to the experience of significantly larger wins and/or bonus features. Alternatively, they may need to larger wins to feel the same level of excitement as non-problem gamblers feel toward small wins.

As previously mentioned, wins and LDWs encountered during EGM gambling are associated with the gradual accumulation of credits, whereas losses are quickly identified as such. Because such presentation of loss and non-loss outcomes are an inbuilt design feature of EGMs, the differential latencies associated with recording these
events were unavoidable and could influence responses. Future research may choose to examine the effect the anticipation of a potential win associated with accumulating credits (as opposed to the identification of the actual outcome) has on psychophysiological responses. Another limitation of the current research was the presence of researchers during the recording process, which possibly influenced participants to gamble differently from how they normally would; however, this effect was constant and unlikely to be responsible for between-event effects observed in the study. To overcome such problems, future research could use a video camera to record the participant gambling, and later match the occurrence of events with the resultant physiological reactions.

Finally, anecdotal evidence has suggested that problem gamblers may be motivated primarily by the experience of bonus ‘features’ during play. Unfortunately, because not all participants in the original sample experienced these outcomes, there was not sufficient power to allow analysis of ‘features’ on normal and abnormal gambling. Future examination of the physiological responses that occur in response to these outcomes may help to further elucidate the motivations of gamblers on EGMs.

5.6.4. Conclusion.

This study is the first to investigate the phasic physiological reactions to gambling outcomes while problem gamblers and non-problem gamblers wager their own money on EGMs in a real gaming environment. Problem gamblers were found to exhibit attenuated responses to reward, whereas wins were found to elicit greater SCRs than losses for non-problem gamblers. These findings suggest that a hyposensitivity to reward that may underlie the problematic behaviours characteristic of this disorder, such as gambling with larger amounts of money and for longer periods of time, presumably
in order to experience the same excitement and satisfaction as non-PGs. Responses following LDW outcomes were not found to differ from losses in either group. While further research is necessary to validate these results, the current study highlights the potential value of this apparent hyposensitive response as a biological marker for this disorder.
CHAPTER SIX – STUDY C

Can Event-related Potentials Serve as Neural Markers for Wins, Losses, and Near-wins in a Gambling Task? A Principal Components Analysis

6.1. Preamble


Chapter Six outlines the third empirical paper of the current thesis. This paper sought to elucidate the latent nature of two event-related potential components, the feedback-related negativity (FRN) and the P300 subcomponent, that have been previously shown to index incentive value processing, and to verify whether these can reliably differentiate win, loss, and near-win outcomes in healthy controls. Aspects of this study were presented at the 22nd Annual Conference for the Australasian Society for Psychophysiology, Sydney, Australia, 28-30 November, 2012 (Appendix N), and at the 51st Annual Meeting of the Society for Psychophysiological Research, Boston, U.S.A., 14-18 September, 2011 (Appendix O).
6.2. **Abstract**

Originally, the feedback-related negativity (FRN) ERP component was considered to be a robust neural correlate of non-reward/punishment processing, with greater negative deflections observed following unfavourable outcomes. More recently, it has been suggested that this component is better conceptualised as a positive deflection following rewarding outcomes. The current study sought to elucidate the nature of the FRN, as well as another component associated with incentive-value processing, the P3b, through application of a spatiotemporal PCA. ERPs were recorded while 17 healthy controls played a computer gambling task that closely simulated events on an electronic gaming machine (EGM). Participants received feedback on credits won or lost on each trial. The frequencies of outcomes (e.g., wins, losses) closely matched outcomes dispensed by a real EGM, with frequent losses, and infrequent wins and near-wins. The PCA revealed that feedback elicited both a frontally maximal negative deflection to losses and a positive deflection to wins (which was also sensitive to reward magnitude), implying that the neural generator/s of the FRN are differentially activated following these outcomes. As expected, greater P3b amplitudes were found for wins compared to losses. Interestingly, near-wins elicited significantly smaller FRN amplitudes than losses (with no differences in P3b amplitude), and may contribute to the maintenance of gambling behaviours on EGMs. The results of the current study are integrated into a response profile of healthy controls to outcomes of varying incentive value. This may provide a foundation for the future examination of individuals who exhibit abnormalities in reward/punishment processing, such as problem gamblers.
6.3. **Introduction**

The neural mechanisms involved in the processing of reward and non-reward/punishment are of particular relevance to addictive disorders, such as problem gambling, as abnormalities in incentive value processing are believed to be one of the causal factors in such disorders. For example, problem gamblers may be hyposensitive to non-reward/punishment (e.g., Reuter et al., 2005) and thus, the repeated losses experienced during gambling activity may not perceived by problem gamblers to be as aversive compared to non-problem gamblers. Instead, they may be hypersensitive to reward (e.g., Hewig et al., 2010; Oberg et al., 2011) and pursue wins more persistently despite their infrequent occurrences; or they may be chronically hyposensitive to reward (e.g., Blum et al., 2000) and engage in compensatory thrill-seeking behaviour (such as trying to obtain large wins) in order to reach the same level of excitement associated with smaller wins in non-problem gamblers.

A particularly valuable index of incentive value processing is the feedback-related negativity (FRN), an apparently robust and reliable ERP component sensitive to valence manipulations. The FRN is maximal at fronto-central scalp sites and there is consensus that medial frontal cortical areas, especially the anterior cingulate cortex (ACC), are involved in its generation (Bellebaum & Daum, 2008; Miltner et al., 1997; Nieuwenhuis, Yeung, Holroyd, Schurger, & Cohen, 2004). Because of potential links to reward mechanisms through activation of the mesencephalic dopamine system (Holroyd & Coles, 2002), the FRN has major significance, particularly for gambling behaviours, as it provides a window through which the effects of reward and non-reward outcomes within the brain might be usefully examined.

Recently, there has been debate regarding the nature of this ERP component; specifically, whether it is best conceptualised as a negative deflection following
unfavourable outcomes or as a positive deflection following favourable outcomes.

Earlier conceptualisations, portrayed the FRN as being characterised by greater negative responses 250-350 ms following feedback that signals monetary losses compared to gains (San Martin et al., 2010; Toyomaki & Murohashi, 2005; Yeung et al., 2005), or by the least desired of two possible outcomes within a certain context (e.g., zero credits elicited larger FRNs than wins when the alternative outcome is to gain credits, compared to when the alternative is to lose credits; Gehring & Willoughby, 2002a; Holroyd et al., 2004), during tasks that resemble gambling activity. Subjective expectancy of an outcome has also been shown to affect the FRN, with larger amplitudes associated with unexpected compared to predicted negative outcomes, although this effect appears to be more subtle and may not always follow objective probabilities of such events (Hajcak et al., 2005; Hajcak et al., 2006). Although the link between FRN and valence appears consistent, manipulations of incentive value have yielded equivocal results. Specifically, some studies suggest that larger losses (compared to smaller losses) and smaller gains (compared to larger gains) yield larger FRN magnitudes (e.g., Bellebaum et al., 2010; Holroyd et al., 2004), whilst others have found no magnitude effects (e.g., Gu et al., 2010; Hajcak et al., 2006; Yeung & Sanfey, 2004).

Although the negative deflection to unfavourable outcomes described above has been reported in a wide variety of circumstances, including simulated gambling (Hewig, Trippe, Hecht, Coles, Holroyd, & Miltner, 2007), guessing tasks (Hajcak et al., 2006; Hajcak et al., 2005, 2007), time estimation tasks (Holroyd & Krigolson, 2007; Miltner et al., 1997; Nieuwenhuis et al., 2005), and learning tasks (De Pascalis et al., 2010), the true nature of the FRN remains somewhat unclear, as this component is commonly superimposed on large amplitude P300 responses that occur immediately after it. It has
been proposed that the reduced amplitude FRN observed following win outcomes may not be an actual attenuated response to these events, but is rather driven by larger P300 amplitudes following favourable outcomes (Yeung & Sanfey, 2004). Furthermore, the relative contribution of negative and positive outcomes to the FRN remains unclear due to the fact that many studies have employed the computation of a difference waveform to measure FRN magnitude (e.g., Dunning & Hajcak, 2007; Foti & Hajcak, 2009; Hajcak et al., 2007; Holroyd et al., 2008; Miltner et al., 1997). Recent research has suggested that, rather than a negative deflection to non-reward outcomes, the FRN is better conceptualised as a positive deflection that is greater following reward compared to non-reward outcomes (Foti et al., 2011; Holroyd et al., 2003; Holroyd et al., 2008).

Regardless of the actual response pattern, investigation of the latent spatial and temporal characteristics of this feedback-related ERP component (whether it be a negative deflection to non-reward or a positive deflection to reward) in healthy controls using a PCA will allow a more reliable and accurate account of the neural correlates associated with incentive value processing. This will encourage the future examination of whether these responses differ in individuals who display deficits in outcome evaluation, such as those with gambling problems.

Typically examined as a global component, the P300 (called the LPC in some studies), has also been shown to be sensitive to various aspects of incentive value on tasks that simulate gambling (Bellebaum et al., 2010; Hajcak et al., 2007). The inverse relationship between probability and P300 amplitude has been well established (Donchin & Coles, 1988), although the understanding of these results is subject to different interpretations (see Gonsalvez et al., 2007; Verleger, 1988). Nevertheless, studies that have controlled for event probability have demonstrated that the P300 remains sensitive to win and loss outcomes (e.g., Hajcak et al., 2007; Wu & Zhou,
2009; Yeung et al., 2005; Zhou et al., 2010), although the pattern of these results is somewhat variable. Some studies report a double dissociation between the FRN and P300, showing the FRN to be affected by valence but not reward magnitude, with the opposite pattern for the P300, regardless of whether the outcome is of positive or negative valence (Sato et al., 2005; Yeung & Sanfey, 2004). In contrast to this, other research has demonstrated that the P300 is influenced by valence, with wins eliciting larger amplitudes than losses (Hajcak et al., 2007; Toyomaki & Murohashi, 2005). Because the P300 is established to be a complex comprising several sub-components, it is possible that different subcomponents are independently sensitive to valence and magnitude. For instance, stimulus salience is known to affect the P3b and win events may elicit larger P3bs on account of their greater salience than losses. Therefore, it is of value to determine which of the sub-components of the P300 are affected by win and loss outcomes.

The current study used a spatiotemporal PCA, to examine the latent nature of both the FRN and the LPC ERP subcomponents, that may not be perceptible using traditional ERP data extraction methods, in response to manipulations of valence and magnitude within a simulated electronic gaming machine (EGM; also called a ‘poker’ or ‘slot’ machine) task. EGMs typically deliver a large number of win and loss outcomes in a short period of time and are of particular clinical significance to problem gambling. A higher percentage of gamblers seeking treatment report addiction to EGMs (see Dowling et al., 2005) compared to other gambling activities. Furthermore, EGM gambling is associated with a faster progression of addiction (Breen & Zimmerman, 2002) and more severe symptoms (Petry, 2003). In the current study all key EGM outcomes were of interest, including large and small wins, losses, and near-wins (see Method section 2.2.2. for details on these outcomes). Traditional ERP research has
shown near-wins to be less aversive (Luo et al., 2011) and more rewarding than losses (Qi et al., 2011), and neuroimaging research has shown that, while these outcomes are rated as more unpleasant than losses, they increase motivation to gamble by recruiting reward related brain circuitry (Clark et al., 2009). The current study sought to examine whether the latent neural correlates of incentive value processing are differentially activated for these outcomes compared to losses, in order to evaluate their role in the development and maintenance of gambling behaviours.

In summary, the current study sought to utilise a PCA to parse two ERP components previously found to index various aspects of incentive value processing from overlapping data, and to evaluate their capacity to discriminate between win, loss, and near-win outcomes, as well as rewards of different magnitudes.

6.4. Method

6.4.1. Participants.

Seventeen undergraduate psychology students (7 male, 10 female, \( M_{age} = 18.7 \) years; \( SD = 4.8 \), age range = 18-23 years) from the University of Wollongong participated in the experiment in return for course credit. Participants reported they abstained from using nicotine, alcohol, or prescription/illicit drugs in the two hours prior to testing, and reported no history of severe brain injury or seizures. Written informed consent was obtained from all participants, who were advised that participation was entirely voluntary, and that they could withdraw from the study at any time (Appendices P and Q). The study’s protocol was approved by the University of Wollongong Human Research Ethics Committee.
6.4.2. **Materials.**

6.4.2.1. **Physiological recording equipment.**

EEG was recorded from a 19-site electrode cap (comprised of tin electrodes fitted in the standard international 10-20 system layout, see Figure 6) using NuAmps 2.0 software (NeuroScan Compumedics, USA). The electrodes were referenced to linked earlobes and grounded by a cap electrode located mid-way between FPz and Fz. Vertical eye movement (vEOG) was monitored with two tin cup electrodes: one placed 2 cm above and the other 2 cm below the left eye. Horizontal eye movement (hEOG) was monitored with two tin cup electrodes placed adjacent to the outer canthus of each eye. Impedance was less than 5 KΩ for cap electrodes and less than 3 KΩ for EOG and reference electrodes. Scalp EEG potentials were amplified × 20 000, EOG potentials were amplified × 5000, and both were sampled at a rate of 500 Hz.

*Figure 6. A) The layout of electrodes according to the international 10-20 system. B) An example of how the 19 site electrode cap is fitted on participants.*
6.4.2.2. **Computer gambling task.**

A gambling task was administered using Presentation software, version 13.0 (Neurobehavioral Systems, Inc., USA) (Figure 7). The task was modelled on games commonly run on EGMs. The screen display comprised a single row of four fruit symbols. Because eight different fruit symbols were used, and each could appear at random in each of the four columns, each trial was unique in terms of its stimulus configuration (symbol and sequence), apart from Large win outcomes, where each of the eight combinations possible could occur numerous times. After each trial, the amount wagered on that trial, the number of credits left to play with, and the amount won (if anything) were prominently displayed, as is typical of EGM displays. On each trial, one of four outcomes were possible: four identical symbols constituted a Large win, yielding ten times the amount bet; Small wins were indicated by three identical symbols that occurred in sequence, yielding five times the amount bet; Near-wins occurred when one different symbol was inserted between three identical symbols, and no credits were returned; and Losses occurred when neither a win nor near-win occurred, with no credits returned. The probability of the three main outcome types (wins, losses, and near-wins) closely matched outcomes from a real EGM (Wilkes et al., 2010). Although presented randomly, an equal number of Near-win (15%) and Win (Small win: 7.5%; Large win: 7.5%) outcomes were presented, with the remaining trials being Losses (70%). The current task differed from commercially available EGMs in that the visual and auditory stimuli that typically accompany EGM play were eliminated, so that ERPs to gambling outcomes were not confounded by these factors.
Participants did not wager their own money, but started the session with a free allocation of 5000 credits (valued at AUD $50). Each trial commenced with participants choosing to either ‘Bet Low’ or ‘Bet High’ by pressing either a ‘Bet 1’ or ‘Bet 10’ button, respectively. Following this, a long inter-stimulus interval of 4 s (± 400 ms), where the reels appeared to spin, was inserted between bet placement and the outcome (all reels stopped ‘spinning’ simultaneously) which ensured that the epoch of interest was not contaminated by EEG activity associated with the immediately preceding event (i.e., activity related to the bet). Following the outcome presentation, a delay of 2 s occurred before the next bet could be placed (signalled by the ‘Bet’ button turning from red to green).

6.4.3. **Procedure.**

After providing informed consent, participants anonymously completed the Canadian Problem Gambling Index (CPGI, Ferris & Wynne, 2001), which was used as a screening tool to assess diagnostic status and severity of gambling behaviours. None of the participants in the current study met the criteria for problem gambling (score of 8 or above). Participants also completed questionnaires that assessed their levels of reward and punishment sensitivity, gambling-related cognitions, levels of depression and anxiety, and impulsivity; due to low variability in these scores, the results of these
inventories will not be reported here. Participants were then fitted with the physiological recording equipment and instructed on how to play the gambling task.

Eye movement data were obtained using an eye calibration task (Croft & Barry, 2000) that allows the offline correction of eye artifacts in task-related brain activity. After engaging in ten practice trials, participants played 450 trials on the gambling task. They were informed they would commence the task with 5000 credits and that they would win one entertainment voucher (valued at AUD $12) if they had accumulated more than 6000 credits at the end of the experiment, and two vouchers if they had accumulated 7000 or more credits. Immediately after the gambling task, participants completed a subjective experience questionnaire (Appendix E) that assessed their levels of task related enjoyment and excitement (e.g., ‘How excited did you feel when you experienced a Large win/Small win/Near-win/Loss while playing the poker machine’, with answers ranging from 1 = not excited to 9 = very excited).

6.4.4. **Data reduction and analysis.**

Ocular artifacts were removed using an eye-movement correction algorithm (Croft & Barry, 2000). EEG data were low-pass filtered below 30 Hz (24 dB) and baseline corrected relative to the pre-stimulus interval (100 ms). Trials that contained muscle or other artifact were manually identified and excluded from further analysis. Once all artifacts were removed from the data, ERPs ranging from 100 ms to 800 ms post-stimulus were created for each participant over 19 sites (Fp1, Fp2, F7, F3, Fz, F4, F8, C7, C3, Cz, C4, C8, P7, P3, Pz, P4, P8, O1, O2). Because there were not enough epochs for Small and Large wins over the two Bet options to allow an examination of Outcome × Bet size interaction effects (see Results section 3.1), and because preliminary analyses showed the effect of Bet size was small (see Figure 8A for effects at Pz and Figure 10A
for effects at Fz), we averaged the data from the Bet Low and Bet High epochs together to derive four Outcome types (Large wins, Small wins, Losses, Near-wins).

Furthermore, to avoid possible habituation effects on the feedback-related response (FRR) component/s being considered in statistical analyses, only the first 15% of Loss outcomes were included in the final analyses.

In order to examine the latent ERP components associated with incentive value processing, a two-step spatiotemporal PCA was conducted on the data using the ERP PCA (EP) Toolkit, version 2.23 (Dien, 2010b). This data reduction technique was chosen over a temporospatial PCA, as the topographies of the ERP components of interest are well-established in the literature, and we wished to minimise the possibility of excluding ERP components with similar latencies but different topographies. A spatial PCA was first conducted on the data in order to identify the variance accounted for by electrode sites over all time points, participants, and conditions. A temporal PCA, which uses all time points over all participants and conditions as variables, was then performed on the spatial components. The covariance matrix with Kaiser normalisation was used for all PCAs. In accordance with the recommendation of Dien (2010a), INFOMAX rotation was performed for the spatial step of the PCA, and PROMAX rotation was performed for the temporal step of the PCA.

Once ERP components relevant to the paradigm used were identified, the data for each component were submitted to a four Outcome (Large win, Small win, Loss, Near-win) repeated measures analysis of variance (ANOVA). Planned contrasts were conducted to examine differences between Wins (Large and Small combined) and Losses (Losses and Near-wins combined), Large wins and Small wins, and between Losses and Near-wins.
6.5. **Results**

6.5.1. **Behavioural data and reports of subjective experience.**

The mean PGSI scores for participants in the current study was 1.6 ($SD = 1.6$). On average, participants selected the Bet Low option on 38.9% of trials. Eight of the seventeen participants chose to Bet High at least 75% of the time, with seven of the total number of datasets containing five or fewer epochs in at least one of the win conditions (Bet Low/Small win, Bet Low/Large win, Bet High/Small win, Bet High/Large win), and fifteen of the total sample containing ten or fewer epochs. Thus, as previously mentioned, outcomes were collapsed across the two bet options for analysis.

A repeated measures ANOVA revealed participants rated Large wins ($M = 5.47$, $SD = 1.91$) as more exciting than Small wins ($M = 3.59$, $SD = 1.46$), $F(1,16) = 48.76$, $p < .001$, $\eta^2_p = .75$, and Small wins as more exciting than Losses ($M = 2.35$, $SD = 1.45$), $F(1,16) = 8.25$, $p = .011$, $\eta^2_p = .34$).

6.5.2. **Physiological data.**

Dien (2010b; 2012) currently recommends using the parallel test (Horn, 1965), which compares the amount of variability explained in the observed dataset using the Scree test (Cattell, 1966) to that derived from uncorrelated variables within a dataset of totally random noise, to determine the number of factors to retain in the first step of a PCA. When applying this criterion, two site groupings (parietal and central, centred on Pz and Fz, respectively) were identified to account for the majority of variance in the dataset.

The minimum percentage criterion calculated by the ERP PCA Toolkit was used to determine the number of temporal factors to be included in the second step of the PCA, as the parallel test recommended only four factors to be included, and none of
these corresponded to the FRR ERP component/s. According to this criterion, seven temporal factors were found to best represent the data.

Parietal factor SF1/TF1, and frontal factor SF2/TF3, most closely corresponded to the P3b and FRN, respectively, in terms of their topography, latency, and response to experimental manipulations. Two of the six remaining parietal factors (positive Slow wave and Error positivity), and two of the six remaining frontal factors (negative Slow wave and P200), were identifiable ERP components; however, these either did not show factor score differences between wins and losses, are of less theoretical importance, and/or are unrelated to the objectives of the current paper, so will not be discussed further.

6.5.2.1. P300.

Although both win and loss outcome types appeared to differ according to bet size (Figure 8A), preliminary Outcome × Bet analyses showed these effects were not significant. Thus, as mentioned above, because there were an insufficient number of epochs for a comparison of small and large wins following decisions to Bet Low and to Bet High, the PCA was conducted on data collapsed across the two Bet options.

The grand average virtual ERPs created by the EP toolkit were found to closely resemble the grand average raw ERP waveforms for this dataset at site Pz (SF1) (Figure 8B). Figure 8C shows how the grand average virtual ERP is comprised of the individual parietal factors that were identifiable ERP components, and/or accounted for at least 1% of the variance in the data.
Figure 8. Waveform data corresponding to the parietally maximal ERP components (SF1; n = 17). A) The raw ERP waveforms based on outcomes for each bet size at site Pz. B) The raw ERP grand average and virtual ERP grand average (over all outcomes and bet types) at site Pz, where the effect of the P3b was found to be greatest. C) The virtual ERP components averaged over all outcomes at site Pz for the five individual temporal factors that were identifiable ERP components, and/or accounted for more than 1% of the variance in the dataset.
The factor that corresponded to the P3b ERP component (SF1/TF1) showed significantly larger amplitudes for Wins compared to Losses, 472 ms post-outcome, $F(1,16) = 61.76, p < .001, \eta_p^2 = .79$. Amplitudes did not differ between Small and Large wins, or between Losses and Near-wins (Figure 9).

*Figure 9.* A) Virtual ERP waveforms for each outcome type for spatiotemporal factor SF1/TF1 at site Pz ($n = 17$). This factor was identified as corresponding to the P3b ERP component. B) Scalp topographies demonstrating the difference between Win types, Near-wins, and Losses 472 ms post-outcome.
6.5.2.2. *Feedback-related responses.*

The raw waveforms for the average ERP responses following decisions to Bet High and to Bet Low for each Outcome type following can be seen in Figure 10A. The grand average virtual ERP was also found to closely resemble the grand average raw ERP waveforms for this dataset at site Fz (SF2) (Figure 10B). Figure 10C shows how the seven individual factors included in the PCA for this site constitute the grand average virtual ERP.
Figure 10. Waveform data corresponding to the frontally maximal ERP components (SF2; n = 17). A) The raw ERP waveforms based on outcomes for each bet size at site Fz. The highlighted section includes the peak that would have been considered to be the FRN using traditional (although non-difference waveform) quantification methods. B) The raw ERP grand average waveform and the virtual ERP grand average waveform (over all outcomes and bet sizes). C) The virtual ERP components averaged over all outcomes at site Fz for the four individual temporal factors that were identifiable ERP components, and/or accounted for more than 1% of the variance in the dataset.
The factor that most closely corresponded to the FRN ERP component (SF2/TF3) peaked at 290 ms post-outcome, and was characterised by both a negative deflection following Losses, and a positive deflection following Wins (Figure 11). The difference between win-loss outcomes was found to be statistically significant, $F(1, 16) = 126.85, p < .001, \eta^2_p = .89$. Henceforth, negative deflections of this factor following losses will be referred to as the FRN, and positive deflections to wins will be called the feedback-related positivity (FRP). Significantly larger FRP amplitudes were observed following Large wins compared to Small wins, $F(1, 16) = 7.21, p = .016, \eta^2_p = .31$. FRN amplitude following Losses were found to elicit significantly greater negative amplitudes compared to Near-wins, $F(1,16) = 23.24, p < .001, \eta^2_p = .59$. 
Figure 11. A) Virtual ERP waveforms for each outcome type for spatiotemporal factor SF2/TF3, which was maximal at Fz (n = 17). This factor was identified as corresponding to the feedback-related negativity and the feedback-related positivity ERP components. B) Scalp topographies demonstrating the difference between wins, near-wins, and losses, 290 ms post-outcome.

6.6. Discussion

The current study used a PCA to investigate the latent nature of two ERP components previously found to index various aspects of incentive value processing. Specifically, we aimed to examine whether the feedback-related responses (the FRN and the FRP) and the P3b could be considered distinct neural measures, and to determine whether they can be used as indices of the cognitive processing of outcome valence and reward magnitude in an ecologically-valid gambling paradigm.
6.6.1. **Physiological effects of wins vs. losses.**

6.6.1.1. **Feedback-related responses.**

By using a spatiotemporal PCA to parse the FRN from overlapping ERP components, we demonstrated that loss outcomes are reflected by a negative deflection approximately 290 ms post-feedback, a result consistent with previous FRN findings using traditional quantification methods (Gehring & Willoughby, 2002a; Hajcak et al., 2005; Hajcak et al., 2006; Holroyd et al., 2006; Yeung et al., 2005; Yeung & Sanfey, 2004). Conversely, we also demonstrated that win outcomes elicit a positive deflection at the same latency, corroborating the findings from other research (Foti et al., 2011; Holroyd et al., 2003; Holroyd et al., 2008). The results of the current study serve to consolidate and help explain the disparate findings of the extant literature. It also elucidates how outcomes of different incentive value are processed in the brain, something that is of major significance, as it affords the examination of the effects of both reward and punishment via independent neural correlates that are likely to be generated within the same cortical structure/s and/or system/s. The reinforcement learning theory (Holroyd & Coles, 2002), posits that outcomes are evaluated within the mesencephalic dopamine system based on previously learned expectations. Within this system, the basal ganglia monitor outcomes and stimulate dopamine levels: accordingly, rewarding outcomes cause dopamine to be released, leading to feelings of pleasure, whereas worse-than-expected outcomes are associated with reduced dopamine transmission (Fiorillo, Tobler, & Schultz, 2003; Schultz, 2007). The results of the current study are compatible with this conjecture; the observed FRN deflection to negative feedback is likely to reflect the disinhibition of the ACC, whereas the FRP following reward outcomes reflects inhibition of the ACC, serving to guide future behaviours to aversive and appetitive stimuli, respectively.
Despite the probability of reward being controlled for in the analysis of the current study (Method, Section 2.4), the positive response to win outcomes observed may be exaggerated due to participants perceiving rewards to occur less frequently during the course of the experiment; however, a large positive deflection (i.e., the FRP) to reward outcomes, similar to that found in the current experiment, was also found in a recent study that used a temporospatial PCA to examine equiprobable win and loss outcomes in a simple two-choice gambling task (Foti et al., 2011). That study also found a much smaller FRN to loss outcomes, the reasons for which are unclear. Such a result would not be predicted by stimulus frequency effects (losses occurred comparatively more frequently in the current study). It is possible that the more centrally maximal distribution of the component identified by Foti and colleagues may indicate a separate ERP component that is different from the feedback-related responses of the current study, or may reflect the different paradigms used; further research is required to consolidate these findings.

Indices of incentive value processing, including the FRN and FRP, are particularly relevant in individuals who display abnormalities in the way rewarding and/or punishing stimuli are evaluated. For example, attenuated or accentuated amplitudes of the positive deflection following wins in problem gamblers may respectively indicate a hyposensitivity or hypersensitivity to rewards, whereas reduced amplitudes of the negative deflection following losses may indicate a hyposensitivity to punishment. If future research confirms that abnormal responses to win and loss stimuli observed in the current study can reliably differentiate problem gamblers from non-problem gamblers, then such a pattern may eventually be used as a biological marker for addiction, and provide a foundation on which clinical treatments and interventions can be developed.
6.6.1.2. **P3b.**

As predicted, we found larger P3b amplitudes for wins compared to losses, a result consistent with prior reports on global P300 amplitudes by some researchers (e.g., Hajcak et al., 2007), but not by others (Sato et al., 2005; Yeung & Sanfey, 2004). Thus, we did not corroborate the double-dissociation pattern suggested previously (Sato et al., 2005; Yeung & Sanfey, 2004), as both P3b and FRN were seen to be sensitive to valence. Although the probability of win (15%), near-win (15%), and loss events (70%) were not matched in the EGM task of the current study, probability differences between these events fail to explain the differences observed. For instance, larger P3b amplitudes were observed to win events compared to equiprobable near-win events (see Figure 9). While it is theoretically possible that probability effects may have cancelled out an actual difference between near-wins and losses, this is unlikely because the two individual effects would be expected to work in the same direction: lower probability and great salience for near-wins would be expected to increase P3b effects. We propose that the larger P3b amplitudes observed for wins (compared with losses) are associated with the motivational significance of rewards to the individual in response to ecologically-valid stimuli, reflecting factors other than probability that are known to affect the P300 (Johnson, 1986).

6.6.2. **Physiological effects of different magnitude rewards.**

The current study added to previous research by examining whether reward magnitude affects FRP and P3b amplitude. The majority of previous studies have found that the traditionally conceptualised FRN is not sensitive to outcome magnitude (Hajcak et al., 2006; Sato et al., 2005; Yeung & Sanfey, 2004; Yu & Zhou, 2006a; cf. San Martin et al., 2010), indicating that it reflects a dichotomous evaluation of outcomes, with events
being contextually better or worse, with no response differences for intermediate-sized outcomes (Hajcak et al., 2006). The finding that the feedback-related responses in the current study not only differentiates the valence of outcomes, but also that the FRP is sensitive to the magnitude of win outcomes is exciting, particularly for the future examination of problem gambling. It could help determine whether the behaviours that are characteristic of this disorder stem from abnormal responses to large and small wins; for example, an attenuated response to small, but not large wins, may suggest a predisposition to seek larger rewards despite personal cost.

Unlike the FRP, similar P3b component responses were found following small and large wins, a result that contrasts with earlier research (Sato et al., 2005; Yeung & Sanfey, 2004). It is unlikely that this result was due to the lack of difference in the perceived value of the amounts returned for Large and Small wins, as these amounts were comparable to those returned in previous studies where differences in P300 amplitude were observed for different magnitude wins (e.g., Bellebaum et al., 2010). Furthermore, although the subjective experience questionnaire did not specifically assess the perceived value of credits returned in the current task, participants did rate Large wins as more exciting than Small wins.

6.6.3. The significance of near-wins.

While P3b amplitudes following near-wins suggest they are not as rewarding as wins (Figure 9), these outcomes were associated with reduced FRN amplitudes (Figure 11), implying that they are subjectively experienced as less aversive than full-loss outcomes, despite returning nil credits. These results are consistent with previous ERP research (Luo et al., 2011; cf. Qi et al., 2011), where near-wins resulted in reduced FRN, but no difference in P300 amplitudes, and suggest that these frequently occurring outcomes
that are unique to EGM gambling, are an important design feature of this gambling medium and may play a role in the development and maintenance of gambling behaviours by reducing the unpleasant nature of loss outcomes.

6.6.4. **Limitations and future directions.**

Although attempts were made to construct an ecologically-valid task, with stimulus characteristics, event-types, and bet options simulating real gambling conditions, key differences between the laboratory task used in the current study and actual gambling need to be acknowledged. In real gambling, significant sums of money are wagered, won, and lost within a short period, whereas in previous laboratory-based studies (e.g., Lole et al., 2012; Wilkes et al., 2010), and the current study, rewards were restricted to small amounts (movie voucher/s in the current study). Further, unlike real gambling, the participant does not suffer the risk of losing their own money, with losses often restricted to loss of free credits allocated to the research participant. It is often assumed that findings from controlled laboratory conditions will be replicated in, and may be generalised to actual gambling behaviours, but this remains to be demonstrated. Within the autonomic realm, research has examined electrodermal and cardiac data in real club settings when patrons bet with their own money (e.g., Coventry & Constable, 1999; Diskin & Hodgins, 2003; Griffiths, 1993; Krueger et al., 2005; Meyer et al., 2000). In a similar context, the implications of ERP findings for problem gambling have been alluded to in most laboratory studies, although we found no more than two studies that compared ERPs in response to reward and punishment sensitivity between problem and non-problem gamblers (Hewig et al., 2010; Oberg et al., 2011). The application of such information in the applied domain is clearly warranted and an urgent priority, although ethical and technical issues (e.g., challenges of recording reliable ERPs in a live-
gambling environment) remain major challenges. Nevertheless, laboratory studies, where variables may be manipulated without the confounding factors that are encountered in vivo (e.g., the presence of others, financial implications, ingestion of psychoactive substances), are essential to establish a foundation of knowledge on which future applied research can build.

6.6.5. Conclusion.
The application of a spatiotemporal PCA in the current study allowed an examination of the feedback-related responses and P3b without the confounding influence of overlapping ERP components. The results of the feedback-related responses from the current study consolidate those from previous research, in that two distinct ERP components sensitive to outcome valence occur at the same latency post-feedback; the FRN, characterised by a negative deflection to losses, and the FRP, characterised by a positive deflection to wins. The P3b was also confirmed to be a reliable index of reward valence, with greater amplitudes following win compared to loss outcomes. The current study was the first to examine the latent nature of the responses to different sized wins, and found the FRP, but not the P3b, to be a reliable index of reward magnitude, with greater amplitudes following large compared to small wins. The current study was also the first to examine near-wins using a PCA; these outcomes were found to elicit smaller FRN amplitudes compared to losses, which implies that they are perceived as less unfavourable than loss outcomes, and that they may play a role in the particular appeal of EGM gambling. Thus, the current study was able to achieve a comprehensive examination of the latent psychophysiological profile of healthy controls in response to gambling stimuli that are commonly encountered in real gambling environments. These findings may guide future research on problem gambling behaviours in order to
determine whether this disorder is caused by an abnormal response to reward and/or punishment/non-reward stimuli.
CHAPTER SEVEN – STUDY D

Reward and Punishment Hyposensitivity in Problem Gamblers: A Study of Event-related Potentials Using a Principal Components Analysis

7.1. Preamble


Chapter Seven outlines the final empirical study of this thesis. This study sought to determine whether problem gamblers exhibit maladaptive responses to reward and/or punishment as indexed by abnormalities in the latent ERP components that were found to be neural correlates of incentive value processing in Study C. Aspects of this study were presented at the Australasian Cognitive Neurosciences Conference (21st Meeting of the Australasian Society for Psychophysiology), Sydney, Australia, 9-12 December, 2011 (Appendix R).
7.2. Abstract

Objective: To investigate whether the latent neural correlates of incentive processing differ between problem gamblers (PGs) and non-PGs.

Methods: Event-related potential (ERP) data were derived while 16 PGs and 20 non-PGs played a computer electronic gaming machine (EGM) task. Psychophysiological responses to outcomes commonly encountered during EGM gambling, including Large wins, Small wins, Near-wins, and Losses, were examined using a spatiotemporal principal components analysis (PCA). Subjects also completed questionnaires that assessed their levels of impulsivity, attraction to appetitive stimuli, and avoidance of aversive stimuli.

Results: Losses elicited a feedback-related negativity (FRN), whereas wins elicited a feedback-related positivity (FRP) at the same latency and topography. PGs exhibited both attenuated FRN amplitudes following Losses and FRP amplitudes following Wins. Greater P3b amplitudes were found following Wins compared to Losses. Trends for reduced P3b amplitudes following all outcome types, and for similar P3b amplitudes following Large and Small wins, were found for the PG group. FRN amplitudes following Near-wins were significantly reduced compared to Losses for both PGs and HCs, whereas the P3b was not sensitive to differences between these two outcome types.

Conclusions: We provide evidence that PGs are hyposensitive to both positive and negative outcomes.

Significance: The finding that PGs are hyposensitive to reward and punishment provides valuable insight into the nature of deficit in this disorder, and provides a foundation for future research and clinical interventions.
7.3. **Introduction**

Previously considered to be an impulse control disorder (APA, 2000), problem gambling (also known as disordered, pathological, or compulsive gambling) shares many similarities with substance use disorder (Blaszczynski et al., 2008; Blum et al., 2000; Potenza, Kosten, & Rounsaville, 2001) and was recently reclassified as an addictive disorder in the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; APA, 2013). It is possible that problem gambling, like substance use disorder, is the result of an abnormally functioning reward system, and affected individuals are therefore, unable to properly evaluate environmental stimuli in terms of its incentive value (i.e., reward vs. non-reward). A focus of recent research has been to investigate the neural correlates associated with ongoing outcome monitoring and the role these play in guiding future behaviours. A particularly exciting avenue of research involves the examination of the feedback-related negativity (FRN) event-related potential (ERP) component. Similar to the error-related negativity (ERN) that is elicited by commission errors in reaction time tasks (Falkenstein et al., 1990; Gehring & Willoughby, 2002a; Miltner et al., 1997), the FRN provides insight into how feedback on reward and non-reward outcomes are evaluated in the brain. This component has been consistently shown to be sensitive to valence and context manipulations. Specifically, larger FRN magnitudes are observed when feedback signals monetary loss compared to gain (San Martin et al., 2010; Toyomaki & Murohashi, 2005; Yeung et al., 2005) or the least desired outcome within a particular context (Holroyd et al., 2004) during tasks that resemble gambling activity. The *reinforcement learning theory* (Holroyd & Coles, 2002) postulates that the ERN and FRN reflect the activity of a high-level error-processing system within the mesolimbic-dopaminergic pathway, a system believed to be involved in the evaluation of environmental stimuli, the activation of motivated
behaviours, and association formation. Within this system, the basal ganglia are hypothesised to monitor outcomes based on previously learned expectations, and in turn, stimulate dopamine (DA) levels; accordingly, rewarding outcomes cause DA to be released, leading to feelings of pleasure, whereas worse-than-expected outcomes are associated with reduced DA transmission (Fiorillo et al., 2003; Schultz, 2007). The observed FRN/ERN response reflects the resultant disinhibition in the anterior cingulate cortex (ACC), which is likely caused by decreased DA transmission (de Bruijn, Hulstijn, Verkes, Ruigt, & Sabbe, 2004; de Bruijn, Sabbe, Hulstijn, Ruigt, & Verkes, 2006; Zinnheld, Carroll, Kieffaber, O’Donnell, Shekhar, & Hetrick, 2004; cf. Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; van Veen, Holroyd, Cohen, Stenger, & Carter, 2004). Individuals with substance use disorder have demonstrated smaller ERN responses compared to healthy individuals (Franken, van Strien, Franzek, & van Dewatering, 2007), and fMRI research has shown abnormally low neural activation in cortical regions that generate the FRN following the experience of both reward and punishment in problem gamblers (de Ruiter et al., 2009; Tanabe, Thompson, Claus, Dalwani, Hutchison, & Banich, 2007, cf. Miedl et al., 2010; van Holst et al., 2012).

While the negative deflection to unfavourable outcomes described above has been reported in a substantial body of literature using various paradigms, including simulated gambling (Hewig et al., 2007) and guessing tasks (Hajcak et al., 2006; Hajcak et al., 2005, 2007), recent research has suggested that this ERP component is better conceptualised as a positive-going ERP response that is greater following reward (compared to non-reward) (Foti et al., 2011; Holroyd et al., 2003; Holroyd et al., 2008). This conjecture is based on the assumption that the FRN is actually an N200 ERP component that is elicited in response to negative feedback, but absent following
positive feedback. Thus, the reduced FRN amplitudes following wins previously observed may not be an attenuated response to these outcomes. Rather, it may be driven by a feedback-related positivity (FRP) that is characterised by greater-amplitude positive deflections following reward in the same latency window as the FRN (i.e., 250-350 ms post-feedback) (Holroyd et al., 2008), or possibly by the large amplitude P300 response that occurs immediately after it (Yeung & Sanfey, 2004). Because the majority of previous research on the FRN has employed a difference waveform to examine valence effects (e.g., Dunning & Hajcak, 2007; Foti & Hajcak, 2009; Hajcak et al., 2007; Holroyd et al., 2008; Miltner et al., 1997), from which it is not possible to identify the source of variance within the data (i.e., whether it is caused by a response to positive or negative stimuli), the nature of this feedback-related component remains unclear. In a recent study that employed a spatiotemporal principal components analysis (PCA) of ERP data to examine the responses of healthy controls to reward and punishment stimuli (Lole et al., 2013), loss outcomes were followed by a frontally maximal negative deflection 290 ms post-feedback (consistent with traditional conceptualisations of the FRN), whereas wins elicited a positive deflection at the same latency and topography.

In order to help elucidate the nature of deficit in problem gambling, the current study investigated whether the latent feedback-related response pattern (i.e., of the FRN and/or the FRP) to outcomes of varying incentive value, as determined using a spatiotemporal PCA, can differentiate problem gamblers from non-problem gamblers. At present, there are a number of hypotheses to explain the characteristic behaviours of problem gambling. It is possible that problem gamblers are hypersensitive to reward, and therefore, the prospect of immediate gain outweighs negative consequences (e.g., Hewig et al., 2010; Miedl et al., 2010; van Holst et al., 2012). Alternatively, PGs may be hyposensitive to punishment and repeated losses have minimal impact, and/or they
may be hyposensitive to wins (e.g., Damasio, 1994, 1996; Reuter et al., 2005), in what has been described as a *reward deficiency syndrome* (Blum et al., 2000), which manifests in thrill-seeking behaviour in order to achieve a normal state of functioning. Theoretical accounts (e.g., Damasio, 1994; Blum et al., 2000) and empirical research using autonomic (Goudriaan et al., 2006) and neuroimaging (fMRI) (de Ruiter et al., 2009; Reuter et al., 2005) methods suggest that problem gamblers are *hyposensitive* to the experience of both reward and punishment (although they may be more sensitive to prospective rewards; see Miedl et al., 2010; van Holst et al., 2012). Despite the potential of using the FRN and/or the FRP to investigate incentive learning processes, only one study to date has explored feedback-related ERP component/s in problem gamblers. Hewig et al. (2010) examined the responses of problem and non-problem gamblers to a realistic Black-Jack task, and concluded that problem gamblers were hypersensitive to reward based on greater amplitude FRP deflections 300 ms following win compared to loss outcomes. However, this finding has been criticised for only comparing responses to ‘bust’ (clear loss) outcomes and ‘no-bust’ (but not necessarily win) outcomes (Oberg et al., 2011). Oberg et al. (2011) reported a hypersensitive response to reward in frequent gamblers based on an ‘early-FRN’ (occurring 185 ms post-outcome at FCz); however, subjects in that study were assigned to groups based on frequency of gambling, but not actual gambling severity (non-problem gamblers can gamble frequently with no associated harm, whereas it is possible for problem gamblers to gamble in an infrequent but dysfunctional manner). To overcome these problems, the current study investigated the ERP responses of individuals classified as problem gamblers using the Problem Gambling Severity Index (PGSI) of the Canadian Problem Gambling Index (CPGI; Ferris & Wynne, 2001) to unambiguous reward and non-reward stimuli, and whether these differ from the responses of non-problem gamblers.
Following the findings of Lole et al., (2013), it was predicted that Loss outcomes would elicit a FRN, whereas Win outcomes would elicit a FRP at the same latency and topography. In order to test the assumptions of several theoretical accounts of problem gambling (e.g., Damasio, 1994; Blum et al., 2000), we examined whether these neural correlates of incentive value processing indicate reduced sensitivity to reward, as indicated by attenuated FRP amplitudes following wins, and punishment/non-reward, reflected in reduced FRN amplitudes following losses, in individuals with this disorder.

7.3.1. **The P300 as an indicator of incentive value processing.**

The current study also sought to evaluate the P300 as an indicator of positive outcome evaluation in problem gamblers. Typically examined as a global component, the P300 (called the late positive complex, LPC, in many studies) has been shown to reflect the processing of motivationally significant aspects of stimuli. Some research has reported greater P300 responses following positive compared to negative outcomes (Hajcak et al., 2007; Toyomaki & Murohashi, 2005; Wu & Zhou, 2009; Zhou et al., 2010), suggesting win events are perceived as more salient and assigned greater importance than losses. Other research has demonstrated greater P300 amplitudes for larger magnitude outcomes in healthy subjects regardless of valence (Sato et al., 2005; Yeung & Sanfey, 2004). A recent study revealed that the P3b subcomponent is likely to be driving the observed valence differences in global P300 amplitude (Lole et al., 2013). Reduced P300 amplitudes to rewarding stimuli have been found for frequent gamblers compared to non-gamblers (Oberg et al., 2011), and in individuals with substance use disorder (Goldstein et al., 2008); it is also of value to confirm whether problem gamblers process the significance of positive outcomes differently than normal. Based
on the findings of previous research, we predict that wins will elicit greater P3b amplitudes compared to losses, and that problem gamblers will exhibit reduced P3b amplitudes following win outcomes, compared to non-problem gamblers.

7.3.2. **The significance of reward magnitude for problem gamblers.**

It is also of value to determine how problem gamblers respond to variations of reward magnitude. Research that has previously investigated the link between the FRN and the value of different magnitude outcomes has yielded conflicting results; some studies suggest that larger losses and smaller gains produce greater FRN amplitudes (e.g., Bellebaum et al., 2010; Holroyd et al., 2004; Lole et al., 2013), whilst others have found no magnitude effects (e.g., Gu et al., 2010; Hajcak et al., 2006; Yeung & Sanfey, 2004). As mentioned above, a similar situation is evident in the extant P300 literature. Individuals with substance use disorder have demonstrated abnormalities in reward perception, assigning equal value to large and small amounts of money ($10 = $1000; Goldstein et al., 2007). To our knowledge, the current study is the first to investigate whether anomalous assessment of the value of different reward magnitudes characterises the responses of problem gamblers. We predicted that non-problem gamblers would exhibit greater amplitude P3b responses following larger compared to smaller wins, and that problem gamblers would not display different responses to large and small wins.

7.3.3. **The significance of near-win outcomes during gambling.**

Electronic gaming machines (EGMs; also called ‘poker’ or ‘slot’ machines) are of particular clinical significance to problem gambling. Compared with other gambling activities, a high proportion people presenting for treatment report EGMs as their
preferred gambling medium (see Dowling et al., 2005), and EGMs are associated with fast progression of addiction (Breen & Zimmerman, 2002) and greater severity of symptoms (Petry, 2003). Moreover, EGMs typically deliver a large number of outcomes in a short period of time, making them particularly suitable for ERP research. For these reasons, we chose to employ a simulated EGM task in the current study. This ecologically valid gambling task allowed the examination of problem gamblers’ responses to all key EGM outcomes, including large and small wins, losses, and outcomes similar to losses disguised as wins (LDWs, i.e., where the amount returned is less than the amount wagered, see Dixon et al., 2010), near-wins. These are outcomes in which the matching combination of symbols needed for a win are ‘almost’ achieved, but, unlike LDWs, are not associated with the return of credits, or superfluous auditory tunes or visual cues. Whereas previous autonomic research has shown that LDWs elicit changes in electrodermal activity similar to wins (Dixon et al., 2010), near-wins have also been shown to increase motivation to gamble by recruiting reward-related brain circuitry, despite loss of money/credits and being subjectively rated as more unpleasant than losses (Clark et al., 2009), particularly for regular EGM players at a higher risk for developing disordered gambling behaviours (Chase & Clarke, 2010).

Electrophysiological research has revealed near-win outcomes to be less punishing (Lole et al., 2013; Luo et al., 2011) and more rewarding (Qi et al., 2011) than full-loss outcomes. The current study sought to investigate whether FRN amplitudes following near-wins are different to those following losses, and whether this neural correlates of incentive processing following these outcomes is able to differentiate the responses of problem and non-problem gamblers.
7.3.4. **The relationship between psychophysiological reactivity to incentive and individual differences in personality.**

There has been a considerable amount of research conducted on the relationship between personality and gambling behaviours. Impulsivity has been found to be strongly associated with problem gambling (see van Holst et al., 2010 for a review); however, the few studies that have investigated self-reported sensitivity to reward and punishment in problem gamblers have reported mixed results (Goudriaan et al., 2006; Loxton et al., 2008). Research on the relationship between individual differences in reward and punishment sensitivity and psychophysiological indicators of incentive processing has shown healthy individuals who score higher on self-reported punishment sensitivity measures display higher FRN amplitudes following unfavourable feedback (Balconi & Crivelli, 2010; De Pascalis et al., 2010; Santesso, Dzyundzyak, & Segalowitz, 2011; Sato et al., 2005; Unger, Heintz, & Kray, 2012), and individuals reporting higher reward sensitivity demonstrate greater P300 amplitudes following positive feedback (Van den Berg, Franken, & Muris, 2011); however, this relationship has not been examined in problem gamblers. Due to the potential of using self-report measures as screening instruments in the identification of individuals at risk of problem gambling, it is worthwhile examining whether these subjective measures correlate with objective neural responses. For example, if attenuated P3b amplitudes to wins are found to be a reliable indicator of reward hyposensitivity in problem gamblers, and lower BAS scores are correlated with P3b amplitudes, then this psychological inventory could potentially be used as a screen for underlying deficits in reward processing. The current study investigated whether the latent neural correlates of incentive processing, determined using a PCA, are related to self-reported reward and punishment sensitivity, and specifically, whether FRN amplitudes following losses correlated with BIS scores,
and whether P3b amplitudes following wins and near-wins correlate with BAS scores. It also sought to investigate whether tendencies toward impulsive behaviour are related to the FRR and P3b responses following wins, losses, and near-wins.

7.3.5. The current study.

The current study sought to investigate the neural correlates of incentive value processing, the FRN and P3b, using a PCA, and whether these differ between problem and non-problem gamblers during play on an EGM task. Responses to outcomes of varying valence and magnitude, including large and small wins, near-wins, and losses, were examined. The relationship between latent psychophysiological responses to outcomes commonly encountered during EGM play and individual differences in personality variables, including impulsivity and sensitivity to reward and punishment was also examined.

7.4. Method

7.4.1. Participants.

Sixteen problem gamblers (PGs) were recruited through public advertisement (11 male, $M_{age} = 34.80$ years; $SD = 16.79$; range 18-67 years), all of whom reported EGMs as their preferred gambling medium. After PGs were recruited, twenty healthy controls (HCs) (9 male, $M_{age} = 28.75$ years; $SD = 11.19$; range 18-49 years) were recruited from the community and from the University of Wollongong. Age did not significantly differ between groups ($p = .242$). The PGSI (Ferris & Wynne, 2001) was used to classify participants as problem (score ≥ 8) or non-problem gamblers. No participants reported a history of seizures or severe head injury, or using nicotine, alcohol, or prescription/illicit drugs in the twelve hours prior to testing. Written informed consent was obtained from
participants, all of whom were advised that participation was entirely voluntary, and that they could withdraw from the study at any time (Appendices S and T). The research protocol was approved by the University of Wollongong Human Research Ethics Committee.

7.4.2. Materials.

7.4.2.1. Physiological recording equipment.
EEG was recorded from a 19-site electrode cap (comprised of tin electrodes) using NuAmps 2.0 software (NeuroScan Compumedics, U.S.A.). Electrodes were fitted according to the international 10-20 system, were referenced to linked earlobes, and grounded by a cap electrode located mid-way between Fpz and Fz. Vertical eye movement (vEOG) was monitored with two tin cup electrodes: one placed 2 cm above, and the other 2 cm below the left eye. Horizontal eye movement (hEOG) was monitored with two tin cup electrodes placed next to the outer canthus of each eye. Impedance was less than 5 kΩ for cap electrodes and less than 3 kΩ for EOG and reference electrodes. Scalp potentials were amplified × 20 000, and EOG potentials were amplified × 5000; both were sampled at a rate of 500 Hz.

7.4.2.2. Psychological inventories.
The PGSI (Ferris & Wynne, 2001) assesses the severity of gambling behaviours (it scores individuals as ‘non-problem’, ‘low-risk’, ‘moderate risk,’ or ‘problem’ gamblers). The group cut-off scores have been shown to accurately classify gamblers’ levels of risk according to the Diagnostic and Statistical Manual of Mental Disorders criteria (4th ed.; DSM-IV; APA, 1994) and comparisons with clinical assessment interviews (Ferris & Wynne, 2001). As it provides a means of identifying PGs in a
confidential and anonymous manner (thus, avoiding the problem of symptom under-reporting commonly associated with this disorder), this quantitative self-report measure was chosen for use in the current study.

The Impulsiveness Questionnaire (Eysenck et al., 1985) was used to assess trait impulsivity, and the Behavioural Inhibition System (BIS) and Behavioural Activation System (BAS) scales (Carver & White, 1994) were used to assess subjective sensitivity to punishment and reward, respectively. The BIS scale assesses an individual’s feelings toward the anticipation of punishment, and the multidimensional BAS scale gauges various aspects of reward desirability, including the constant pursuit of goals (Drive subscale), the desire to experience the excitement associated with new rewards and the readiness to spontaneously approach potential rewards (Fun-seeking subscale), and how favourably the individual anticipates or responds to rewards (Reward-responsiveness subscale).

The Depression Anxiety and Stress Scale (DASS-21; Lovibond & Lovibond, 1995) was also administered to participants in order to determine whether other comorbid conditions, particularly depression, influence patterns of physiological responding to gambling stimuli.

7.4.2.3. **Computer gambling task.**

A gambling task was administered using Presentation software (version 13.0, Neurobehavioral Systems, www.neurobs.com). The task was designed to mimic a game commonly run on EGMs. On the screen at all times was a display of a single row of four fruit symbols. Eight different fruit symbols were used, and each could appear at random in any of the four columns. Each trial was unique in terms of its stimulus configuration (symbol and sequence), except for Large win outcomes, where each of the
eight combinations possible could occur numerous times. The amount wagered on each trial, the number of credits left to play with, and the amount won (if applicable) were displayed prominently after each trial. One of four outcomes was possible on each trial: a Large win occurred when all four symbols were identical, yielding a return ten times the amount bet; Small wins were indicated by three of the same symbols occurring in sequence, returning five times the amount bet; Near-wins occurred when one different symbol was inserted between two or three identical symbols, with no credits returned; and Losses occurred when neither a win nor near-win occurred, with no credits returned. The probability of each outcome closely matched outcomes from real EGM play (Wilkes et al., 2010), with a few minor changes. An equal number (15%) of Near-win and Win (Large and Small combined) outcomes were presented, with the remaining trials being Losses (70%). Participants did not wager their own money, but started the session with a free allocation of 5000 credits (valued at AUD $50).

Each trial commenced with participants choosing to either ‘Bet Low’ or ‘Bet High’ by pressing either a ‘Bet 1’ or ‘Bet 10’ button, respectively. An inter-stimulus interval of 2 s (±400 ms), marked by all the reels on the display appearing to spin, was inserted between bet placement and outcome presentation (all reels were revealed at the same time, not in a sequential left-to-right stop that is common on most commercially available EGMs). Following the outcome presentation, a delay of 2 s occurred before the next bet could be placed (signalled by the ‘Bet’ buttons turning from red to green). The attendant attention-getting auditory and visual stimuli that typically accompany EGM play were not presented in order to avoid contamination of the ERPs to gambling outcomes.
7.4.3. **Procedure.**

After providing informed consent, participants completed the PGSI, Impulsivity Questionnaire, and BIS/BAS scale inventories. They were then fitted with the physiological recording equipment and instructed to complete a five-minute eye calibration task (Croft & Barry, 2000). Participants were instructed on how to play the gambling task and engaged in ten practice trials. Following this, they played 450 trials of the task. They were informed that they were able to win one entertainment voucher (valued at AUD $12) if they accumulated more than 6000 credits by the end of the session, and two vouchers if they had accumulated 7000 credits or more.

7.4.4. **Data reduction and analysis.**

An algorithm for the offline correction of eye artefacts in task-related brain activity was used to remove EOG artefacts from the data (Croft & Barry, 2000). Because of an insufficient number of ‘Bet Low’ epochs, outcome data were collapsed across the two bet conditions. The EEG data were subjected to a low-pass filter (30 Hz; 24 dB/oct) and baselined relative to the pre-stimulus interval (100 ms). ERPs were created by extracting epochs (100 ms to 800ms post-stimulus) around each individual outcome over nineteen sites (Fp1, Fp2, F7, F3, Fz, F4, F8, C7, C3, Cz, C4, C8, P7, P3, Pz, P4, P8, O1, O2). Because there were not enough epochs for Small and Large wins over the two Bet options to allow an examination of Outcome × Bet size interaction effects, and because preliminary analyses showed the effect of Bet size was small (see Figure 12A for effects at Fz and Figure 12B for effects at Pz), we averaged the data from the Bet Low and Bet High epochs together to derive four Outcome types (Large wins, Small wins, Losses, Near-wins). Each epoch was manually examined and those that contained muscle or movement artefacts were excluded from further analysis. In order to prevent
possible habituation effects on the FRR component/s, only 15% of Loss outcomes, matched in time to the occurrence of each win outcome, were included in the analyses.

The remaining artefact-free epochs for each participant were averaged together according to Outcome type. The data were subjected to a two-step spatiotemporal PCA using the ERP PCA (EP) Toolkit, version 2.23 (Dien, 2010b) in order to examine the latent nature of the FRN and P300 responses without the influence of overlapping ERP components. A spatial PCA was first conducted on the data in order to identify the variance accounted for by electrode sites over all time points, participants, and conditions. Dien (2010a; 2012) recommends that the number of factors retained for the first data reduction in a two-step PCA be determined using the parallel test (Horn, 1965). This test considers the amount of variability in the observed dataset using the Scree test (Cattell, 1966) and compares it to uncorrelated variables within a random noise dataset. A temporal PCA, which uses all time points, over all participants and conditions as variables, was then performed on the spatial components. Because the EP toolkit currently determines the best number of temporal factors based on a parallel test conducted across all spatial factors, and not what is necessarily best suited to the individual factor, the number of temporal factors was independently selected for each spatial factor based on visual examination of the data. The covariance matrix and Kaiser normalisation was used for all PCAs. Dien (2010a) recommends that INFOMAX rotation be performed for spatial PCAs, and PROMAX rotation for temporal PCAs. When the oblique INFOMAX rotation was applied to the spatial step in the current dataset, the two factors that accounted for the majority of variance according to the parallel test were centred on Pz and O2 (this was likely due to the large, parietally maximal P3b being reflected in surrounding occipital topographies, since electrode sites are allowed to correlate in non-orthogonal rotations); the frontally maximal FRN/FRP
components were not identified. When an orthogonal rotation, VARIMAX, was applied, two factors centred on Pz and Fz accounted for the majority of variance in the dataset. Because of this, and because the topographies of the ERP components of interest are well established, VARIMAX rotation was applied to the spatial step of the PCA (Spencer et al., 2001). PROMAX rotation was applied to the temporal step of the PCA.

Once the factors corresponding to the feedback-related response/s and P3b were identified, two separate 2 Group (PG, HC) × 4 Outcome (Large win, Small win, Near-win, Loss) mixed model repeated measures analysis of variance (ANOVA) were performed, with Group as a between-subjects factor and Outcome as a within-subjects factor. Three orthogonal planned contrasts were conducted on the Outcome factor to test: i) valence differences (Small and Large Wins combined vs. Losses and Near-wins combined), ii) magnitude differences (Small vs. Large Wins), and iii) the salience of Near-wins (Near-wins vs. Losses).

Pearson correlations were performed in order to examine whether mean factor scores for the latent neural correlates of incentive processing during the gambling task, determined using a PCA, correlated with self-reported scores on the Impulsiveness Questionnaire, and the BIS and BAS subscales for each Group. Specifically, we wanted to examine whether scores on the BIS correlated with mean FRN factor scores following Losses, and whether scores on the BAS correlated with P3b factor scores following Wins and Near-wins. We also examined whether scores on the Impulsiveness questionnaire correlated with feedback-related responses (i.e., the FRN and FRP) and P3b factor scores following wins, losses, and near-wins. Bonferroni corrections for multiple comparisons were employed for this personality variable; accordingly, Pearson correlations were evaluated at an alpha level of .0125.
7.5. **Results**

7.5.1. **Physiological data.**

Two spatial factors, centred on Pz and Fz, were retained in the final PCA, as mentioned above. The grand average virtual ERPs created by the EP toolkit were found to closely resemble the grand average raw ERP waveforms created in NeuroScan for this dataset at site Fz (SF1) (Figure 13A) and site Pz (Figure 14A). Figures 13B and 14B show how the grand average virtual ERP for sites Fz and Pz, respectively, are comprised of the individual factors that were identifiable ERP components, and/or accounted for at least 1% of the variance in the data.
Figure 12. The raw ERP waveforms based on Outcome for the problem gambler (PG; n = 16) and healthy control (HC; n = 20) Groups at sites Fz (panel A) and Pz (panel B). Note the different y-axis scales for each panel.
Figure 13. A) The raw ERP grand average waveform and the virtual ERP grand average waveform (over all Outcomes and Groups; \( n = 36 \)) at Site Fz. B) The virtual ERP components averaged over all Outcomes and Groups for the individual frontal factors that were identifiable ERP components, and/or accounted for more than 1% of the variance in the dataset.
Figure 14. A) The raw ERP grand average waveform and the virtual ERP grand average waveform (over all Outcomes and Groups; \( n = 36 \)) at Site Pz. B) The virtual ERP components averaged over all Outcomes and Groups for the individual parietal factors that were identifiable ERP components, and/or accounted for more than 1% of the variance in the dataset.
Four temporal factors were found to best represent the data for the parietal (Pz) spatial factor (SF1) and seven temporal factors best represented the frontal (Fz) spatial factor (SF2). Parietal factor SF1/TF1, and frontal factor SF2/TF2, corresponded to the P3b and the feedback-related responses, respectively, in terms of their topography and latency; the P3b was defined as being a parietally maximal positive deflection peaking 250-600 ms post-stimulus, and the FRR as being a frontally maximal negative deflection to unfavourable outcomes and/or a positive deflection following beneficial outcomes 250-350 ms post-stimulus presentation. The N100 was also examined in the current study, in order to determine whether there were any inherent differences in the way the different outcomes were processed between the two groups. This component was identified at Fz (factor SF2/TF5) and was maximal 142 ms post-outcome (Figure 13B). The results of the PCA revealed no main effect of Outcome ($p = .589$) or Group ($p = .509$), and no Outcome $\times$ Group interaction ($p = .309$). The three remaining parietal factors, and three of the five remaining frontal factors, were identifiable ERP components; however, these are of less theoretical importance and/or are outside the scope of the current paper, so will not be discussed further.

7.5.1.1. Feedback-related responses.

The factor identified as corresponding to the FRR ERP components (SF2/TF2) was comprised of both a negative deflection following Losses (the FRN), and a positive deflection following Wins (the FRP), peaking at 314 ms post-outcome (Figure 16).
Figure 15. A) Virtual ERP waveforms for spatiotemporal factor SF2/TF2 for problem gambler (PG; n = 16) and healthy control (HC; n = 20) groups in response to each Outcome type at site Fz. This factor was identified as corresponding to both the feedback-related negativity and the feedback-related positivity ERP components. B) Scalp topographies demonstrating the different response patterns of PGs and HCs following each outcome type, which was maximal at 314 ms post-feedback.
A main effect of Outcome was found, $F(3,102) = 44.71, p < .001, \eta^2_p = .57$. The main effect of Group for this factor was not significant ($p = .243$), and the Outcome × Group interaction was only marginally significant ($p = .095$).

Planned contrasts revealed a significant difference in factor scores between the FRN following Losses and the FRP following Wins as indicated by a significant main effect of Outcome for this comparison, $F(1, 34) = 93.27, p < .001, \eta^2_p = .73$. This effect was more pronounced in the HC group, as indicated by a significant Group × Outcome interaction, $F(1, 34) = 4.89, p = .034, \eta^2_p = .13$. Significantly larger FRP amplitudes were observed following Large wins compared to Small wins, $F(1, 33) = 16.17, p < .001, \eta^2_p = .32$, although the Group × Outcome interaction for this comparison was not significant ($p = .905$). FRN amplitudes following Losses were found to be significantly greater than Near-wins, $F(1,34) = 13.07, p = .001, \eta^2_p = .28$, but the Outcome × Group interaction for this comparison was not significant ($p = .463$).

7.5.1.2. **P3b.**

The factor identified as corresponding with the P3b ERP component (SF1/TF1) was characterised by a parietally maximal positive deflection that peaked 520 ms post-outcome. For this factor, a main effect of Outcome was found, $F(3,102) = 12.90, p < .001, \eta^2_p = .27$. The main effect of Group for this factor was approaching significance ($p = .094$), with a tendency for greater P3b factor scores in the HC group for all outcomes ($M = 8.77, SE = 1.18$) compared to the PG group ($M = 5.72, SE = 1.32$). The Outcome × Group interaction was also significant, $F(3,102) = 2.72, p = .048, \eta^2_p = .07$ (Figure 16).
Figure 16. A) Virtual ERP waveforms for spatiotemporal factor SF1/TF1 for the problem gambler (PG; *n* = 16) and healthy control (HC; *n* = 20) groups in response to each Outcome type at site Pz. This factor was identified as corresponding to the P3b ERP component. B) Scalp topographies demonstrating the difference between each outcome type for PGs and HCs, at 520 ms post-feedback.

Planned contrasts revealed greater P3b amplitudes following Wins compared to Losses, *F*(1, 34) = 14.76, *p* = .001, ηₚ² = .30, although the Group × Outcome interaction for this comparison was not significant (*p* = .116). While Large wins elicited greater
P3b factor scores than Small wins, $F(1, 34) = 4.51, p = .041, \eta^2_p = .18$, the between-group effect for this comparison, as indicated by the Group × Outcome interaction, only approached significance ($p = .074$). The planned contrast between Losses and Near-wins revealed no significant difference in P3b factor scores following these Outcome types ($p = .529$), and the Group × Outcome interaction for this comparison was not significant ($p = .682$).

7.5.2. **Personality measures.**

Apart from a marginally significant negative correlation between PGSI scores and BIS scores for participants in the PG group ($p = .081$), no other personality measure was found to correlate with ratings of gambling severity for either Group. No between-group differences were found for Impulsiveness, or BIS and BAS subscale scores (Table 2). An independent groups t-test revealed no difference in depression scores between the problem gambler and healthy control group ($p = .248$).

**Table 2.** Mean (and standard error) scores of the problem gambler (PG; n = 16) and healthy control (HC; n = 20) groups for each of the personality variables assessed. The column on the far right hand side presents results from the between-group comparison for each of these variables.

<table>
<thead>
<tr>
<th>Personality Variable</th>
<th>PG</th>
<th>HC</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Problem Gambling Severity Index</td>
<td>13.44 (1.38)</td>
<td>1.25 (.38)</td>
<td>.000</td>
</tr>
<tr>
<td>Impulsivity Questionnaire</td>
<td>11.07 (1.23)</td>
<td>9.55 (1.11)</td>
<td>.370</td>
</tr>
<tr>
<td>BAS Drive</td>
<td>7.93 (.71)</td>
<td>9.55 (.60)</td>
<td>.091</td>
</tr>
<tr>
<td>BAS Fun-seeking</td>
<td>7.93 (.65)</td>
<td>9.05 (.44)</td>
<td>.152</td>
</tr>
<tr>
<td>BAS Reward Responsiveness</td>
<td>6.93 (.44)</td>
<td>7.35 (.30)</td>
<td>.426</td>
</tr>
<tr>
<td>BIS</td>
<td>14.20 (.79)</td>
<td>14.00 (.65)</td>
<td>.845</td>
</tr>
</tbody>
</table>
Factor scores for the FRN and FRP responses at Fz, and factor scores for P3b responses at Pz, did not correlate with Impulsiveness scores for any Outcome type for either Group (Table 3).

Table 3. Pearson correlations for scores the Impulsiveness Questionnaire and factor scores on the FRR and P3b ERP components following each outcome type for the problem gambler (PG; n = 16) and healthy control (HC; n = 20) groups. The p-values for each test are presented in parentheses. None of the correlations reached statistical significance after multiple comparisons were controlled for using the Bonferroni procedure.

<table>
<thead>
<tr>
<th>ERP Component</th>
<th>PG (p-value)</th>
<th>HC (p-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FRR</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large win</td>
<td>.054 (.850)</td>
<td>-.154 (.516)</td>
</tr>
<tr>
<td>Small win</td>
<td>-.269 (.332)</td>
<td>-.185 (.435)</td>
</tr>
<tr>
<td>Near-win</td>
<td>-.100 (.722)</td>
<td>.172 (.467)</td>
</tr>
<tr>
<td>Loss</td>
<td>-.037 (.897)</td>
<td>.153 (.521)</td>
</tr>
<tr>
<td><strong>P3b</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large win</td>
<td>-.155 (.582)</td>
<td>-.049 (.837)</td>
</tr>
<tr>
<td>Small win</td>
<td>-.065 (.817)</td>
<td>.037 (.877)</td>
</tr>
<tr>
<td>Near-win</td>
<td>-.233 (.404)</td>
<td>.027 (.912)</td>
</tr>
<tr>
<td>Loss</td>
<td>-.207 (.458)</td>
<td>.236 (.316)</td>
</tr>
</tbody>
</table>

No significant relationships between P3b amplitude and self-reported sensitivity to reward were found for the HC group. However, several significant correlations between these measures were found for the PG group. Specifically, PGs who reported higher scores on the BAS Fun-seeking scale were found to elicit greater P3b amplitudes in response to both Small and Large wins. P3b amplitudes following Near-wins were found to be higher in PG individuals who reported higher scores on the Fun-seeking and Drive BAS subscales (Table 4). Subjective self-reported sensitivity to punishment, as
indicated by scores on the BIS, did not correlate with FRN scores following Losses for the PG \( (r = -0.26, p = .344) \) or HC group \( (r = -0.05, p = .831) \).

Table 4. Pearson correlation scores on the subscales of the BAS and factor scores on the P3b ERP components following each outcome type for the problem gambler (PG; \( n = 16 \)) and healthy control (HC; \( n = 20 \)) groups. The values in parentheses indicate the \( p \)-value for each test; comparisons that reached statistical significance are marked with an asterisk (*).

<table>
<thead>
<tr>
<th></th>
<th>PG (( p )-value)</th>
<th>HC (( p )-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drive</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large win</td>
<td>.308 (.265)</td>
<td>-.071 (.767)</td>
</tr>
<tr>
<td>Small win</td>
<td>.335 (.221)</td>
<td>.053 (.825)</td>
</tr>
<tr>
<td>Near-win</td>
<td>.532 (.041)*</td>
<td>-.169 (.476)</td>
</tr>
<tr>
<td><strong>Fun-seeking</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large Win</td>
<td>.524 (.045)*</td>
<td>-.035 (.882)</td>
</tr>
<tr>
<td>Small Win</td>
<td>.532 (.041)*</td>
<td>.018 (.939)</td>
</tr>
<tr>
<td>Near-win</td>
<td>-.634 (.011)*</td>
<td>-.074 (.757)</td>
</tr>
<tr>
<td><strong>Reward Responsiveness</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Large Win</td>
<td>.257 (.300)</td>
<td>-.320 (.169)</td>
</tr>
<tr>
<td>Small Win</td>
<td>.271 (.328)</td>
<td>-.311 (.182)</td>
</tr>
<tr>
<td>Near-win</td>
<td>.445 (.097)</td>
<td>-.295 (.207)</td>
</tr>
</tbody>
</table>

7.6. Discussion

The current study utilised a spatiotemporal PCA to examine whether the latent ERP components previously found to index incentive value processing, the FRRs and the P3b, could differentiate problem gamblers’ reactions to outcomes of varying valence (i.e., good vs. bad) and reward magnitude (large vs. small) in an ecologically-valid EGM task paradigm. It also sought to determine whether these responses correlate with
self-reported individual differences in impulsivity, and reward and punishment sensitivity.

7.6.1. The physiological effects of wins vs. losses

7.6.1.1. Feedback-related responses.

The application of a PCA in the current study allowed the examination of the latent ERP responses to win and loss feedback without the influence of overlapping ERP components. Consistent with previous FRN findings using traditional ERP quantification methods (Gehring & Willoughby, 2002a; Hajcak et al., 2005; Hajcak et al., 2006; Holroyd et al., 2006; Yeung et al., 2005; Yeung & Sanfey, 2004), Loss outcomes were found to elicit a negative deflection approximately 300 ms post-feedback. Wins were followed by a positive deflection at the same latency, replicating the findings of Lole et al. (2013). PGs were found to exhibit both attenuated FRN responses following Losses and reduced FRP amplitudes following Wins, suggesting a hyposensitivity to punishing and rewarding stimuli, respectively. These results are consistent with empirical neuroimaging evidence, which has demonstrated reduced neural activation in problem gamblers when they are processing both reward and punishment stimuli (de Ruiter et al., 2009; Reuter et al., 2005; Tanabe et al., 2007; cf. Miedl et al., 2010; van Holst et al., 2012).

The dissociation of the feedback-related responses into the FRN and FRP components further consolidates the disparate findings of previous studies that have employed traditional ERP quantification methods, and elucidates how problem gamblers process feedback of differing incentive value. It is unlikely that the FRN identified in the current study is actually an N200 component (Holroyd et al., 2008); if this were the case, we would expect to find greater negative deflections following Near-
wins compared to more frequent Loss outcomes, not the reduced responses actually observed. It is also unlikely that the FRP component is a P3a/Novelty P3 since the EP Toolkit parses separate ERP components based on common variance in the raw ERP data at particular points in time, and because this P300 subcomponent has not been previously linked to the FRN. Thus, we conclude that the FRP and FRN are distinct neural correlates of reward and punishment processing, which are likely to be generated within the same cortical system/s. We propose that the reduced FRN and FRP responses of PGs are potentially a manifestation of deficit within this system, a notion consistent with theoretical accounts of problem gambling (Damasio, 1994). Interpreted in light of the reinforcement learning theory (Holroyd & Coles, 2002), the FRN and FRP may reflect disinhibition and inhibition of the ACC, respectively, a cortical structure which has previously been shown to guide current and future reactions to aversive stimuli (e.g., Campbell-Meiklejohn et al., 2008). The attenuated FRN and FRP amplitudes demonstrated by problem gamblers in the current study may indicate abnormal neurotransmitter diffusion, structural abnormalities, or some other form of suboptimal functioning within the mesolimbic-dopaminergic system, potentially accounting for the problematic gambling behaviours associated with this disorder.

The reduced FRP amplitudes found for PGs in the current study contrasts with previous electrophysiological research, which advocates heightened reward responsiveness as a defining response pattern of problem gamblers (Hewig et al., 2010; Oberg et al., 2011). It is possible that differences in the tasks used to assess neural sensitivity to outcome valence contributed to the discrepant FRP results between the current study and the study by Hewig and colleagues. As previously mentioned (Section 2.3 and 7.3.), Hewig et al. (2010) examined the neural responses following decisions that led to ‘bust’ outcomes compared to ‘no-bust’ outcomes that occurred on a computer
Black-Jack task. It is possible that the greater FRP amplitudes of problem gamblers observed to ‘no-bust’ outcomes do not actually reflect a hypersensitive response to reward, but rather, an increased sensitivity to the excitement associated with the anticipation of future reward (since there is still an opportunity to win on such trials). This notion is consistent with previous fMRI research, in which problem gamblers were found to demonstrate greater activation in brain regions associated with reward processing when anticipating rewards (Miedl et al., 2010; van Holst et al., 2012). Further research using both neuroimaging and psychophysiological methods is required to determine the specific aspects of incentive processing the FRP component reflects, and the extent to which problem gamblers are motivated by the anticipation of future reward and the actual experience of reward while gambling. The current study did not find evidence of an ‘early-FRN’ waveform as described by Oberg et al. (2011); the EGM paradigm used did produce a factor that closely resembled the N100 component (i.e., SF2/TF5) at a comparable latency, but this component did not differ according to problem gambling status or the incentive value of the outcome.

7.6.1.2. **P3b.**

Consistent with previous findings on global P300 research (e.g., Hajcak et al., 2007, Wu & Zhou, 2009; Yeung et al., 2005; Zhou et al., 2010; cf. Sato et al., 2005; Yeung & Sanfey, 2004), and research on valence-sensitive P300 subcomponents (Lole et al., 2013), greater P3b amplitudes were found following Win compared to Loss outcomes. These results suggest that the P3b subcomponent was likely to be driving the valence differences observed for global P300 amplitude in previous research (e.g., Bellbaum & Daum, 2008; Bellbaum et al., 2010; Hajcak, Moser, Holroyd, & Simons, 2007; San Martin et al., 2010; Toyomaki & Murohashi, 2005; Wu & Zhou, 2009; Yeung et al.,
The trend for attenuated P3b responses following all outcomes among PGs (Figure 16), may suggest hypoarousal to gambling outcomes in general, or may reflect a reduced responsiveness/long-term habituation due to repeated exposure to gambling activity. Despite PGs displaying a trend toward attenuated P3b response to Wins compared to HCs (Figure 16), the between-group effect for the Win vs. Loss planned comparison was non-significant, a finding that is inconsistent with prior research which found attenuated P300 amplitudes following win outcomes for frequent gamblers vs. non-gamblers (Oberg et al., 2011), and with research on individuals with substance use disorders, which found attenuated P300 amplitudes following rewarding stimuli for individuals with cocaine use disorder compared to healthy controls (Goldstein et al., 2008; cf. Ramsey & Finn, 1997). The reason for this discrepancy may be due to a power issue in the current study, and further research to test whether problem gamblers are hyposensitive to reward is warranted.

Although Win (15%) outcomes were presented more frequently than Losses (70%) in the current EGM task, probability effects are unlikely to explain the valence effects observed in P3b amplitude between these outcomes; specifically, although each trial was broadly classified by outcome type (win, loss, or near-win), the exact presentation of stimuli (combination and sequence of fruit symbols) within these outcomes varied from trial-to-trial. Furthermore, larger P3b amplitudes were observed following Win events compared to equiprobable Near-win events, suggesting that the importance of these outcomes, not the frequency of occurrence, is reflected by increased P3b amplitudes (see Figure 16). While it is possible that probability effects may have cancelled out an actual difference between Near-wins and Losses, this is unlikely, as lower probability and greater salience of Near-wins would be expected to increase, rather than decrease, P3b amplitudes. Thus, we conclude that the larger P3b amplitudes
observed for Wins (compared to Losses) for both PGs and HCs reflect the salience of rewards to the individual (Johnson, 1986). Moreover, the primary aim of the current study was to investigate whether problem gamblers respond abnormally to win and loss outcomes, using an EGM task that is ecologically valid for the population of interest. The between-group comparisons would not have been influenced by stimulus probability, as each group experienced approximately the same proportion of Wins and Losses.

7.6.2. **The effect of manipulating reward magnitude.**

Consistent with previous P300 research that used conventional ERP peak-picking methodology (Hajcak et al., 2007; Wu & Zhou, 2009; Yeung et al., 2005; Zhou et al., 2010; cf. Lole et al., 2013; Sato et al., 2005; Yeung & Sanfey, 2004), Large wins elicited greater P3b amplitudes than equiprobable Small wins; this result provides further support for the notion that this ERP component reflects the significance of outcomes rather than their probability of occurrence.

The tendency for PGs to elicit similar P3b amplitudes following Small and Large wins as equally valuable (as indicated by a marginally significant Group × Outcome effect) suggests that these individuals are less effective at evaluating the value of reward, a result is consistent with research on individuals with substance use disorder (Goldstein et al., 2007). While this pattern of results needs to be treated with caution, taken with the trend for the PG group to elicit reduced P3b amplitudes over all outcome types, it implies that problem gamblers are less responsive to reward stimuli. Further research is needed to verify whether this tendency toward a hyposensitivity to gambling outcomes observed for the PG group is caused by a genuine hyposensitivity to reward, or repeated exposure to gambling activity.
In contrast with the majority of previous studies on the traditionally-conceptualised FRN (Hajcak et al., 2006; Sato et al., 2005; Yeung & Sanfey, 2004; Yu & Zhou, 2006b; cf. San Martin et al., 2010), the FRP was found to be sensitive to reward magnitude (although the between-group difference was not significant). The greater FRP factor scores for larger compared to smaller wins is inconsistent with previous observations of the FRN component reflecting the binary classification of outcomes as being either good or bad, or better or worse than expected (e.g., Hajcak et al., 2006; Holroyd et al., 2004; Gu et al., 2010; Sato et al., 2003; Toyomaki & Murohashi, 2005; Yeung & Sanfey, 2004; Yu & Zhou, 2006b; cf. Donamayor et al., 2011; San Martin et al., 2010; Yu & Zhou, 2009). The separation of the FRP from the FRN is likely to contribute to these discrepant findings; specifically, once this was achieved the latent nature of the FRRs to different magnitude wins could be observed. The finding of greater FRP amplitudes following Large compared to equiprobable Small wins further supports the notion that this component is separate from the novelty P3/P3a subcomponent, and corroborates previous research that employed a PCA to examine the latent nature of the FRR in healthy controls (Lole et al., 2013).

7.6.3. Psychophysiological responses to near-wins.

Greater FRN factor scores were found following Losses compared to Near-wins, although no between-group differences were found in response to these outcomes. No difference in P3b factor scores were found between Near-wins and Losses, and the Outcome × Group interaction for this planned contrast was not significant, suggesting that problem gamblers do not evaluate Near-wins as more motivationally significant than Losses compared to their non-addicted counterparts.
The reduced FRN amplitudes following Near-wins compared to Losses is consistent with previous ERP research (Lole et al., 2013; Luo et al., 2011) and further refutes the conjecture that the FRR is a reflection of a binary classification of outcomes being better or worse than expected (the proportion of near-wins, while realistic, may have contributed to the lack of a significant difference in P3b amplitude; see Kassinove & Schare, 2001). The fact that these outcomes occur so frequently during EGM gambling, and that they are less punitive than bona fide losses (seemingly reducing the aversive nature of non-rewarding/punishing stimuli, as indicated by reduced FRN amplitudes), may at least partly explain why EGMs have such wide appeal. It is important to note that, although the Near-wins presented in the current study did not involve monetary ‘return,’ these outcomes still elicited reduced FRN amplitudes compared to Losses; future research is required to determine whether true losses disguised as wins, as experienced during real EGM gambling, would actually elicit a FRP.

7.6.4. The relationship between psychophysiological and personality measures.
The latent neural correlates of incentive value processing identified using a PCA were not related to self-reported levels of impulsiveness in either group, or with reward sensitivity in the HC group. The current study also failed to find a correlation between FRN amplitude following Losses and ratings of punishment sensitivity in either the PG or HC group, as indicated by BIS scores, which is in contrast to previous research that used traditional ERP quantification methods (Balconi & Crivelli, 2010; De Pascalis et al., 2010; Santesso et al., 2011; Unger et al., 2012; Van den Berg et al., 2011). This was perhaps due to the fact that the relationship between the FRN and BIS scores was only quantified at Fz in the current study, and not at fronto-central (Santesso et al., 2011;
Unger et al., 2012), central (De Pascalis et al., 2010), or centro-posterior (Balconi & Crivelli, 2010) sites. Several self-report measures of reward sensitivity were found to correlate with psychophysiological indices of incentive processing in the PG group. PGs who reported experiencing new rewards as more exciting and being more willing to approach opportunities for reward (indicated by higher scores on the BAS Fun-seeking subscale) demonstrated larger P3b amplitudes upon experiencing Win outcomes, a result consistent with previous research on non-problem gamblers (Van den Berg et al., 2011). The fact that this relationship was not found for HCs in the current study is perplexing, but may be explained by the different psychological inventories used to assess BIS/BAS functioning and perhaps by the lower variability in reward sensitivity scores for the HC group between the two studies. Although PGs did not directly demonstrate attenuated P3b amplitudes following Win and Near-win outcomes, the tendency to elicit reduced P3b amplitudes following all Outcome types, suggests that further research is warranted to verify whether the personality variables found to correlate with this ERP component are useful indicators of underlying deficits in incentive processing.

7.6.5. Limitations and future directions.

The EGM task used in the current study was specifically chosen for its ecological validity and its clinical relevance for problem gambling; however, due to ethical reasons, participants were not allowed to wager (and lose) their own money, a limitation shared by previous ERP studies (Bellebaum & Daum, 2008; Bellebaum et al., 2010; Foti et al., 2011; Hajcak et al., 2006; Lole et al., 2013; Moser & Simons, 2009). Consequently, participants may have perceived Losses as non-reward rather than punishment, which may have resulted in reduced ERP component amplitudes (Van den
Berg, Shaul, Van der Veen, & Franken, 2012), particularly for the PG group (Hollander, Pallanti, Baldini Rossi, Sood, Baker, & Buchsbaum, 2005). Because small incentives (vouchers) were offered in our study, the results only indicate that PGs are less sensitive to wins of relatively small magnitude. It is possible that they respond differently to large ‘jackpot-sized’ wins, a proposition that can only be tested by transporting laboratory-based technologies into the noisy and artefact-ridden world of casinos or other licensed gaming venues, where there is the potential of winning and losing large amounts of money.

It is possible that individual differences in personality variables, or comorbid mental conditions (e.g., Shaffer & Korn, 2002), not accounted for in the current study are driving the attenuated FRN (e.g., Luu, Collins, & Tucker, 2000; see Segalowitz & Dywan, 2009) and the tendency toward reduced P3b amplitudes (Polich & Kok, 1995) observed in problem gambler group. Certain factors previously shown to influence ERN amplitude, such age (e.g., Falkenstein, Hoorman, & Hohnsbein, 2001; Gehring & Knight, 2000; Kok, 2000; Mathewson, Dywan, & Segalowitz, 2005; Nieuwenhuis et al., 2002; Wiersema, van der Meere, & Roeyers, 2007), levels of impulsivity (Martin & Potts, 2009), and reward and punishment sensitivity (Torrubia, Ávila, Moltó, & Caseras, 2001), were ruled out as influencing the dependent measures because PGs and HCs did not differ in age or on psychological inventories designed to measure these personality variables (i.e., the Impulsiveness questionnaire and BIS/BAS scales). However, more control over other individual differences in personality factors, such as levels of neuroticism (Pailing & Segalowitz, 2004) and socialisation (Dikman & Allen, 2000), is required before the attenuated responses found in this study can be purely attributed to between-group differences in incentive value processing.
The reason problem gamblers are less effective at assessing environmental cues remains unclear, and requires further research. Unfortunately, the current study was unable to determine whether the hyposensitivity to gambling outcomes observed in PGs was the result of an inherent disposition or was primarily a consequence of repeated exposure to gambling activity. Further investigation on several issues is warranted in order to determine the aetiology and lifetime progression of problem gambling. To this end, it would be of value to determine whether the attenuated responses of the problem gambler cohort are the result of dysfunctional neural processes related to the production and transmission of appropriate error signals and/or the neural structures associated with reward and punishment processing, and how such factors may interplay with psychosocial factors (e.g., ethnicity, cultural and social norms, personality, emotional development, early experiences, and exposure to gambling situations).

7.6.6. Conclusion.

The current study provides valuable insight into the underlying neural mechanisms associated with incentive processing in problem gamblers through application of a PCA. Using this analysis method, two distinct feedback-related responses, the FRN and FRP, were identified, further consolidating results from previous research using conventional ERP quantification methods. These responses were found to be reliable indices of outcome valence and magnitude, and to differentiate the responses of problem gamblers from healthy controls. The P3b was also found to differentiate valence and magnitude manipulations. Although no between-group differences were found following Wins and Losses, problem gamblers tended to elicit similar P3b amplitudes following Large and Small wins. The attenuated FRN amplitudes following Losses, attenuated FRP amplitudes following Wins, and the tendency for problem gamblers to elicit similar P3b
amplitudes following both Large and Small wins, provides evidence for a hyposensitivity to punishment/non-reward and reward in these individuals. These findings have the potential to, at least partly, explain the problem behaviours characteristic of this disorder, including continued gambling despite adverse consequences, and gambling with larger amounts of money and for longer periods of time in order to experience the same amount of excitement and satisfaction as non-problem gamblers. The current study was the first to examine the ERP responses of problem gamblers to Near-wins; these outcomes were found to be perceived as less unfavourable than Loss outcomes for both PGs and HCs, indicating that they may increase the attractiveness of EGM gambling. In summary, the current study provides important information on how problem gamblers respond to outcomes commonly encountered during EGM gambling. The results obtained may provide a foundation from which future research, clinical treatments, and interventions can be developed.
CHAPTER EIGHT – General Discussion

The primary aim of the current thesis was to investigate whether the psychophysiological responses of problem gamblers to various EGM outcomes differentiate them from non-problem gamblers. Following confirmation that the autonomic and cortical measures employed in the current body of work reliably reflect incentive value processing, and are sensitive to differences in the valence and magnitude of gambling outcomes (Study A/Chapter Four and Study C/Chapter Six), an investigation on whether these measures indicate abnormally high or low reactivity to reward and/or punishment in problem gamblers was accomplished (Study B/Chapter Five and Study D/Chapter Seven). A number of key differences in the psychophysiological responses of problem gamblers to outcomes encountered during gambling on EGMs were discovered.

This chapter includes a summary and discussion of i) the reliability of the autonomic and cortical measures used in the current program of research to indicate incentive value processing (Section 8.1.), ii) the hyposensitive response to reward and punishment/non-reward observed in problem gamblers compared to non-problem gamblers (Section 8.2.), and the implications this finding has for theoretical conceptualisations of problem gambling as a disorder (Section 8.2.1.) and for clinical settings (Section 8.2.2.), iii) the psychophysiological responses of problem and non-problem gamblers in response to outcomes of varying magnitude (large vs. small) (Section 8.3.), iv) the responses of problem gamblers and non-problem gamblers to losses disguised as wins (LDWs) and near-wins (Section 8.4.), and v) the relationship between psychophysiological correlates of incentive processing and individual differences in personality traits, including impulsivity and sensitivity to reward and
punishment (Section 8.5.). This chapter then goes on to consider the strengths (Section 8.6.) and shortcomings of the current body of work, and suggestions for future research are proffered (Section 8.7.).

8.1. Psychophysiological Measures are Able to Reliably Index Incentive Processing

A number of methodological and analytical advances were achieved in the current thesis, overcoming the limitations associated with past research. These approaches allowed a more accurate representation of the psychophysiological responses that occur following outcomes of varying incentive value during gambling activity, and a clearer understanding of the nature of deficit in problem gamblers.

To date, the majority of previous psychophysiological gambling research has employed tonic methods to examine the global effect of gambling activity on autonomic nervous system arousal, often in comparison to periods of non-gambling activity (see Section 4.3.2.). The current body of work employed phasic methods to examine the immediate effect individual gambling outcomes have on psychophysiological responses, allowing a better understanding of the significance win and loss outcomes have for both problem and non-problem gamblers. Such an approach is vital to advancing theoretical conceptualisations of problem gambling; specifically, whether problem gamblers are more or less excited by the experience of reward and/or punishment.

The use of LedaLab software (Benedek & Kaernbach, 2010) to analyse skin conductance responses (SCRs) collected in licensed gaming venues in Study B (Chapter Five), allowed an accurate estimation of the psychophysiological responses to wins, losses, and losses disguised as wins (LDWs), without the influence of consecutively occurring outcomes. This was particularly important, since EGM gambling allows bets
to be placed in rapid succession, meaning that both bet and outcome are experienced in a short period of time. Ledalab uses sophisticated algorithms to estimate sympathetic nervous system arousal in response to individual stimulus presentations and separates it from tonic skin conductance levels. Analysing electrodermal activity in this manner reduces the likelihood of underestimating SCR amplitudes, a problem that is common in paradigms that employ short interstimulus intervals between consecutive stimulus presentations (Bouscein, 1992). Moreover, the use of ERP-style averaging techniques to examine electrodermal activity data further facilitated the investigation of responses to gambling activity outside of sanitised laboratory settings, in the artefact-ridden gaming lounges of licensed club venues in Study B (Chapter Five).

The computer task used in Studies A, C, and D (Chapters Four, Six, and Seven) improved on methodology from previous research (e.g., Wilkes et al., 2009, 2010) by time-locking psychophysiological responses to the exact time each outcome occurred. This approach allowed the investigation of cortical ERP responses of problem gamblers to manipulations of valence, something that has only been achieved by only one other study to date (Hewig et al., 2010). The superior temporal resolution is a major advantage of using ERPs to examine underlying cognitive processing; thus, using this measure is particularly useful in examining the immediate cortical activity of both problem gamblers and non-problem gamblers following instances of reward and punishment/non-reward. Moreover, the application of a principal components analysis (PCA) to investigate the ERP correlates of incentive processing in problem gambling represents a novel contribution of this body of work, since such an approach has not been employed in previous research. This data analysis procedure parsed the feedback related responses (FRR, i.e., the FRN and FRP) and P3b from overlapping ERP components, uncovering differential response patterns to the same psychological
stimuli, something that is particularly exciting, as the FRRs and P3b components each present independent indices of incentive processing. It is possible that these components reflect the functioning of different cortical structures (although both may involve activity of the neurotransmitter dopamine, see Volkow et al., 2004); for instance, the FRN may reflect activity in the ACC (Bellbaum & Daum, 2008; Donamayor et al., 2011; Luu et al., 2000; Miltner et al., 1997; Nieuwenhuis et al., 2005; Potts et al., 2011; Yu & Zhou, 2009a; Zhou et al., 2010), whereas the P3b may reflect activity in the temporal-parietal junction (Halgren et al., 1980; Johnson, 1988; McCarthy et al., 1989; Nieuwenhuis et al., 2005), both structures which are likely to be part of a larger reward and punishment processing system (e.g., Polich, 2007). The findings of the PCA resorted in Studies C (Chapter Six) and D (Chapter Seven) help to consolidate the disparate findings of previous literature. Whereas earlier conceptualisations characterised the FRN as a greater negative response following negative (Gehring & Willoughby, 2002a; Holroyd et al., 2004; San Martin et al., 2010; Toyomaki & Murohashi, 2005; Yeung et al., 2005), more recently research has suggested that this component may be better conceptualised as a positive deflection following favourable outcomes (Foti et al., 2011; Holroyd et al., 2003; Holroyd et al., 2008; see Sections 2.2.2.1., 6.3., and 7.3). The results of these two studies suggest that the feedback related response is made up of both these components, a finding that may help to elucidate the underlying mechanisms involved in incentive value processing.

In summary, the autonomic and neural measures used in the current thesis were found to reliably index incentive value processing in non-problem gamblers, and were successfully used to investigate the nature of deficit in problem gambling. The methodological and analytical advancements made in this body of work are likely to prove valuable in future investigations of incentive value processing, particularly in
individuals with disorders that are associated with abnormal reward and punishment/non-reward sensitivity, such as problem gambling.

8.2. **Problem Gamblers are Hyposensitive to Both Reward and Punishment**

The results of the current body of research provide compelling evidence that wins are more motivationally significant than losses for healthy, non-problem gamblers; however, the most noteworthy finding of this thesis is the hyposensitive response exhibited by problem gamblers following the experience of both reward (Studies B and D/Chapters Five and Seven) and non-reward (Study D/Chapter Seven). Rather than being hypersensitive to reward, as posited by some theoretical accounts of problem gambling (see Section 8.2.1.), the results provide evidence that problem gamblers are less reactive to win (at least to small wins) and loss outcomes compared to their healthy counterparts. This hyposensitive response was found in electrodermal (Study B/Chapter Five) and ERP (Study D/Chapter Seven) measures, and presents a robust finding that is consistent with previous neuroimaging research demonstrating reduced activation in the reward circuitry of the brain of problem gamblers following the actual experience of both positively and negatively valenced outcomes (de Ruiter et al., 2009; Reuter et al., 2005; Tanabe et al., 2007). The discrepancy between these results and the findings of previous studies that have reported a hypersensitivity to reward in problem gamblers (e.g., Hewig et al., 2010; Miedl et al., 2010; Oberg et al., 2011; van Holst et al., 2012) may reflect differential effects of anticipating vs. actually experiencing rewarding outcomes (see Section 2.3.). Specifically, problem gamblers may be more excited by potential reward, but less sensitive to actual reward attainment. Further research is required to verify and directly examine the motivational significance anticipated wins compared to the actual experience of wins have in problem gamblers, and the respective
roles these reward contingencies play in the development and maintenance of this disorder. Nevertheless, the current body of research was the first to examine the psychophysiological reactions of problem gamblers that occur following the actual experience of unambiguous win and loss outcomes using an EGM paradigm. This is an important contribution to the current field of gambling research, considering the potential for harm associated with this form of gambling, and furthers the understanding of the nature of deficit in this disorder.

8.2.1. Theoretical implications of the current research findings.

By investigating the immediate responses to win and loss outcomes that occur during gambling on electronic machines, the current thesis helps clarify and advance theoretical conceptualisations of problem gambling. In this section, the attenuated response to reward and punishment/non-reward observed in the problem gambler cohorts of Studies C (Chapter Six) and D (Chapter Seven) are discussed in terms of theoretical accounts that posit problem gambling stems from a state of abnormal arousal levels (Section 8.2.1.1.), and those that hypothesise that this disorder results from dysfunction in cortical structures believed to be involved in performance monitoring and incentive processing (Section 8.2.1.2.). It should be noted that such theoretical accounts interpret the maladaptive behaviours associated with problem gambling as the result of abnormal brain functioning that precedes the development of problem gambling; however, further research is required to determine whether this disorder is indeed the result of a biological predisposition toward addiction, or if it is the result of repeated exposure to gambling (see Section 8.7. for a further discussion on this point).
8.2.1.1. **Deficits in arousal mechanisms.**

The hyposensitive response of problem gamblers observed in both autonomic (Study B/Chapter Five) and cortical (Studies D/Chapter Seven) domains reported in this thesis may be indicative of a general state of hypoarousal in these individuals; for instance, problem gamblers may be characterised by lower tonic levels of arousal before they develop this disorder, and/or by a lower reactivity to both win and loss outcomes during gambling activity. Problem gamblers in the current body of work exhibited attenuated skin conductance responses to wins during actual gambling activity (Study B/Chapter Five), and a tendency to elicit lower P3b amplitudes following all outcome types during play on the computer EGM task (Study D/Chapter Seven). Both of these measures have been previously shown to reflect levels of cortical arousal (Barry, 1996; Barry et al., 2004; Kahneman, 1973; Lykken & Venables, 1971; Raskin, 1973), and reduced P300 amplitudes have been found in individuals with low arousal levels (Gurrera et al., 2001; Stelmack & Houlihan, 1994; cf. Brocke, 2004; Brocke et al., 1997; De Pascalis, 2004; Stenberg, 1992); thus the results observed in the current thesis imply that problem gamblers may be hypoaroused. Accordingly, the individual outcomes experienced during gambling activity may not be the main motivator for the continued harmful gambling activity associated with this disorder. Likewise, the reduced FRN amplitudes to losses and near wins may indicate that negatively valenced outcomes are not sufficient deterrents for problem gamblers. Rather, individuals with this disorder may gamble in order to increase arousal levels and compensate for a chronically underaroused state.

This interpretation of the results found in the current thesis is consistent with some arousal-based theories of problem gambling, but not others. The results from the current body of work are not compatible with the postulation made in the cognitive-
behavioural model proposed by Sharpe and Tarrier (1993), which predicted problem
gamblers to be hypersensitive to the intermittent reinforcement schedule of reward
experienced during EGM gambling resulting from previous conditioning effects and
other mediating cognitive factors. It is to be determined if this hyposensitivity observed
for small wins extends to larger wins, and to subtypes of gamblers addicted to other
gambling mediums (e.g., Blaszczynski & Nower, 2002).

The results of the current thesis may be consistent with other arousal-based
behavioural theories that posit problem gamblers are hypoaroused and gamble in order
to increase arousal levels (e.g., Brown, 1986; Jacobs, 1986; McConaghy, 1980).
Unfortunately, the lack of a between-group comparison of tonic pre-gambling baseline
levels of arousal, and how they are related to activation during gambling was not
examined, so the assumptions of these theories could not be adequately tested.
Moreover, because the role that arousal plays in the modulation of the FRN has not
been thoroughly researched, it is not clear whether the attenuated FRN amplitudes
found for problem gamblers in Study D (Chapter Seven) are consistent with theories
that postulate that the aberrant behaviours exhibited by problem gamblers are due to
these individuals being hypoaroused. Thus, although the results of the current thesis are
largely consistent with theoretical accounts that posit problem gambling is caused by a
general state of hypoarousal; further research is required to verify this notion.

Other theoretical interpretations suggest that the main motivator for problem
gamblers to participate in gambling activity is that is provides a means of escape from
negative emotional states (such as depression) and provides an escape from life
problems (e.g., Blaszczynski & Nower, 2002); thus, devaluing the role of arousal in this
form of gambling. Previous research has shown that, compared to problem gamblers
whose preferred gambling medium is horse racing, problem gamblers who favour EGM
gambling report that they prefer lower levels of arousal (e.g., Cocco et al., 1995). However, in this study only subjective evaluations were examined, and the levels of physiological activity during gambling were not directly examined. Other research has shown that increased severity of gambling behaviours is associated with higher levels of self-reported arousal during EGM gambling (e.g., Brown et al., 2004; Diskin & Hodgins, 2003; Griffiths, 1990, 1991, 1995), although this is not reflected in higher physiological arousal levels during EGM gambling (Carroll & Huxley, 1994; Coulombe et al., 1992; Coventry & Constable, 1999; Diskin & Hodgins, 2003; Diskin et al., 2003; Griffiths, 1993; Stewart et al., 2006). While these results suggest that arousal plays a limited role in the development and maintenance of gambling behaviours, such research is inconclusive. For example, the majority of the studies mentioned above have only examined between-group differences in subjective report or have used measures such as heart rate to examine physiological arousal, not skin conductance. Moreover, although EGM gambling may be less associated with arousal compared to other forms of gambling, research has suggested that gambling-related arousal is mode-specific, and is dependent on the preferred gambling medium of the gambler. For example, problem gamblers whose preferred gambling medium is EGMs have been shown to elicit higher arousal levels when exposed to EGM cues, compared to cues related to other gambling forms, such as horse racing (Sharpe et al., 1995); thus, although EGM gamblers may prefer comparatively lower levels of arousal, the excitement experienced during this form of gambling may still play an important role in this disorder. Furthermore, depression levels did not significantly differ between the problem gambler and healthy control groups in Study D/Chapter 7, implying that the between group differences found are not the result of comorbid mental health conditions. Anecdotal evidence suggests that EGM problem gamblers are primarily interested in obtaining features during play;
whether this is because they are seeking to experience the excitement associated with these outcomes, or the extended time it allows them to play (and thus, continue to escape outside problems and/or validate their gambling behaviours) is unclear. As mentioned above, further research is required to determine the role the experience of physiological arousal plays in ongoing problematic gambling behaviours associated with this disorder is required.

8.2.1.2. **Dysfunction in specific cortical structures.**

Alternatively, the attenuated responses to reward and punishment/non-reward stimuli may be the result of deficits in specific cortical structures and/or systems that are involved in goal-driven action planning and in performance monitoring (Ridderinkhof, van den Wildenberg, Segalowitz, & Carter, 2004). These structures have been shown to be common neural generators of electrodermal activity and the FRR and P300 ERP components examined in the current thesis. Thus, the reduced responsivity to gambling outcomes displayed by problem gamblers may reflect dysfunction in such neural generators. The following sections interpret the findings of the current thesis in terms of theoretical accounts of problem gambling that posit the abnormal behaviours associated with this disorder are the result of dysfunction in structures of the mesolimbic reward system, specifically in the ventromedial prefrontal cortex (Section 8.2.1.2.1.) and the anterior cingulate cortex (Section 8.2.1.2.2.)

8.2.1.2.1. **Deficits according to neurobiological accounts of addiction.**

Interpreted in light of neurobiological accounts of addiction, the reduced response to reward in problem gamblers found in Study B (Chapter Five) and Study D (Chapter Seven) is consistent with the assumptions of the *reward deficiency syndrome* hypothesis
(Blum et al., 2000), potentially indicating malfunction of dopamine D₂ receptors within the mesolimbic reward system of affected individuals. Other theoretical accounts posit that poor evaluation of incentive value is the result of reduced functioning in specific structures within this system, such as the ventromedial prefrontal cortex (VMPFC) and/or the orbitofrontal prefrontal cortex (OFC; Damasio, 1994; Lodge, 2011). This cortical region has been shown to be associated with the executive control of behaviour and with emotional responding (Damasio, 1994, 1996). It has also been implicated in the regulation of electrodermal activity, particularly in situations involving emotional processing (Tranel & Damasio, 1994), decision-making (Bechara, Damasio, Tranel, & Damasio, 1997; Bechara, Tranel, Damasio, & Damasio, 1996; Critchley et al., 2000), stimulus orienting (Raine et al., 1991), as well as during gambling scenarios (Patterson et al., 2002); thus, the reduced SCRs following reward outcomes observed in Study B is consistent with abnormal functioning within this cortical region. Structural MRI research has indicated grey matter volume in the VMPFC, the OFC, and the dorsolateral prefrontal cortex is associated with reduced P300 amplitudes following monetary reward (Parvaz, Konova, Tomasi, Volkow, & Goldstein, 2012); accordingly, the tendency for problem gamblers to exhibit attenuated P3b amplitudes following all outcome types in Study D (Chapter Seven) may be explained by abnormalities in these brain regions in individuals with this disorder.

The hyposensitivity to both reward and punishment observed in problem gamblers may be explained by the somatic marker hypothesis (Damasio, 1994). The reduced sensitivity to reward and punishment may be the result of atypical somatic marker production, resulting from malfunction in the VMPFC and/or the OFC, may lead to the development of problem gambling behaviours (see also Cavendi, Riboldi, Keller, D’Annucci, & Bellodi, 2002; Lodge, 2011); specifically, affected individuals
may be unable to effectively associate appropriate emotional reactions (somatic markers) to the physiological responses that occur following both win and loss outcomes, and therefore cannot use these markers to guide future decision making. As such problem gamblers are hypothesised to choose short-term gratification over long-term advantages (i.e., choosing to continue gambling despite repeated and often severe losses). Previous neuroimaging research has provided support for the assumptions of the somatic marker hypothesis. Reduced activation of the cortical regions implicated in somatic marker production (i.e., the VMPFC and related structures) has been demonstrated in problem gamblers during reward and punishment/non-reward processing (De Ruiter et al., 2009; Reuter et al., 2005; Tanabe et al., 2007). This pattern of reduced responding has been found to be mediated by the severity of gambling symptoms (Reuter et al., 2005). Thus, the findings of the current thesis are also consistent with the theoretical interpretation that dysfunction of the VMPFC underlies the problematic behaviours observed in individuals with addictive disorders, including problem gambling.

8.2.1.2.2. Dysfunction of the anterior cingulate cortex.

It is also possible that the pattern of reduced responding found across autonomic and cortical domains in the current thesis are the result of abnormal activity in the anterior cingulate cortex (ACC). The reduced skin conductance responses to reward stimuli observed in Study B (Chapter Five), may be indicative of abnormal activity in the ACC, since this cortical structure has been previously shown to be associated with the generation of electrodermal activity, particularly with the autonomic regulation of motivational states (Naqvi et al., 2007). The reduced FRN amplitudes following losses, attenuated FRP amplitudes following wins, and tendency for reduced responses
following all gambling outcomes for problem gamblers in Study D (Chapter Seven) are also consistent with the notion of reduced ACC functioning. While some research has provided preliminary evidence for activity in the VMPFC to be associated with FRN amplitudes (Oberg et al., 2011), the majority of evidence suggests that the ACC is the primary neural generator of the FRN ERP component (as outlined in Section 2.1.); thus, the attenuated FRN amplitudes following losses and FRP amplitudes following wins for the problem gambler group in Study D (Chapter Seven) are likely to reflect abnormal activity within the ACC (cf. Carlson et al., 2011; van Veen et al., 2004).

According to the reinforcement learning hypothesis (Holroyd & Coles, 2002), these attenuated responses reflect the inability of the ACC in affected individuals to transmit appropriate dopaminergic signals to the basal ganglia (see Section 2.1.). The problems associated with problem gambling may reflect a reduced ability to properly evaluate the incentive value of environmental stimuli and to learn from previous experience. This interpretation has received support from a neuroimaging study by Campbell-Meiklejohn et al. (2008), which demonstrated reduced activation in the ACC following decisions to chase losses (see Section 2.1.). Thus, attenuated FRN amplitudes of problem gamblers observed in Study D, and the problematic behaviours that characterise this disorder are consistent with the theoretical interpretation that dysfunction in the ACC leads to a reduced ability to evaluate environmental outcomes in terms of their incentive value, leading to the problematic behaviours associated with this disorder, such as continued gambling despite repeated and often severe losses.

Suboptimal functioning of the ACC may also explain the tendency for problem gamblers in Study D (Chapter Seven) to elicit attenuated P3b amplitudes following all outcome types. The inhibition hypothesis outlined by Polich (2007) posits that the P300 complex reflects the cognitive process that serves to select relevant information from
the environment whilst disregarding non-important aspects. According to this supposition, the P3a subcomponent reflects the processing of attention-driven neural activity towards novel stimuli that are perceived to be important according to principles of the orienting reflex. This component is believed to be generated in the ACC and other related frontal cortical structures. This information is believed to be transmitted to temporal-parietal areas for memory updating, which is reflected by the P3b subcomponent (Gazzaniga et al., 2000); thus, dysfunction in the ACC may have carry-over effects for P3b amplitude. The findings from structural MRI research conducted by Parvaz et al. (2012) provide some support for this notion. This study found that reduced grey matter volume in the ACC (in addition reduced volume in the VMPFC, the OFC, and dorsolateral prefrontal cortex) is associated with reduced P300 amplitudes following monetary reward.

8.2.1.3. Conclusions on the theoretical interpretations of the current research findings.

While the current thesis provides valuable information on which theoretical accounts of problem gambling have empirical support, unfortunately, the precise biological mechanisms behind the attenuated responses to positive and negative outcomes observed in the current thesis were not able to be determined. It seems likely that the hyposensitive responses found in both electrodermal and cortical activity reflect dysfunction in a common underlying mechanism or network of cortical processes, perhaps resulting from abnormalities in the neural generators of the psychophysiological responses examined in this thesis and/or in arousal mechanisms. Further research using neuroimaging methods, preferably in conjunction with electrophysiological methods such as event-related potentials, is required to determine
the cortical structures that are implicated in the attenuated autonomic and ERP responses observed in the problem gambler samples within the current body of work. As mentioned previously, it is also vital to determine the direction of causality for the observed abnormalities in incentive value processing (see Section 8.7.).

8.2.2. **Clinical implications of the current research findings.**

The current body of research did not examine whether the observed hyposensitivity to gambling outcomes is the result of an inherent disposition that contributes to the development of this disorder, or whether it is a consequence of problem gambling behaviours (see Section 8.7.). Nevertheless, the observed pattern of reduced sensitivity to reward and punishment has important clinical implications. The findings of the current body of research provide valuable insight into the nature of deficit in problem gambling, and have the potential to explain the problematic behaviours associated with this disorder. As mentioned above (Section 8.2.1.), the reduced reactivity to reward observed in both autonomic and cortical ERP domains may indicate a common underlying deficit in evaluating the incentive value of stimuli and/or in arousal mechanisms (however, further research is required to validate this notion). For example, the phasic hyposensitive response to reward may contribute to the desire of problem gamblers to seek larger magnitude rewards or bonus features (e.g., free games, second screen games, scatters, or substitutes), and to gamble with higher stakes and/or for longer of periods of time in order to experience the same level of excitement as non-problem gamblers do during non-harmful gambling activity. If the conjecture that a state of tonic hypoarousal underlies this disorder, problem gamblers may seek out exciting activities such as gambling, and engage in increasingly harmful gambling behaviours to achieve a state of optimal arousal. The reduced FRN following losses
exhibited by problem gamblers in Study D (Chapter Seven) indicates that these individuals may be less effective at processing negatively valenced stimuli. Such dysfunction may explain the tendency for problem gamblers to continue gambling despite repeated and often severe losses. When combined with the capacity for large amounts of money that can be spent on EGMs, this abnormal responding to both wins and losses has the potential to lead to devastating financial, familial, and psychological problems. Endeavours to reduce the adverse effects of the EGM by systematically manipulating key variables (e.g., decreasing the rapidity of events, enhancing the negative impact of losses by associating it with appropriate stimuli, and increase arousal effects by means other than increasing bet size) may yield productive outcomes for the prevention of problem gambling.

If the pattern of hyposensitivity to reward and non-reward/punishment stimuli is a sufficiently robust and reliable phenomenon that can be replicated in further research, these blunted responses have the potential to serve as a biomarker for this disorder. Should this occur, a characteristic profile could be applied in clinical settings to aid diagnosis, and to guide clinically-based interventions, including cognitive-behavioural therapy and psychoeducation programs (e.g., Gooding & Tarrier, 2009; Ladouceur & Walker, 1996; Petry, Alessi, Carroll, Hanson, MacKinnon, Rounsaville, & Sierra, 2006; Walker et al., 2007), motivational therapies (Brewer, Grant, & Potenza, 2008; Hodgins, Currie, Currie, & Fick, 2009), and biobehavioural-style (e.g., biofeedback) treatments. For example, clinical treatments for problem gambling have focussed on trying to reduce gambling urges when exposed to gambling stimuli (Oakes, Battersby, Pols, & Cromarty, 2008; Tolchard, Thomas, & Battersby, 2006; Townshend, 2005); the results from the current body of research suggest that treatment outcomes for problem gamblers may be improved if there is a focus on addressing a response deficit to reward and
punishment stimuli, or to a hypoaroused state, perhaps through neurofeedback training. Furthermore, changes in such abnormal responses may be used to indicate the success of a course of clinical intervention (e.g., Freidenberg, Blanchard, Wulfert, & Malta, 2002), or as an indicator of likely relapse. An advantage of using such physiological measures for these purposes over self-report ratings of subjective experience is that the former are more objective, and less prone to deliberate and unintentional distortions. Moreover, if future research confirms that the observed hyposensitive response to reward and punishment/non-reward is an inherent pattern of responding that precedes addictive gambling behaviours (as predicted by several theoretical models, e.g., Blum et al., 2000; Damasio, 1994), there is the potential for this response pattern to be used as a screening tool; specifically, the attenuated responses to reward and punishment stimuli may be used to aid the early detection and intervention for individuals at risk of developing problematic gambling behaviours.

8.3. **Effects of Reward Magnitude and Bet Size on Psychophysiology**

Previous research on substance use disorder revealed that addicted individuals abnormally process the incentive value of reward (e.g., Goldstein et al., 2007). The current thesis was the first to examine whether the psychophysiological responses of problem gamblers following different magnitude (i.e., large vs. small) outcomes also indicate dysfunctional reward processing.

Larger magnitude rewards were found to elicit greater autonomic responses in a sample of naive gamblers compared to smaller wins (Study A/Chapter Four), indicating that individuals who do not report gambling-related problems associate more value with these outcomes compared to smaller magnitude reward. Unfortunately, due to technological limitations (see Section 5.6.3.), the autonomic reactions of problem
gamblers to different magnitude reward during actual gambling activity was unable to be examined in Study B (Chapter Five).

The results from ERP studies C (Chapter Six) and D (Chapter Seven) revealed the FRP component to be sensitive to reward magnitude, with greater amplitudes following large compared to small wins found for both problem and non-problem gamblers. While non-problem gamblers also elicited greater P3b amplitudes following larger compared to smaller wins in these studies, the problem gamblers in Study D were found to elicit similar P3b amplitudes these outcome types, providing further evidence for deficiency in reward processing in this disorder (e.g., Blum et al., 2000). As mentioned above (Section 8.2.1.), it is possible that problem gamblers are hypersensitive to the promise and/or achievement of increased profits associated with very large wins (e.g., Blaszczynski et al., 2001), such as what is experienced when winning the jackpot, major prize, or free games (i.e., features), but are less motivated by smaller magnitude wins. Unfortunately, due to the relatively infrequent occurrence of very large magnitude wins during actual EGM play in Study B (Chapter Five), and the relatively small magnitude of large win outcomes (compared to actual gambling activity) presented in Study D (Chapter Seven), this was not able to be determined in the current body of work. Nevertheless, this thesis provides compelling evidence that problem gamblers are at least less reactive to more commonly occurring smaller magnitude wins.

Surprisingly, placing different sized wagers was not reflected in SCRs or heart activity in Study A (Chapter Four). Likewise, preliminary analyses in ERP Studies C and D (Chapters Six and Seven) showed that bet size did not influence the amplitudes of the P3b or FRN ERP components, indicating that placing high or low value bets does not differentially affect psychophysiological responses during EGM play. It is possible
that the lack of a significant difference following decisions to place high or low wagers was due to the small differences in the bet size options available (bet sizes on the computer tasks used in Studies A, C, and D ranged from credit values equivalent to AUD $0.01 to AUD $0.10); thus, participants may have not have perceived the different amounts lost after placing different sized wagers to be remarkable, especially since they were not losing their own money. Further research is required before accepting the null hypothesis that placing different sized wagers does not affect psychophysiological responses, preferably by examining larger differences in the amount bet, and when participants wager their own money rather than allocated credits.

8.4. The Significance of Almost Winning: Losses Disguised as Wins and Near-wins

Losses disguised as wins (LDWs), outcomes in which the amount returned is less than the amount wagered, are a salient design feature of EGMs. Because these outcomes are technically losses, but are accompanied by auditory and visual information similar to wins, they are believed to give gamblers the illusion that they are winning more often than they actually are, and therefore, promote continued gambling behaviours (Dixon et al., 2010). Previous autonomic research on naive gamblers has demonstrated LDWs to elicit similar SCRs to wins (Dixon et al., 2010). Losses disguised as wins have been suggested to encourage the maladaptive and persistent gambling behaviours associated with problem gambling (Dixon et al., 2010; Livingstone & Woolley, 2008); however, previous research has been restricted to naive gamblers who experience these outcomes during laboratory-based EGM play, and the psychophysiological responses of problem gamblers to these outcomes have not been examined. Moreover, it remains unclear as to whether gamblers are primarily motivated by the visual and auditory stimuli similar to
what accompany wins, and/or the small monetary ‘returns,’ associated with these outcomes, or rather, by the significance of the symbols ‘almost’ matching.

The current thesis sought to address these issues. Study B (Chapter Five) in the current body of work outlines the findings from the first study in the extant gambling literature to examine phasic SCRs of experienced non-problem gamblers and problem gamblers to LDWs during actual gambling in real club settings. Autonomic and cortical reactions to near-wins, outcomes in which the matching combination of symbols needed for a win are ‘almost’ achieved, but are not associated with the return of credits, or superfluous auditory tunes or visual cues, were also examined in samples of naive gamblers (Study A/Chapter Four and Study C/Chapter Six), experienced non-problem gamblers, and problem gamblers (Study D/Chapter Seven).

8.4.1. Autonomic reactivity to near-wins and losses disguised as wins.

The lack of a significant difference in heart rate or skin conductance responses between near-wins and losses reported in Study A (Chapter Four) suggests that near-win outcomes are not more exciting than losses for naive gamblers. Corroborating this finding, the lack of a difference between losses and true LDWs (i.e., where actual credits are returned) in Study B indicates that LDWs do not induce more excitement than losses for either problem gamblers or experienced non-problem gamblers. These findings are in contrast with previous autonomic research which found naive gamblers to elicit greater SCRs to true LDWs during gambling in laboratory conditions (Dixon et al., 2010).

When considered together, the results from the current thesis and previous research suggest that, for naive gamblers, who have had either limited or no previous exposure to gambling, the excitement caused by small monetary returns, and/or the
flashy visual and catchy auditory stimuli that are associated with LDWs, contribute to the perception that these outcomes are rewarding (Dixon et al., 2010); nearly winning (i.e., where the symbols nearly match up but no credits are returned, and no associative auditory and flashy visual stimuli are presented) is not important (Study A/Chapter Four). Recent research that directly manipulated the presence of auditory stimuli during EGM gambling supports this notion. Dixon, Harrigan, Santesso, Graydon, Fugelsang, and Collins (2013) found that, when the auditory theme music normally associated with EGMs was presented to naive gamblers during gambling, higher SCRs were elicited compared to when no such auditory stimuli were presented. (The results of Study A suggest that the return of credits associated with LDWs is also important; win outcomes, which, unlike near-wins, were associated with the return of credits, elicited greater SCRs than losses in that study, despite the absence of associated visual and auditory cues).

The fact that no difference between electrodermal activity following LDWs and losses was observed for the non-problem gambler or the problem gambler groups in Study B (Chapter Five) is somewhat surprising, considering previous research findings (e.g., Dixon et al., 2010) and theoretical conjectures that hypothesise these outcomes contribute to the development and maintenance of problematic gambling behaviours (e.g., Dixon et al., 2010; Livingstone & Woolley, 2008). Unlike the near-win stimuli presented in Study A, the monetary returns and the visual and auditory cues normally associated with LDWs were presented in Study B. It is possible that the large SCR to wins observed in Study B/Chapter 5 are primarily the result of experiencing large wins. Further research needs to be conducted to test whether smaller magnitude wins also elicit an increased SCR, since anecdotal evidence suggests that these outcomes elicit no observable behavioural reactions. It is possible that the large SCR to wins observed in
Study B/Chapter 5 is primarily the result of experiencing large magnitude wins. Further research needs to be conducted to test whether smaller magnitude wins also elicit an increased SCR, since anecdotal evidence suggests that these outcomes elicit no observable behavioural reactions. It is possible that the lack of different results between LDWs and Losses in Study B/Chapter 5 was due to participants being uninterested in outcomes that are more similar to small magnitude wins (than they are to large magnitude wins). Unfortunately, because the responses to large and small win outcomes were averaged together, that study was not possible to test this proposition. Alternatively, the findings of the current thesis may suggest that LDWs (particularly their associative visual and auditory stimuli and/or monetary ‘return’) contribute to the development of gambling behaviours in naive gamblers, but do not elicit the same reactions in experienced non-problem gamblers or problem gamblers, perhaps due to repeated exposure to these outcomes. Further research is required to test these possibilities.

8.4.2. Cortical reactivity to near-wins.

The current dissertation was the first to examine the responses of problem gamblers following near-wins using ERP indices of incentive processing. Previous electrophysiological research on naive gamblers reported greater P300 amplitudes following near-wins compared to losses (Qi et al., 2011). In contrast to these findings, the lack of a difference in P3b amplitude between losses and near-wins for both non-problem gamblers (Study C/Chapter Six and Study D/Chapter Seven) and problem gamblers (Study D/Chapter Seven) indicates that these outcomes are not as rewarding as wins. These results are also inconsistent with previous fMRI research that found near-wins experienced during play on a computer gambling task elicit similar neural activation to wins for naive gamblers in laboratory environments (Clark et al., 2009),
particularly for regular EGM players at higher risk for developing disordered gambling behaviours (Chase & Clarke, 2010). The reason for these discrepant findings is unclear. It is possible that increased activation following reward found in previous neuroimaging research (Chase & Clarke, 2010; Clark et al., 2009) better reflects frontal-lobe incentive value processing, rather than processes associated with P3b generation. The discrepant ERP findings may be due to the increased salience of nearly winning in previous research paradigms; specifically, win outcomes in that study occurred when three matching symbols were presented, whereas near-wins occurred when two out of three symbols matched (e.g., Qi et al., 2011).

The finding of reduced FRN amplitudes following near-wins compared to losses in Studies C (Chapter Six) and D (Chapter Seven) is consistent with previous electrophysiological research (Luo et al., 2011), suggesting these outcomes are less aversive than ‘full-loss’ outcomes, despite no credits being returned. The lack of a significant Group × Outcome interaction for the loss vs. near-win planned contrast in Study D (Chapter Seven) implies that these individuals did not evaluate these outcomes differently than their non-addicted counterparts. Further research is required to determine whether true LDWs elude between-group differences in the ERP measures examined in this thesis, and whether these outcomes would elicit greater P3b amplitudes than losses and/or an FRP instead of a FRN response.

8.4.3. Conclusions on the significance of almost winning while gambling.

The results of the current thesis have interesting implications for conceptualisations on the role LDWs play in the development and maintenance of normal and problem gambling behaviours. Considered with the findings of previous research (Dixon et al., 2010; Dixon et al., 2013), the results from autonomic Study A (Chapter Four) suggest
that the auditory and visual cues and/or the small monetary returns associated with LDWs play an important role in the development of gambling behaviours in naive gamblers, and initially contribute to the great appeal of this gambling medium; however, the lack of a difference in electrodermal activity following LDWs and losses in Study B (Chapter Five), and the lack of a difference in P3b amplitude following near-wins and losses in Study D (Chapter Seven), suggest that experienced non-problem gamblers and problem gamblers do not evaluate these outcomes as equivalent to wins. The FRN results from Study D extend these findings and imply that, despite these outcomes not being considered as reward, they are appraised as being less punishing than losses by problem and non-problem gamblers.

These results indicate that the presence of LDWs during EGM play may contribute to sustained gambling behaviours through the illusion nearly winning, and to the underestimates of monetary expenditure associated with EGM gambling (Blaszczynski et al., 2008; Productivity Commission, 2010), particularly in individuals with little previous exposure to this form of gambling. Thus, reducing the frequency of occurrence of these outcomes may reduce the appeal and addictive potential of these machines, something that may be of particular benefit to individuals at risk of developing disordered gambling behaviours.

8.5. **Personality Variables as Indicators of Underlying Physiological Processes**
The current thesis examined the relationship between self-reported individual differences in several personality variables and psychophysiological responses to outcomes of varying incentive value across autonomic (Study A/Chapter Four) and cortical ERP (Study D/Chapter Seven) domains. It was of value to examine these relationships for several reasons. The identification of a relationship between self-report
measures and psychophysiological responding means that such personality variables may be used to indicate possible risk of abnormal incentive processing and for developing problem gambling behaviours in vulnerable individuals (see Sections 2.4. and 3.1.). Examination of these variables also allows an examination of whether such factors influence the between-group differences in ERP responses (see Section 7.6.5.).

The results extend previous research findings in several ways. Study A was the first to examine the relationship between impulsive tendencies and autonomic reactivity following outcomes commonly encountered during EGM gambling in a sample of healthy controls. It also extended previous research (e.g., Goudriaan et al., 2006) by examining the relationship between reward and punishment sensitivity and autonomic nervous system reactivity to not only wins and losses, but also near-win outcomes. Study D was the first to examine the relationship between cortical ERP correlates of incentive processing, the FRR and the P3b, and self-reported impulsivity and sensitivity to reward and punishment/non-reward in problem gamblers (Study D), and whether these psychological inventories are able to indicate underlying deficits in incentive processing in these individuals. Moreover, due to the improved accuracy in estimations of psychophysiological responses through application of a PCA, these personality variables were correlated with responses that were not contaminated by overlapping ERP components. The following sections include a summary and discussion of the findings of the investigation of the relationship between implicit psychophysiological responses and explicit self-report levels of impulsivity (Section 8.5.1.) and sensitivity to reward and punishment (Section 8.5.2.) examined in this thesis.
8.5.1. Measures of impulsivity.

The lack of a correlation between Impulsiveness and either autonomic (Study A/Chapter Four) or ERP (Study D/Chapter Seven) correlates of incentive value processing suggests that the tendency towards impulsive behaviours is not related to underlying psychophysiological responses to the experience of reward and punishment/non-reward. Moreover, no between-group differences in trait impulsivity in were found in Study D, which is in contrast to previous research that has reported greater levels of impulsivity in problem gamblers (Nordin & Nylander, 2007; Steel & Blaszczynski, 1998; cf. Allcock & Grace, 1988). The tendency for problem gamblers to report greater levels of impulsivity compared to non-problem gamblers in Study D (although this result was not significant) suggests these results may reflect a power issue in that study and further investigation of this relationship is warranted. It is possible that impulsivity may be more closely implicated in certain subtypes of problem gamblers, such as the antisocial impulsivist subtype (Blaszczynski & Nower, 2002); thus, perhaps due to insufficient power in the analyses of the current body of research, the relationship with psychophysiological measures may have been averaged away with the inclusion of other subtypes of problem gamblers, e.g., behaviourally conditioned and emotionally vulnerable subtypes for whom other individual differences, such as depression, may be associated with the development and maintenance of gambling problems. It would be of value for future research to examine this notion, and to also investigate the objective psychophysiological correlates of inhibitory control and impulsivity (e.g., N200 responses during a Go/NoGo or Stop Signal Task) in problem gamblers, and their relationship with indices of incentive value processing described in this thesis.
8.5.2. Measures of reward and punishment sensitivity.

The results from the analysis between self-reported reward and punishment sensitivity and autonomic responses to gambling outcomes examined in Study A (Chapter Four) revealed that the responsiveness to the anticipation or experience of reward (assessed using the BAS Reward-responsiveness subscale) and the tendency to persistently pursue desired goals (indicated by scores on the BAS Drive subscale) are associated with the electrodermal responses of naive gamblers following win and loss outcomes. Unfortunately, due to constraints on what materials were allowed to be given to club patrons in Study B, this relationship was not able to be examined in problem gamblers while they participated in real gambling activity.

While no between-group differences in reward or punishment sensitivity were found between problem gamblers and healthy controls in Study D (Chapter Seven), problem gamblers who reported a greater severity of symptoms also reported a reduced tendency to avoid punishment stimuli (indicated by higher CPGI and BIS scores, respectively). The investigation of the relationship between self-reported individual differences in reward and punishment sensitivity and cortical indices of incentive value processing revealed that problem gamblers who reported experiencing new rewards as more exciting and being more willing to approach opportunities for reward (as indicated by greater scores on the BAS Fun-seeking subscale) demonstrated greater P3b amplitudes upon experiencing win outcomes. Likewise, problem gamblers who reported higher reward sensitivity (BAS Fun-seeking and Drive subscales) elicited greater P3b amplitudes and lower FRN amplitudes following near-wins. Since problem gamblers showed a tendency to elicit lower P3b amplitudes following all outcome types, lower scores on these BAS subscales may prove to be a useful indicator of abnormal neural processing associated with this disorder, although further research is required to validate
this notion. In contrast to the findings for the problem gambler group, self-reported sensitivity to reward and punishment was not related to the ERP indices of incentive processing for healthy controls in Study D. This finding is also in disaccord with previous research findings (Amodio et al., 2007; Balconi & Crivelli, 2010). The reason for these discrepant findings is unclear, but may be explained by the use of different quantification methods (see Section 7.6.4).

Any conclusions about the relationship between the physiological responses to reward and punishment/non-reward and individual tendencies toward seeking reward and avoiding punishment need to be treated with caution at this stage, given the relatively small sample sizes in the current body of research for this type of analysis. As mentioned above, further research is required to determine whether self-report measures are useful indicators of underlying deficits in psychophysiological responding to incentive value in problem gamblers.

8.6. **Strengths of the Current Research**

The current body of research makes several important contributions to the field of gambling research. A summary of key achievements, in terms of the methodological improvements employed in the analysis and collection of psychophysiological data (Section 8.6.1), and the specific contribution of the research findings for conceptualisations of incentive processing in problem gambling (Section 8.6.2.), is listed below:
8.6.1. **Methodological improvements.**

- Robust and reliable autonomic and electrophysiological indices of incentive processing were identified. These are likely to prove valuable in the further investigation of problem gambling and other addictive disorders.

- Objective psychophysiological indices of incentive value processing were examined rather than subjective self-report measures of reward and punishment sensitivity, which are more susceptible to distortion.

- The application of a PCA to ERP data (Studies C and D) clarified the nature of the latent feedback-related responses (the FRN and the FRP) to valence and magnitude manipulations.

- Examination of the immediate phasic responses to reward and punishment stimuli, rather than the examination of tonic arousal levels associated with gambling activity more generally, allowed the investigation of which theoretical accounts of problem gambling have empirical support.

- Study D (Chapter Seven) was the first to examine the effect of different magnitude rewards in problem gamblers using ERP indices of incentive processing.

- Study B (Chapter Five) was the first to examine the significance LDWs for problem and non-problem gamblers during actual EGM play, and the first to examine the cortical electrophysiological responses of problem gamblers to near-wins.

- A comprehensive examination of the psychophysiological responses during ecologically-valid EGM gambling scenarios outcomes was achieved in this body of work, something that is especially pertinent, considering the potential for harm associated with this gambling medium. Participants gambled with and lost
their own money in Study B, meaning that the responses to punishment, rather than merely non-reward, could be examined, and that the motivational significance of true gambling activity could be explored.

8.6.2. **Contribution of results.**

- Problem gamblers were found to display a hyposensitivity to both reward and punishment outcomes during gambling on EGMs. This pattern of responding could be investigated as a potential biological marker of gambling addiction. The hyposensitive response to reward may explain the tendency for these individuals to place higher bets and to seek larger wins than non-problem gamblers; the hyposensitive response to losses may explain why they continue to gamble despite repeated losses and severe psychosocial, familial, legal, financial, and vocational distress.

- Larger magnitude wins were found to be more motivationally significant than smaller magnitude wins for non-problem gamblers, and the results suggest that problem gamblers tend to evaluate (relatively) large and small magnitude wins as equivalent.

- The small monetary returns and/or the sensory stimuli associated with losses disguised as wins may contribute to the initiation of EGM play in novice gamblers. While these outcomes are not evaluated to be rewarding, they appear to be less punishing than losses, and perhaps contribute to the maintenance of gambling behaviours despite repeated losses.

- Self-report levels of reward sensitivity were found to correlate with psychophysiological responses to win outcomes while gambling, indicating that
individual differences in this personality trait may be a suitable indicator of underlying phasic physiological processes and/or deficits.

8.7. **Limitations and Future Directions**

Due to ethical restrictions, participants did not wager their own money in three of the four studies included in this thesis (i.e., Studies A, C, and D). Gambling with (and losing) allocated credits meant that the losses encountered in the laboratory-based studies may be distorted compared to true gambling activities (van den Berg et al., 2012). Nevertheless, responses to these ‘non-reward losses’ were still found to elicit responses that were significantly different from wins, indicating that participants evaluated these outcomes to be equivalent to actual punishment to some extent.

Similarly, the rewards returned to the gamblers in the laboratory based studies would be a pale comparison to the actual or potential winnings during actual gambling, particularly for problem gamblers in Study D (Chapter Seven). Nevertheless, attenuated responses to reward were found in problem gamblers during real gambling in Study B (Chapter Five), where problem gamblers wagered their own money and the losses encountered were *bona fide* punishers. Moreover, these findings are consistent with neuroimaging research (e.g., de Ruiter et al., 2009; Reuter et al., 2005; Tanabe et al., 2007), indicating that problem gamblers are genuinely hyposensitive to (at least small) rewards and punishment/non-reward. To overcome the potential confound of participants being less concerned about the losses experienced, or undervaluing the rewards encountered in ERP Studies C (Chapter Six) and D (Chapter Seven), event-related potentials would need to be recorded whilst participants gambled in a realistic gambling environment; however, this would be complicated due to the artefact-ridden world of field data collection, and the extreme sensitivity of ERPs to small
environmental changes. Whether the pattern of hyposensitivity to reward and
punishment of problem gamblers extends to larger magnitude wins and losses (and
perhaps, bonus features) remains to be demonstrated and further research should aim to
investigate this possibility in realistic gambling environs.

Although the current body of research provides valuable information on the
nature of deficit in problem gambling, due to its cross-sectional nature, it does not
clarify to what extent inherent biological predispositions (e.g., genetics, heredity factors,
individual differences in personality) and environmental factors (e.g., early experiences
with gambling, gambling reward schedules, cognitive distortions, and sociological and
cultural factors) affect the observed hyposensitive response to reward and punishment stimuli observed in the problem gambler cohort. The inability to determine a direction of causality is a key limitation of this program of research, and is shared by other gambling research (see Abbott & Clarke, 2007).

Some previous research has suggested strong environmental influences on the
development of problem gambling behaviours. The tolerance effects observed in
problem gambling (Blaszczynski, Walker, Sharpe, & Nower, 2008), and the fact that
early positive experiences with gambling are associated with the development of this disorder (Blaszczynski & Nower, 2002; McCowan & Chamberlain, 2000; Sharpe,
2002), suggest that participation in gambling activity contributes to the development and maintenance of problem gambling, even in individuals with no other predisposition toward addictive behaviours. The results from a prospective study by Dickerson et al. (2003) revealed that inherent individual differences (such as a higher propensity toward impulsive behaviours) only account for a quarter of the variance in explaining the lack of control associated with problematic gambling behaviours, and that repeated exposure to regular, high intensity EGM gambling was a better predictor of gambling problems.
Moreover, longitudinal research on substance use disorders, has demonstrated that various neurological, personality, and social attributes commonly associated with these forms of addiction are the result of problematic behaviours rather than being causal factors (Abbott, 1984; Sobell et al., 1996; Vaillant, 1995; Zinberg, 1984).

On the other hand, not everyone who gambles on EGMs develops disordered gambling behaviours (although most gamblers purportedly lose control of their gambling at some stage, Dickerson et al., 2003) suggesting inherent biological vulnerabilities influence the development of problem gambling. Large-scale twin studies have demonstrated that approximately 50-62% of problem gambling symptomology has a genetic basis (Comings, Rosenthal, Lesieur, Rugle, Muhleman, Chiu, Dietz, & Gade, 1996; Eisen et al., 1998; Shah, Eisen, Xian, & Potenza, 2005), and family history has been identified as an important predisposing risk factor for the development of problem gambling (Gambino, Fitzgerald, Shaffer, Renner, & Courtage, 1993; Jacobs, 1988; Lesieur & Rothschild, 1989). As mentioned above (Section 2.1.), several theories posit that reduced functioning in the dopaminergic neurotransmitter system predates the development of addictive behaviours, and this vulnerability is compounded by repeated exposure to gambling, leading to a further reduction of dopamine transmission and functioning (Goldstein & Volkow, 2002; Volkow et al., 2004; Volkow, Fowler, Wang, Baler, & Telang, 2008). Moreover, because of the between-group similarities in physiological responses to reward and punishment stimuli (e.g., Blum et al., 2000; Goldstein et al., 2008; Goldstein et al., 2007; Kamarajan et al., 2010; Porjesz et al., 1987), and other symptomology (Blaszczynski et al., 2008; Leeman & Potenza, 2012; Shaffer & Hall, 2002; Wareham & Potenza, 2010; cf. Abbott et al., 2001) observed in individuals with substance use disorder and problem gambling, it has been posited that these disorders are different manifestations of the same underlying
predisposition toward deficit in the reward circuitry of the brain (Blum et al., 2000), or a common problem behavioural syndrome (e.g., Barnes, Welte, Hoffman, & Dintcheff, 2002; Jessor & Jessor, 1977; Vitaro, Brendgen, Ladouceur, & Tremblay, 2001).

It is likely that the hyposensitive responses observed in the current program of studies is the result of a combination of inherent disposing factors and environmental exposure. Indeed, animal research using drug stimuli has demonstrated that diminished reward sensitivity is both a cause and a consequence of drug ingestion (Nader et al., 2006), although punishment sensitivity has not been examined in this context. It is also possible that the primary determinant of problem gambling symptoms for some individuals are biological or genetic in nature, but for others learning history may better explain the development of this disorder (Blaszczynski & Nower, 2002). Although there have been a number of longitudinal investigations of problem gambling, these have primarily focussed on gambling participation, gambler demographics, personality variables, and gambling-related cognitions (e.g., Abbott, 2001; Dickerson et al., 2003; Schrams, Schellinck, & Walsh, 2000; Shaffer & Hall, 2002; Slutske, Jackson, & Sher, 2003; Vitaro, Wanner, Ladouceur, Brendgen, & Tremblay, 2004; Wardle, Dobbie, Kerr, & Reith, 2009; Wiebe, Cox, & Falkowski-Ham, 2003; Weibe, Single, & Falkowski-Ham, 2003; Winters, Stinchfield, Botzet, & Anderson, 2002). A fruitful direction for future research would be to examine the extent to which the hyposensitive response to reward and punishment/non-reward observed in the current thesis is due to inherent biological or environmental factors. It would also be worthwhile investigating the progression problem gambling over the course of illness (e.g., at the onset of problematic behaviours, the height of problems, remission, and recovery), and potentially, over the course of an individual’s lifetime (e.g., changes throughout childhood, adolescence, adulthood, and later adulthood). Finally, it is commonly
accepted that problem gamblers are a heterogeneous group, with definitive subtypes each associated with different determinants of abnormal gambling behaviour (Blaszczynski & Nower, 2002); thus, the findings in the current study may not generalise to all individuals within the problem gambling population. It would be of value for future research to examine whether and how the psychophysiological correlates of incentive processing and arousal reported in this thesis differ between different subtypes of problem gamblers.

8.8. Conclusion

The novel approaches applied to the collection and analysis of autonomic and ERP data in this thesis allowed an ecologically-valid examination of how problem gamblers, experienced non-problem gamblers, and naive gamblers respond to outcomes commonly encountered during EGM gambling. The examination of the immediate psychophysiological responses that occur during gambling is valuable in advancing the understanding of the motivational significance of reward and punishment/non-reward stimuli for these individuals, and how abnormal responses to such stimuli are implicated in the complex and devastating disorder that is problem gambling. The finding that problem gamblers are not only less sensitive to reward, but also punishment has important theoretical implications for conceptualisations of this disorder, and may explain the aberrant behaviours displayed by individuals afflicted by problematic gambling patterns. Importantly, this pattern of results has the potential to serve as a biological marker for gambling addiction, and may aid in the treatment of this disorder, which will ultimately benefit not only afflicted individuals, but also their families, and the wider community.


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Appendices
Appendix A: Problem Gambling Severity Index (PGSI) Questionnaire

**PGSI**

Some of the next questions may not apply to you, but please try to be as accurate as possible. **THINKING ABOUT THE LAST 12 MONTHS...**

1. Have you bet more than you could really afford to lose? Would you say never, sometimes, most of the time, or almost always (please indicate your responses with a tick)?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

2. Still thinking about the last 12 months, have you needed to gamble with larger amounts of money to get the same feeling of excitement?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

3. When you gambled, did you go back another day to try to win back the money you lost?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

4. Have you borrowed money or sold anything to get money to gamble?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

5. Have you felt that you might have a problem with gambling?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

6. Has gambling caused you any health problems, including stress or anxiety?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

7. Have people criticized your betting or told you that you had a gambling problem, regardless of whether or not you thought it was true?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

8. Has your gambling caused any financial problems for you or your household?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>

9. Have you felt guilty about the way you gamble or what happens when you gamble?

<table>
<thead>
<tr>
<th>Never</th>
<th>Sometimes</th>
<th>Most of the time</th>
<th>Almost always</th>
<th>Don't know</th>
</tr>
</thead>
</table>
Appendix B: Behavioural Inhibition System/Behavioural Activation System Scale

**BIS/BAS Scale**

For each statement below please indicate how much you agree or disagree with what the item says as follows:

1 = very true for me  
2 = somewhat true for me  
3 = somewhat false for me  
4 = very false for me

Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can. Respond to each item as if it were the only item. That is, don’t worry about being "consistent" in your responses.

<table>
<thead>
<tr>
<th>Statement</th>
<th>RESPONSE (1 to 4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. A person’s family is the most important thing in life.</td>
<td></td>
</tr>
<tr>
<td>2. Even if something bad is about to happen to me, I rarely experience</td>
<td></td>
</tr>
<tr>
<td>fear or nervousness.</td>
<td></td>
</tr>
<tr>
<td>3. I go out of my way to get things I want.</td>
<td></td>
</tr>
<tr>
<td>4. When I’m doing well at something I love to keep at it.</td>
<td></td>
</tr>
<tr>
<td>5. I’m always willing to try something new if I think it will be fun.</td>
<td></td>
</tr>
<tr>
<td>6. How I dress is important to me.</td>
<td></td>
</tr>
<tr>
<td>7. When I get something I want, I feel excited and energised.</td>
<td></td>
</tr>
<tr>
<td>8. Criticism or scolding hurts me quite a bit.</td>
<td></td>
</tr>
<tr>
<td>9. When I want something I usually go all-out to get it.</td>
<td></td>
</tr>
<tr>
<td>10. I will often do things for no other reason than that they might be</td>
<td></td>
</tr>
<tr>
<td>fun.</td>
<td></td>
</tr>
<tr>
<td>11. It’s hard for me to find the time to do things such as get a haircut.</td>
<td></td>
</tr>
<tr>
<td>12. If I see a chance to get something I want I move on it right away.</td>
<td></td>
</tr>
<tr>
<td>13. I feel pretty worried or upset when I think or know somebody is</td>
<td></td>
</tr>
<tr>
<td>angry at me.</td>
<td></td>
</tr>
<tr>
<td>14. When I see an opportunity for something I like I get excited right</td>
<td></td>
</tr>
<tr>
<td>away.</td>
<td></td>
</tr>
<tr>
<td>15. I often act on the spur of the moment.</td>
<td></td>
</tr>
<tr>
<td>16. If I think something unpleasant is going to happen I usually get</td>
<td></td>
</tr>
<tr>
<td>pretty &quot;worked up.&quot;</td>
<td></td>
</tr>
<tr>
<td>17. I often wonder why people act the way they do.</td>
<td></td>
</tr>
<tr>
<td>18. When good things happen to me, it affects me strongly.</td>
<td></td>
</tr>
<tr>
<td>19. I feel worried when I think I have done poorly at something</td>
<td></td>
</tr>
<tr>
<td>important.</td>
<td></td>
</tr>
<tr>
<td>20. I crave excitement and new sensations.</td>
<td></td>
</tr>
<tr>
<td>21. When I go after something I use a &quot;no holds barred&quot; approach.</td>
<td></td>
</tr>
<tr>
<td>22. I have very few fears compared to my friends.</td>
<td></td>
</tr>
<tr>
<td>23. It would excite me to win a contest.</td>
<td></td>
</tr>
<tr>
<td>24. I worry about making mistakes.</td>
<td></td>
</tr>
</tbody>
</table>
Appendix C: Impulsiveness Questionnaire (I7)

For each statement below, please answer by putting a circle around the 'YES' or 'NO' option. There are no right or wrong answers, and no trick questions. Work quickly and do not think too long about the exact meaning of the question.

<table>
<thead>
<tr>
<th></th>
<th>Statement</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Do you often buy things on impulse?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Do you generally do and say things without stopping to think?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Do you often get into a jam because you do things without thinking?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Are you an impulsive person?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Do you usually think carefully before doing anything?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Do you often do things on the spur of the moment?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Do you mostly speak before thinking things out?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Do you often get involved in things you later wish you could get out of?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Do you get 'so carried away' by new and exciting ideas, that you never think of possible snags?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>Do you need to use a lot of self-control to keep out of trouble?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>Would you agree that almost everything enjoyable is illegal or immoral?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Are you often surprised at people's reactions to what you do or say?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>Do you think an evening out is more successful if it unplanned or arranged at the last moment?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>Do you usually work quickly, without bothering to check?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>Do you often change your interests?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>Before making up your mind, do you consider all the advantages and disadvantages?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>Do you prefer to 'sleep on it' before making decisions?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>When people shout at you, do you shout back?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>Do you usually make up your mind quickly?</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix D: Abstract (Study A): Australasian Society for Psychophysiology Conference (2009, November), Newcastle, Australia

Heart-rate and electrodermal changes to win and loss events during a computer-simulated gambling task

Lisa Lole¹, Craig Gonsalvez¹, Alex Blaszczynski², Adam Clarke¹, & Renata Hadzic¹

1. Brain and Behaviour Research Institute, School of Psychology, University of Wollongong
2. School of Psychology, University of Sydney

Gambling on electronic gaming machines (EGM) is the most common and most addictive type of gambling within Australia, but there is limited psychophysiological research on the topic. Preliminary research has shown that heart rate (HR) and skin conductance level (SCL) differ for win and loss events while healthy participants gamble on EGMs. However, it remains unclear whether these results are a consequence of the elaborate visual or auditory cues normally associated with these events. We designed a computer task that simulated the EGM experience, and simultaneously recorded HR and SCL while participants ‘gambled’. Different types of win and loss events were tagged by electronic markers, and the differences between these events examined. The results showed a significant difference between wins and losses on SCL, but not HR. No differences were found between high and low bet options. These events were also compared to pre-play tonic HR and SCL. The results suggest that psychophysiological measures are sufficiently sensitive to reliably differentiate between winning and losing in the absence of elaborate auditory or visual cues, and even when participants do not bet with their own money. Future investigation into how problem gamblers respond when they play is warranted and is underway.
Appendix E: Computer Task Used in Study A
Appendix F: Subjective Experience Questionnaire (Studies A and C)

Questionnaire about Poker Machine Experience

1. How pleasant was your experience of playing the poker machine (please circle your answer)?

Not Excited  Moderately Excited  Very Excited
1 2 3 4 5 6 7 8 9

2. Were you bored at any time while you played the poker machine (please circle)?

Not Bored  Moderately Bored  Very Bored
1 2 3 4 5 6 7 8 9

3. Did time feel like it went by quickly while you played the poker machine (please circle)?

Not Quickly  Moderately Quickly  Very Quickly
1 2 3 4 5 6 7 8 9
4. How excited and engrossed did you become at various points while playing the poker machine (please circle)?

i. Before playing the poker machine:

<table>
<thead>
<tr>
<th>Not Excited</th>
<th>Moderately Excited</th>
<th>Very Excited</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

ii. While playing the poker machine:

<table>
<thead>
<tr>
<th>Not Excited</th>
<th>Moderately Excited</th>
<th>Very Excited</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

iii. After playing the poker machine:

<table>
<thead>
<tr>
<th>Not Excited</th>
<th>Moderately Excited</th>
<th>Very Excited</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>
5. How excited did you feel when you experienced the following events while playing the poker machine (please circle)?

   i. A small win:

   Not Excited         Moderately Excited         Very Excited
     1         2         3         4         5         6         7         8         9

   ii. A big win:

   Not Excited         Moderately Excited         Very Excited
     1         2         3         4         5         6         7         8         9

   iii. A small loss:

   Not Excited         Moderately Excited         Very Excited
     1         2         3         4         5         6         7         8         9

   iv. A big loss:

   Not Excited         Moderately Excited         Very Excited
     1         2         3         4         5         6         7         8         9
6. Compared to playing for a movie voucher, please rate how you would feel if you were playing the poker machine with your own money (please circle your answer):

<table>
<thead>
<tr>
<th>Less Stressed</th>
<th>No Change</th>
<th>More Stressed</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>2</td>
<td>3</td>
</tr>
<tr>
<td>4</td>
<td>5</td>
<td>6</td>
</tr>
<tr>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

7. Consider your experience playing the poker machine today. Please tick the option below that best represents how much you agree or disagree with the following statements:

<table>
<thead>
<tr>
<th>Strongly disagree</th>
<th>Moderately disagree</th>
<th>Mildly disagree</th>
<th>Neither agree or disagree</th>
<th>Mildly agree</th>
<th>Moderately agree</th>
<th>Strongly agree</th>
</tr>
</thead>
<tbody>
<tr>
<td>Playing poker machines is exciting.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>It is easy to win on a poker machine.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>It is easy to lose on a poker machine.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Poker machines are fun.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I would play poker machines in the future.</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
8. If you were bored and had nothing to do, for how long could you see yourself playing the pokies (please circle)?

0-30 minutes  30-60 minutes  1-2 hours  2-3 hours  3+ hours

9. If you decided to have a one-off experience on the pokies, just for fun, and decided to set a limit for yourself, please indicate what would this limit would be:

$..........................

10. What were the minimum and maximum credits you accumulated during the experiment (if you are unsure please just provide an estimate):

Minimum: .................................................................

Maximum: .................................................................

11. Please indicate the number of wins and losses you experienced during the experiment (if you are unsure please just provide an estimate):

Number of wins: ..........................................................

Number of losses: ..........................................................

12. Please indicate how many times (as a percentage) you bet 1 and 10 credits [and 20 credits for Study A] (if you are unsure please just provide an estimate):

1 credit:.............% of the time

10 credits:.........% of the time

[20 credits:..............% of the time for Study A]
Appendix G: Participant Information Sheet (Study A)

University of Wollongong

University Population Participant Information Sheet

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

INVESTIGATORS
Researcher:
Lisa Lole
Ph: [Redacted]
Email: lrw968@uow.edu.au

Supervisor:
Associate Professor Craig Gonsalvez
Ph: [Redacted]
Email: craigg@uow.edu.au

Secondary supervisors:
Professor Alex Blaszczynski
Ph: [Redacted]
Email: alexb@psych.usyd.edu.au

Professor Adam Clarke
Ph: [Redacted]
Email: adam_clarke@uow.edu.au

PURPOSE OF THE RESEARCH
The research being conducted is part of PhD project associated with the School of Psychology at the University of Wollongong. Using technology that will allow us to accurately record physiological data, this project aims to compare heart rate and skin conductance response patterns to win and loss events while individuals gamble. Although this project is part of a larger program of research that includes recording data from problem gamblers, the current project will examine gambling in healthy controls.

METHODS AND DEMANDS ON PARTICIPANTS
Participation in this research requires participants’ heart rate and skin conductance level to be recorded while gambling on a computer-simulated task designed to mimic a real-life electronic gaming machine (EGM; otherwise known as a poker machine) for around 30 minutes. We will record physiological responses using an ambulatory monitoring system. This is a non-invasive, small and comfortable portable monitoring system that measures participants’ electrocardiogram (heart activity) and skin conductance. Four
electrodes will be placed on the surface of the skin: two on the fingers and two on the chest. To maintain your privacy, you will place your own chest electrodes. The fitting of recording equipment should take 5-10 minutes to set up.

**None of your own money will be wagered in this experiment, and no money accumulated while gambling will be paid to participants.** During the course of your participation you have opportunity to win movie vouchers, through accumulating credits while playing.

Participants will also be asked to complete a few questionnaires which will take approximately 20-30 minutes to complete; the data from which will remain confidential. The questionnaires will measure participants’ subjective experience of the gambling task, and aspects of personality, depression and anxiety, and past gambling behaviour.

**PRIVACY AND CONFIDENTIALITY**

The results obtained from this study will be used as part of a PhD thesis and the information will also be used in publications and conferences; however, only group means and trends will be published. Participation in this research does not require your name to be collected. Participants’ physiological and questionnaire data will be matched using a code rather than their name to ensure that all data collected remains confidential.

**POSSIBLE RISKS, INCONVENIENCES, AND DISCOMFORTS**

Participation in this study involves filling out several questionnaires, set up of recording equipment, and playing a computer-simulated gambling task while physiological responses are recorded using an ambulatory monitoring system (which in total, will require no more than one and a half hours of your time). Participants will be awarded one and a half credit points towards an elected subject. All tasks are designed not to cause any harm or distress to the participant. If during the course of active participation you feel uncomfortable, you are free to withdraw from the study. After participation in this study your data will not be able to be withdrawn. Your relationship with UoW and the School of Psychology will not be affected in any way if you withdraw from this study. **This study does involve gambling, so if you have a problem with gambling, do not participate in this project without consulting a counsellor.**
Please feel free to ask the researcher any questions or concerns you may have, alternatively you may contact Associate Professor Craig Gonsalvez or Lisa Lole.

ETHICS REVIEW AND COMPLAINTS

This study has been reviewed by the Human Research Ethics Committee (Social Science, Humanities and Behavioural Science) of the University of Wollongong. If you have any concerns, questions, or complaints regarding the way this research has been conducted, you can contact the UoW Complaints Officer on (02) 42214457. If gambling is a problem for you or anyone you know, contact the G-Line (1800633635), a 24hr, 7 day a week counselling line for problem gamblers and their families.

Thank you for your interest in this study. If you would like to participate, please complete the attached consent form and return it to the experimenter.
Appendix H: Participant Consent Form (Study A)

University of Wollongong

University Population Participant Consent Form

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

Researcher: Lisa Lole

Supervisors: Associate Professor Craig Gonsalvez, Associate Professor Adam Clarke, & Professor Alex Blaszczynski

I have read, understood, and discussed the information sheet for the study “Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?” I understand that this study is being conducted as part a PhD project associated with the School of Psychology at the University of Wollongong, and is being conducted by Lisa Lole and/or her supervisor, Associate Professor Craig Gonsalvez.

I have been advised of the potential risks and burdens associated with this research. I understand that if I consent to participate in this study, I will be required to fill in personality and other questionnaires relating to gambling, and will have my physiological data recorded. I understand that I will be required to wear four electrodes (two on my chest and two on my fingers) in order for physiological data to be recorded. I understand that I can use the restroom to ensure privacy whilst I position the chest electrodes for myself. I am aware that I will not wager my own money and that any credits won while playing the computer-simulated gambling task will not be paid to myself, although I may be eligible to win one or two movie vouchers depending on the outcome of the task.

I am aware that any data collected from me will remain confidential. I have had the opportunity to ask Lisa Lole and/or her supervisor, Associate Professor Craig Gonsalvez, any questions I may have about the research and my participation. I understand that my participation in this research is voluntary, I am free to refuse to participate, and I am free to withdraw from the study at any time before my data is submitted to the researcher, as I will not be able to do so afterwards. My refusal to
participate or withdraw my consent will not affect my relationship with the School of Psychology at the University of Wollongong in my course/program of study.

I understand that the data collected from my participation will be used primarily for research purposes. I understand that the results may be published in academic journals and/or presented at conferences, and I consent for it to be used in that manner. I understand that my individual data will not be published and that only group means and trends will be used.

If I have any enquires about the research, I can contact Lisa Lole (ph. [redacted]) or Associate Professor Craig Gonsalvez ([redacted]). If I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Ethics Officer, Human Research Ethics Committee, University of Wollongong on 42214457.

By signing below, I am indicating my consent to participate in the research described to me in the information sheet.

Signed

Date

........................................... ...........................................

Name (please print)

...........................................
Appendix I: Abstract (Study B): Australasian Society for Psychophysiology Conference (2012, November), Sydney, Australia

Problem gamblers are hyposensitive to wins: An analysis of skin conductance responses during live gambling

Lisa Lole, Craig J. Gonsalvez, & Robert J. Barry
Centre for Psychophysics, Psychophysiology, and Psychopharmacology; Brain & Behaviour Research Institute; and School of Psychology, University of Wollongong, Wollongong 2522, Australia

Aims: Physiological arousal is purportedly a key determinant in the development and maintenance of gambling behaviours, with problem gambling being conceptualised in terms of an autonomic response deficit. Specifically, it has been suggested that problem gamblers may be hyposensitive to losses and/or hypersensitive to wins however, previous research examining phasic electrodermal responses of these individuals has been limited to laboratory settings. To test the nature of deficit in this disorder, reactions to real gaming situations, where gamblers wager their own money, needs to be examined.

Methods: Skin conductance data to losses, wins and, fake-wins (outcomes where the amount returned is less than that wagered) were recorded in real-time from ten problem and ten non-problem gamblers while they played an electronic gaming machine (EGM). Participants wagered their own money on a gaming machine of their choice within a real gambling venue.

Results: While win outcomes elicited a skin conductance response (SCR) in the non-problem gambler group, problem gamblers demonstrated significantly reduced SCRs.
Losses and fake-win outcomes did not appear to differ between problem and non-problem gambler groups.

**Discussion:** The current study allowed an examination of autonomic arousal in problem and non-problem gamblers in an ecologically-valid setting. The results suggest that, rather than being hypersensitive to reward as previous theory predicted, problem gamblers are less reactive to win outcomes on an electronic gaming machine than are non-problem gamblers. This hyposensitivity to positive outcomes implies a malfunction in incentive value processing is implicated in problematic gambling behaviours, and presents as a potential marker for addiction.
Appendix J: Abstract (Study B): Society for Psychophysiological Research Conference (2011, September), Boston, U.S.A.

Electrodermal activity to win events during gambling differentiates problem and non-problem gamblers: A Field study.

Craig J. Gonsalvez¹, Lisa Lole¹, Amrit Grewal¹, & Alex Blaszczynski²

1. University of Wollongong; 2. University of Sydney

Current psychological conceptualizations posit that physiological arousal is a key determinant in the development and maintenance of gambling behaviors. Specifically, it has been suggested that problem gamblers may have a hypersensitivity to rewards and/or a hyposensitivity to losses. Although considerable research has been conducted within laboratories, these predictions have not been examined systematically in real gambling situations. The current study reports the results from two experiments conducted in clubs where patrons gambled on electronic gaming (slot) machines (EGM). EGMs administer a series of rapid bet-outcome trials (3-6 seconds) with each trial resulting in a loss, win, or feature (signalling a series of wins). In Experiment 1, electrodermal activity to losses, wins, and features was recorded in a sample of non-problem gamblers (n = 22) during EGM play. Features produced large SCRs whereas losses did not. In Experiment 2, electrodermal activity was recorded from both problem (n = 21) and non-problem gamblers (PG; n = 21). For non-PGs, large SCRs to wins but not to losses were observed. Unlike non-PGs, PGs manifested a blunted psychophysiological profile, with no evidence of significant changes to any of the three events. Thus, these results indicate support for an autonomic hyposensitivity to win events, not the reverse, for PGs.
Appendix K: In-club Research Participation Advertisement (Poster)

University of Wollongong

ARE YOU GOING TO PLAY THE POKIES TODAY?

IF SO...
We would like to invite you to be a part of a ground breaking study conducted by the University of Wollongong

PARTICIPANTS WILL RECEIVE A FOOD VOUCHER FOR THE BISTRO IN APPRECIATION OF THEIR TIME.

If you are interested in participating please see someone at the service desk for more information, or contact Lisa on ph: [redacted] or email: lrw968@uow.edu.au

Your assistance to help out with this research will be greatly appreciated!!

bbri
Brain & Behaviour Research Institute
Appendix L: Participant Information Sheet (Study B)

University of Wollongong

Participant Information Sheet

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

Researchers: Lisa Lole (Ph: 4221 4513); Craig Gonsalvez (Ph: 4221 3674).

PURPOSE OF RESEARCH

This study aims to track and measure the “buzz” or “suspense” levels (called arousal mechanisms in psychology, measured by looking at changes in heart rate and skin conductance responses [i.e., how much we sweat on the palms of our hands]) that the body experiences in response to wins and losses during play on the pokies. This study also examines whether these reactions are different for persons who gamble excessively and those who don’t. In doing so, we hope to develop a reliable physiological test that will allow the early identification of persons at risk for gambling-related problems.

What exactly does participation involve and what will be required of me?

This study will involve a researcher providing you with a heart rate and skin conductance monitoring device (the size of a large mobile phone) to wear whilst you play an electronic gaming machine of your choice:

- You will be asked to wear five electrodes (two placed on your fingers and three placed on your chest) whilst you go about playing on the poker machines in any way that you choose. The electrodes will help monitor your heart rate and skin conductance. It will take no more than 10 minutes to fix the electrodes, after which you will be required to wear this equipment for up to 30 minutes (or longer if you choose). You can unhook yourself from the device at any time when you choose to do so. Your gambling will not be interrupted in any way.

- During the above experiment time, the researcher will be sitting next to or behind you (whichever you choose) and will press buttons on a small remote control device (like a car key) each time you win or lose on the machine. This will allow them to
study differences between your body’s responses to wins and losses while you are gambling.

- Finally, you will be asked to fill out a couple of short questionnaires about your previous gambling activity, which should take you around five to ten minutes to complete.

- It is expected that your participation in this task will not entail a commitment of greater than 30 minutes of your time (this time may be less or more depending on how long you allow the researcher to record your responses while you play on the gaming machine).

Agreement to participate should in no way oblige you to gamble differently from what you were already planning. You are free to stop gambling at any time or to stop the researcher recording while you gamble. We would like your data to remain confidential, so you will not be required to reveal your name. In order to match your questionnaire and physiological data, a code will be used.

POSSIBLE RISKS, INCONVENIENCES, AND DISCOMFORTS

The task is designed not to cause you any harm or distress. If during the course of active participation you feel uncomfortable or embarrassed, you are free to withdraw from the study. After participation in this study your data will not be able to be withdrawn as it is anonymous. In appreciation for your time and participation you will receive a $40 voucher for the Insert Club Name bistro.

The University of Wollongong is independent of the club/casino management, and does not influence gambling outcomes. In other words, we cannot change wins/losses on the gambling machines in any way. The researchers, the University of Wollongong and/or Insert Club Name WILL NOT be responsible for, and will NOT provide compensation for any losses you incur during the course of your gambling.

ETHICS REVIEW AND COMPLAINTS

If you have any concerns or complaints regarding the way the research is being, or has been conducted, you can contact the Complaints Officer, Human Research Ethics Committee, Research Services Office, University of Wollongong on 4221 4457. If you feel you have a problem with gambling, or you feel you need to talk to a trained
counsellor please call the G-Line (NSW), a telephone counselling service for problem gamblers and their families, on 1800 633 635. Referrals for face-to-face services can also be made with G-Line’s assistance.

Please feel free to ask the researcher any questions or concerns you may have, alternatively contact Associate Professor Craig Gonsalvez or Lisa Lole. Thank you for your consideration to participate. Please indicate to the researcher if you would like to participate in the study.
Appendix M: Participant Consent Form (Study B)

University of Wollongong

Participant Consent Form

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

Researchers: Lisa Lole (Ph: 4221 4513); Craig Gonsalvez (Ph: 4221 3674).

I have been given information about the research project entitled “Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?” I have had the opportunity to discuss the project with Lisa Lole, who is conducting this research as part of a PhD study supervised by Associate Professor Craig Gonsalvez in the School of Psychology, Faculty of Health and Behavioural Sciences, University of Wollongong.

I understand that, if I consent to participate in this project, I will be asked to wear five small electrodes, and equipment (about the size of a large mobile phone) used to monitor my physiological responses (including heart rate and skin conductance level) for up to 30 minutes (or for longer if I choose) whilst I play on electronic gaming machines that I was already intending to use. I also understand that I will be asked to fill in a couple of questionnaires on previous gambling activity which will take around 5-10 minutes to complete. I understand that all data collected from me will remain confidential, and only a code will be used to match my physiological and questionnaire data. I also understand that I will receive a $40 voucher for the Insert Club Name bistro in appreciation of my time.

I have been advised of the potential risks and burdens associated with this research, which include the inconvenience of wearing 5 electrodes (3 to be placed on my chest by myself, and 2 to be placed on my fingers by the researcher) and have had an opportunity to ask the researcher, Lisa Lole, any questions I may have about the research and my participation.

I understand that my participation in this research is voluntary. I am free to refuse to participate and I am free to withdraw from the research at any time.
during the recording (I also understand that I will not be able to withdraw my data after the time of recording, as the data is anonymous). I understand that the researchers, the University of Wollongong, and/or Insert Club Name will not be responsible for any monetary losses incurred during the course of my gambling or participation in this study.

I understand that if I have any enquiries about the research, I can contact Lisa Lole (ph: [redacted] or email: lrw968@uow.edu.au) and/or Craig Gonsalvez (Ph: [redacted]), or if I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Ethics Officer, Human Research Ethics Committee, Research Services Office, University of Wollongong on 4221 4457.

By signing below, I am indicating my consent to participate in the research entitled “Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?” conducted by Lisa Lole as it has been described to me in the information sheet and in discussion with her. I understand that the data collected from my participation will be used for her thesis, journal publications, and conferences, and I consent for it to be used in that manner.

Signed Date

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Name (please print)

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Appendix N: Abstract (Study C): Australasian Society for Psychophysiology Conference (2012, November), Sydney, Australia

Incentive processing during a gambling task: A principal components analysis

Lisa Lole, Craig J. Gonsalvez, Robert J. Barry, & Frances De Blasio

Brain & Behaviour Research Institute, School of Psychology, University of Wollongong

Aims: Over the past two decades, the feedback-related negativity (FRN) event-related potential (ERP) component has been shown to be a robust neural correlate of reward processing, in that it consistently differentiates unfavourable outcomes from beneficial ones. This component is of particular significance in the investigation of the role aversive outcomes play in the development and maintenance of gambling behaviours.

Methods: A spatial-temporal principal components analysis (PCA) using Varimax rotations was employed in order to clarify the latent neural correlates involved in incentive-value processing. Eighteen healthy participants played a computer electronic gaming machine (EGM) task that presented feedback regarding the success of each trial, and the elicited brain activity was recorded. The distribution of reward/non-reward outcomes closely matched that of a real gaming machine, with frequently presented losses and infrequently presented wins, as well as ‘losses-disguised-as-wins’ (near-win) events.

Results: The componential analysis revealed the FRN to be characterised by a negative deflection to losses, but a positive deflection to wins, at frontal sites (Fz). As expected, the error positivity and slow wave ERP components also differed according to outcome valence. Interestingly, responses to near-win outcomes were more similar to wins than losses.
**Conclusions:** These results suggest that the neural generators of the FRN and thus, those responsible for incentive-value processing are differentially activated following reward and non-reward/punishment feedback. The finding that near-wins are perceived as more favourable than losses, suggests they constitute a design feature of EGMs that maintains the attention of gamblers. These results are integrated to form an account of the spatial and temporal characteristics associated with normal incentive-value processing during an ecologically-valid gambling task.
Appendix O: Abstract (Study C): Society for Psychophysiological Research Conference (2011, September), Boston, U.S.A.

ERP effects of Wins, Near-Wins and Losses During a Computer-Simulated Gaming Task

Lisa Lole, Craig J. Gonsalvez, & Adam R. Clarke.

Brain & Behaviour Research Institute, School of Psychology, University of Wollongong

Recent research has indicated that the feedback negativity (sometimes called the error-related negativity or ERN) is sensitive to loss/win manipulations in addition to incorrect/correct manipulations. These new findings are salient because of important theoretical and clinical implications for our understanding of reward mechanisms in general, and the effect of reinforcement contingencies on ERPs within gambling tasks in particular. The current study examined the effects on feedback negativity and P3 in response to win, near-win and loss outcomes while healthy controls ($n = 19$) played a computer-simulated gambling task, designed to mimic an electronic gaming machine. Participants were able to choose from two bet sizes: small and large. As predicted, feedback negativity was sensitive to the valence of the outcome, with losses and Near-wins producing larger components than wins. No significant difference was found between losses and near-wins. Further, wins produced significantly larger P3 amplitudes compared to losses and near-wins. In contrast to the pattern of findings for feedback negativity, P3 amplitudes differentiated near-wins (larger amplitudes) from losses. All events, but particularly wins, were found to produce larger P3 magnitudes at Pz compared to lateral parietal sites. No significant effect of bet size on feedback negativity or P3 was found. Further investigation into ERP differences between problem and non-problem gamblers across win and loss outcomes is warranted.
Appendix P: Participant Information Sheet (Study C)

University of Wollongong

Participant Information Sheet

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

INVESTIGATORS

Researcher:  
Ms Lisa Lole 
Ph: [redacted]  
Email: lrw968@uow.edu.au

Supervisor:  
Associate Professor Craig Gonsalvez 
Ph: [redacted]  
Email: craig@craig.edu.au

Secondary supervisors:  
Professor Alex Blaszczynski 
Ph: [redacted]  
Email: alexb@psych.usyd.edu.au

Professor Adam Clarke 
Ph: [redacted]  
Email: adam.clarke@uow.edu.au

PURPOSE OF THE RESEARCH

The research being conducted is part of PhD project. Using technology that will allow us to accurately record physiological data, this project aims to compare electroencephalogram (EEG), heart rate, and skin conductance response patterns to win and loss events while individuals gamble. Although this project is part of a larger program of research that includes recording data from problem gamblers, the current project will examine gambling in healthy controls.

METHODS AND DEMANDS ON PARTICIPANTS

Participation in this research requires participants’ EEG, heart rate, and skin conductance to be recorded while gambling on a computer-simulated task designed to mimic a real-life electronic gaming machine (EGM; otherwise known as a poker machine) for around 30 minutes. We will record EEG responses using an electrode cap (which contains 19 cortical electrodes, four eye electrodes, and 2 ear electrodes). The
EEG cap will be used to measure the brain’s electrical activity in response to the gambling task. Heart rate and skin conductance responses will be measured using an ambulatory monitoring system. This is a non-invasive, small, and comfortable portable monitoring system that measures electrocardiogram (heart activity) and skin conductance that requires four electrodes to be placed on the surface of the skin; two on the fingers and two on the chest. To maintain your privacy you will place your own chest electrodes. **None of your own money will be wagered in this experiment, and no money accumulated while gambling will be paid to participants.** During the course of your participation you have opportunity to win movie vouchers, through accumulating credits while playing. Participants will also be asked to complete a few questionnaires; the data from which will remain confidential. The questionnaires will measure participants’ subjective experience of the gambling task, and aspects of personality, depression, anxiety, and past gambling behaviour.

**PRIVACY AND CONFIDENTIALITY**

The results obtained from this study will be used as part of a PhD thesis and the information will also be used in publications and conferences. However, only group means and trends will be published. Participation in this research does not require your name to be collected. Participants’ physiological and questionnaire data will be matched using a code instead of their names to ensure that all data collected remains confidential.

**POSSIBLE RISKS, INCONVENIENCES, AND DISCOMFORTS**

Participation in this study should take no longer than two and a half hours, in which time you will be required to fill out several questionnaires and play the computer task while your physiological responses are recorded. For your participation you will be awarded two and a half credit points towards your elected subject. All tasks are designed not to cause any harm or distress to the participant. If during the course of active participation you feel uncomfortable, you are free to withdraw from the study. After participation in this study your data will not be able to be withdrawn. Your relationship with UoW and the school of psychology will not be affected in any way if you withdraw from this study. **This study does involve gambling, so if you have a problem with gambling, do not participate in this project without consulting a counsellor.**
Please feel free to ask the researcher any questions or concerns you may have, alternatively you may contact Associate Professor Craig Gonsalvez or Lisa Lole.

ETHICS REVIEW AND COMPLAINTS

This study has been reviewed by the Human Research Ethics Committee (Social Science, Humanities and Behavioural Science) of the University of Wollongong. If you have any concerns, questions, or complaints regarding the way this research has been conducted, you can contact the UoW Complaints Officer on (02) 42214457. If gambling is a problem for you or anyone you know contact the G-Line (1800633635) a 24hr, 7 day a week counselling line for problem gamblers and their families.

Thank you for your interest in this study. If you would like to participate, please complete the attached consent form and return it to the experimenter.
Appendix Q: Participant Consent Form (Study C)

University of Wollongong

University Population Participant Consent Form

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

Researcher: Lisa Lole

Supervisor: Associate Professor Craig Gonsalvez

I have read, understood, and discussed the information sheet for the study “Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?” I understand that this study is being conducted as part a PhD project associated with the School of Psychology at the University of Wollongong by Lisa Lole and her supervisor, Associate Professor Craig Gonsalvez.

I have been advised of the potential risks and burdens associated with this research. I understand if I consent to participate in this study that I will be required to have my physiological data recorded while I play a computer-simulated gambling task for around 30 minutes. I understand that I will be required to be fitted with an electrode cap containing 19 cortical, 4 eye and 2 ear electrodes in order for my EEG activity to be recorded. I understand that I will also be required to wear four electrodes (two on my chest and two on my fingers) in order for my heart rate and skin conductance to be recorded. I understand that I can use the restroom to ensure privacy whilst I position the chest electrodes for myself. I am aware that I will not wager my own money and that any credits won while playing the computer-simulated gambling task will not be paid to myself, although I may be eligible to win one or two movie vouchers depending on the outcome of the task. I also understand that I will be asked to complete a few questionnaires on previous gambling activity that will take approximately 10-20 minutes. In total, participation in this study should require no longer than two and a half hours of my time.

I am aware that any data collected from me will remain confidential and that only a code, and not my name or any personal information, will be used to match my questionnaire and physiological data. I have had the opportunity to ask Lisa Lole and/or her supervisor, Associate Professor Craig Gonsalvez, any questions I may have about
the research and my participation. I understand that my participation in this research is voluntary, I am free to refuse to participate, and I am free to withdraw from the study at any time before my data is submitted to the researcher. My refusal to participate or withdraw my consent will not affect my relationship with the School of psychology at the University of Wollongong in my course/program of study.

I understand that the data collected from my participation will be used primarily for research purposes. I understand that the results may be published in academic journals and/or presented at conferences, and I consent for it to be used in that manner. I understand that my individual data will not be published and that only group means and trends will be used.

If I have any enquiries about the research, I can contact Lisa Lole (ph. [number]) or Associate Professor Craig Gonsalvez ([number]). If I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Ethics Officer, Human Research Ethics Committee, University of Wollongong on 42214457.

By signing below I am indicating my consent to participate in the research described to me in the information sheet.

Signed
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Date
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Name (please print)

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Appendix R: Abstract (Study D): Australasian Cognitive Neurosciences Conference (2011, December), Sydney, Australia

Problem Gamblers Are Less Sensitive To Losses in a Gambling Task: An ERP Study

Lisa Lole & Craig Gonsalvez

Brain & Behaviour Research Institute, School of Psychology, University of Wollongong

It is well established that amplitudes of the feedback-related negativity (FRN) ERP component are greater following negative compared to positive outcomes. This sensitivity of the FRN to outcomes along the reward/non-reward continuum has major implications for gambling research. The reinforcement learning theory posits that FRN amplitudes reflect changes in the mesencephalic dopamine system, with higher dopamine levels associated with larger FRN amplitudes. Psychological theories of gambling have proposed that problem gamblers (PGs) may be hypersensitive to rewards and/or hyposensitive to losses. However, differences in reward and non-reward processing between PG and non-PGs have rarely been examined. The link between FRN amplitudes and negative outcomes provides an opportunistic means to test such a prediction, namely that compared to non-PGs, PGs will evidence smaller FRN amplitudes to losses in a gambling task. The current study investigated the impact of win and loss events on the FRN while 12 non-PGs and 12 PGs played a computer-simulated gambling task. As predicted, compared to non-PGs, PGs exhibited smaller FRN amplitudes following loss outcomes. These results are consistent with the notion that PGs are hyposensitive to losses, and that this processing deficit may contribute to the development and/or the maintenance of problematic gambling behaviours.
Appendix S: Participant Information Sheet (Study D)

Community Population Participant Information Sheet

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

Researchers: Lisa Lole (Ph: 4221 4513); Craig Gonsalvez (Ph: 4221 3674).

PURPOSE OF RESEARCH

This study aims to track and measure the “buzz” or “suspense” levels that the body experiences in response to wins and losses during play on the pokies. This study also examines whether these reactions are different for persons who have gambling problems and those who don’t. In doing so, we hope to develop a reliable physiological test that will allow the early identification of persons at risk for gambling related problems.

What exactly does participation involve and what will be required of me?

You will be required to play a poker machine task on a computer in one of the laboratories in the School of Psychology, University of Wollongong, while your EEG (i.e., your brain’s electrical activity) is monitored. The outcomes (i.e., wins and losses) on this task are random and have been set up to yield a 95% payout ratio to players. Participation in the task will involve:

- Being fitted with an EEG cap consisting of nineteen electrodes which measure your brain’s electrical activity. Two electrodes will be fitted on your ears and four electrodes, which measure your eye and facial muscle activity on the area, around your eye (approximately 2-3cm way from your eyeball).
- Fitting of the EEG cap and eye electrodes will take approximately 20-30 minutes and you will be asked to wear this equipment for up to one hour, including around 30 minutes whilst you play the computer task.
• Once you have completed the computer task you will be asked to complete a few questionnaires on previous gambling activity which will take around 5-10 minutes to complete.
• It is expected that your participation will not entail a commitment of greater than two hours of your time.

Your brain’s responses while you play the poker machine task will be recorded in order to see the effects of gambling on EEG activity.

None of your own money will be wagered in this experiment, and no money accumulated while gambling will be paid to participants. You will be given 5000 free credits (worth $50.00) at the beginning of the task. Credits won on the task WILL NOT be paid to you, and credits lost will not be charged to you. You will win 1 entertainment/food voucher if, by the end of a specified number of trials, you accumulate 6000-6999 credits; and 2 vouchers if you accumulate 7000 or more credits.

In order to maintain your anonymity and the confidentiality of your responses, only a code, and not your name or any personal information, will be used to match your questionnaire and physiological data.

Will I be compensated for my time and travel costs?

You will be reimbursed for travel expenses with a $30 Coles Myer voucher. In appreciation of your participation (which will take approximately two hours of your time), and slight discomfort whilst wearing the EEG electrode cap, you will receive an entertainment/food voucher.

POSSIBLE RISKS, INCONVENIENCES, AND DISCOMFORTS

This task is designed not to cause any harm or distress. The researchers have given your counsellor information about the task, and they have agreed to discuss your participation in the task with you. If you wish to participate they will be required to sign the participant consent form, in order to acknowledge it is appropriate for you to do so. If during the course of active participation you feel uncomfortable you are free to withdraw from the study. After participation in this study your data will not be able to be withdrawn as the data is anonymous. This study does involve gambling therefore please consider if this project is appropriate for you. Please feel free to ask the
researcher any questions or concerns you may have, alternatively contact Associate Professor Craig Gonsalvez or Lisa Lole.

ETHICS REVIEW AND COMPLAINTS

If you have any concerns or complaints regarding the way the research is or has been conducted, you can contact the Complaints Officer, Human Research Ethics Committee, Research Services Office, University of Wollongong on 4221 4457. G-Line (NSW) is a telephone counselling service for problem gamblers and their families. If you feel you need to talk to trained counsellors please call 1800 633 635. Referrals for face-to-face services can also be made with G-Line’s assistance.

Thank you for your consideration. If you wish to participate in this study you can make an appointment for a testing time with the researcher now, or please feel free to call Lisa Lole on [redacted], or email her on lrw968@uow.edu.au.
Appendix T: Participant Consent Form (Study D)

University of Wollongong

Community Population Consent Form

“Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”

Researchers: Lisa Lole (Ph: 4221 4513); Craig Gonsalvez (Ph: 4221 3674).

I have been given information about the research project entitled “Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?” I have had the opportunity to discuss the research project with Lisa Lole who is conducting this research as part of a PhD project supervised by Associate Professor Craig Gonsalvez in the School of Psychology within the Faculty of Health and Behavioural Sciences, University of Wollongong.

I understand that, if I consent to participate in this project, I will be asked to wear an electrode cap used to monitor brain activity, 4 small electrodes to be placed on the area around my eye, and 2 ear electrodes while I play a computer-simulated gambling task for approximately 30 minutes.

I also understand that I will be asked to complete a few questionnaires on previous gambling activity that will take approximately 5-10 minutes, but this data will not have my name linked to it, so my identity will not be known. In total, participation in this study should require no longer than two hours of my time.

I have been advised of the potential risks and burdens associated with this research, which include the inconvenience of wearing an electrode cap, 4 electrodes around my eyes, and 2 ear electrodes, and I have had an opportunity to ask Lisa Lole any questions I may have about the research and my participation. I understand that none of my own money will be wagered in this experiment, and no credits accumulated while gambling will be paid to me. However, I understand that I will be reimbursed for my travel expenses with a Coles Myer voucher (valued at $30) and that I will receive one movie voucher for my time. I understand that I may receive up to two additional movie vouchers depending on the outcome of the experiment.
I understand that in order to maintain my anonymity, only a code, and not my name or any personal information, will be used to match my questionnaire and physiological data. I understand that my participation in this research is voluntary. I am free to refuse to participate and I am free to withdraw from the research at any time up until the time of publication or thesis submission.

If I have any enquiries about the research, I can contact Lisa Lole (Ph: [redacted]) and/or Craig Gonsalvez (Ph: [redacted]). If I have any concerns or complaints regarding the way the research is or has been conducted, I can contact the Ethics Officer, Human Research Ethics Committee, Research Services Office, University of Wollongong on 4221 4457.

By signing below, I am indicating my consent to participate in the research entitled “Problem Gambling: Can subtle physiological reactions to wins and losses help identify the problem gambler?”, conducted by Lisa Lole as it has been described to me in the information sheet and in discussion with her. I understand that the data collected from my participation will be used for her thesis, journal publications, and conferences, and I consent for it to be used in that manner.

Signed                                      Date

..................................................................................................  ....../....../.....

Name (please print)

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