Processing of Near Outcomes and Outcome Sequences in Gambling: Implications for the Biopsychological Basis of Problem Gambling

Verarbeitung von knappen Ergebnissen und Ergebnissequenzen im Glücksspiel: Implikationen für die biopsychologische Basis von problematischem Glücksspielverhalten

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Abbreviations

ACC	Anterior Cingulate Cortex
AMS	Achievement Motives Scale
ANOVA	Analysis of Variance
BA	Brodman Area
BIGL	Belief in Good Luck Questionnaire
DOSPERT	Domain-Specific Risk-Taking Scale
ECG	Electrocardiography
EDA	Electrodermal Activity
EEG	Electroencephalography
EMG	Electromyography
ERN	Error-Related Negativity
ERP	Event-Related Potential
FDR	False Discovery Rate
fMRI	Functional Magnetic Resonance Imaging
FRN	Feedback-Related Negativity
GRCS	Gambling Related Cognitions Scale
IBI	Interbeat Interval
ICA	Independent Component Analysis
ITI	Intertrial Interval
KFG	Kurzfragebogen zum Glücksspielverhalten
LC-NE system	Locus Coeruleus-Norepinephrine System
lPFC	Lateral Prefrontal Cortex
M	Mean
Max	Maximum
MEG	Magnetencephalography
Min	Minimum
Mini-DIPS	Diagnostisches Kurz-Interview bei psychischen Störungen
MRI	Magnetic Resonance Imaging
SCID-PG	Structured Clinical Interview for DSM-Disorders Pathological Gambling
SCL	Skin Conductance Level
SCR	Skin Conductance Response
SD	Standard Deviation
SEM	Standard Error of the Mean
SOGS	South Oaks Gambling Screen
TPJ	Temporoparietal Junction
UPPS	UPPS Impulsive Behavior Scale

Abstract

Gambling is a popular activity in Germany, with 40% of a representative sample reporting having gambled at least once in the past year (Bundeszentrale für gesundheitliche Aufklärung, 2014). While the majority of gamblers show harmless gambling behavior, a subset develops serious problems due to their gambling, affecting their psychological well-being, social life and work. According to recent estimates, up to 0.8% of the German population are affected by such pathological gambling. People in general and pathological gamblers in particular show several cognitive distortions, that is, misconceptions about the chances of winning and skill involvement, in gambling. The current work aimed at elucidating the biopsychological basis of two such kinds of cognitive distortions, the illusion of control and the gambler's and hot hand fallacies, and their modulation by gambling problems. Therefore, four studies were conducted assessing the processing of near outcomes (used as a proxy for the illusion of control) and outcome sequences (used as a proxy for the gambler's and hot hand fallacies) in samples of varying degrees of gambling problems, using a multimethod approach.

The first study analyzed the processing and evaluation of near outcomes as well as choice behavior in a wheel of fortune paradigm using electroencephalography (EEG). To assess the influence of gambling problems, a group of problem gamblers was compared to a group of controls. The results showed that there were no differences in the processing of near outcomes between the two groups. Near compared to full outcomes elicited smaller P300 amplitudes. Furthermore, at a trend level, the choice behavior of participants showed signs of a pattern opposite to the gambler's fallacy, with longer runs of an outcome color leading to increased probabilities of choosing this color again on the subsequent trial. Finally, problem gamblers showed smaller feedback-related negativity (FRN) amplitudes relative to controls.

The second study also targeted the processing of near outcomes in a wheel of fortune paradigm, this time using functional magnetic resonance imaging and a group of participants with varying degrees of gambling problems. The results showed increased activity in the bilateral superior parietal cortex following near compared to full outcomes.

The third study examined the peripheral physiology reactions to near outcomes in the wheel of fortune. Heart period and skin conductance were measured while participants with varying degrees of gambling problems played on the wheel of fortune. Near compared to full outcomes led to increased heart period duration shortly after the outcome. Furthermore, heart period reactions and skin conductance responses (SCRs) were modulated by gambling problems. Participants with

high relative to low levels of gambling problems showed increased SCRs to near outcomes and similar heart period reactions to near outcomes and full wins.

The fourth study analyzed choice behavior and sequence effects in the processing of outcomes in a coin toss paradigm using EEG in a group of problem gamblers and controls. Again, problem gamblers showed generally smaller FRN amplitudes compared to controls. There were no differences between groups in the processing of outcome sequences. The break of an outcome streak led to increased power in the theta frequency band. Furthermore, the P300 amplitude was increased after a sequence of previous wins. Finally, problem gamblers compared to controls showed a trend of switching the outcome symbol relative to the previous outcome symbol more often.

In sum, the results point towards differences in the processing of near compared to full outcomes in brain areas and measures implicated in attentional and salience processes. The processing of outcome sequences involves processes of salience attribution and violation of expectations. Furthermore, problem gamblers seem to process near outcomes as more win-like compared to controls. The results and their implications for problem gambling as well as further possible lines of research are discussed.

Zusammenfassung

Glücksspiel ist eine verbreitete Aktivität in Deutschland. 40% einer repräsentativen Stichprobe gaben an mindestens einmal im vergangenen Jahr um Geld gespielt zu haben (Bundeszentrale für gesundheitliche Aufklärung, 2014). Während die Mehrheit der Glücksspieler unbedenkliches Spielverhalten zeigt, entwickelt ein Teil der Spieler ernsthafte Probleme durch das Spielen, die das psychische Wohlergehen, sowie das soziale und Arbeitsleben beeinträchtigen. Nach aktuellen Schätzungen sind bis zu 0,8% der deutschen Bevölkerung von solch pathologischem Glücksspielen betroffen. Generell zeigen Menschen verschiedene kognitive Verzerrungen im Sinne falscher Einschätzungen der Gewinnwahrscheinlichkeit und der Beteiligung von Fähigkeiten in Bezug auf Glücksspiel. Dies trifft insbesondere für pathologische Glücksspieler zu. Das Ziel der vorliegenden Arbeit ist die Untersuchung der biopsychologischen Grundlagen zweier solcher kognitiver Verzerrungen, der Kontrollillusion sowie der Gambler's Fallacy (bisweilen auch Spielerfehlschluss genannt) und Hot-Hand-Phänomene, sowie deren Modulation durch Glücksspielprobleme. Zu diesem Zweck wurden vier Studien durchgeführt, die die Verarbeitung knapper Ergebnisse (stellvertretend für die Kontrollillusion) und Ergebnissequenzen (stellvertretend für die Phänomene der Gambler's Fallacy und Hot Hand) untersuchten. Dazu wurden Stichproben mit unterschiedlichem Schweregrad der Glücksspielproblematik sowie ein Multi-Methoden Ansatz verwendet.

Die erste Studie untersuchte die Verarbeitung und Bewertung knapper Ergebnisse sowie das Wahlverhalten in einem Glücksrad Paradigma mittels Elektroenzephalographie (EEG). Um den Einfluss der Glücksspielproblematik zu erfassen, wurde eine Gruppe von Problemspielern mit einer Kontrollgruppe verglichen. Es zeigten sich keine Unterschiede in der Verarbeitung knapper Ergebnisse zwischen den beiden Gruppen. Im Vergleich zu vollen Ergebnissen führten knappe Ergebnisse zu kleineren Amplituden in der P300. Des Weiteren zeigte sich auf Trendniveau im Wahlverhalten der Probanden Anzeichen für ein der Gambler's Fallacy entgegengesetztes Muster. Längere Sequenzen einer Ergebnisfarbe führten zu einer höheren Wahrscheinlichkeit diese Farbe im folgenden Durchgang erneut zu wählen. Schließlich zeigten Problemspieler relativ zur Kontrollgruppe kleinere Amplituden in der Feedbacknegativierung (FRN).

Die zweite Studie zielte ebenfalls auf die Verarbeitung knapper Ergebnisse im Glücksrad Paradigma ab, allerdings unter Verwendung funktioneller Magnetresonanztomographie sowie einer Probandengruppe mit variierendem Ausmaß der Glücksspielproblematik. Es zeigte sich eine verstärkte Aktivierung im bilateralen superioren parietalen Cortex nach knappen im Vergleich zu vollen Ergebnissen.

Die dritte Studie untersuchte peripherphysiologische Reaktionen auf knappe Ergebnisse im Glücksrad. Hierzu wurden Herzperiode und Hautleitfähigkeit erfasst während eine Probandengruppe mit unterschiedlichem Ausmaß an Glücksspielproblemen am Glücksrad spielte. Knappe Ergebnisse führten im Vergleich zu vollen Ergebnissen zu verlängerten Herzperioden kurz nach dem Ergebnis. Des Weiteren wurden die Herzperiodenreaktion und Hautleitfähigkeitsreaktion durch Glücksspielprobleme moduliert. Probanden mit einem hohem im Vergleich zu einem niedrigen Ausmaß an Glücksspielproblemen zeigten gesteigerte Hautleitfähigkeitsreaktionen auf knappe im Vergleich zu vollen Ergebnissen, sowie ähnliche Herzperiodenreaktionen auf knappe Ergebnisse und volle Gewinne.

Die vierte Studie untersuchte das Wahlverhalten sowie Einflüsse vorheriger Sequenzen auf die Verarbeitung von Ergebnissen in einem Münzwurf Paradigma. Hierzu wurde ein EEG bei einer Gruppe von Problemspielern und Kontrollprobanden abgeleitet. Problemspieler zeigten wiederum generell kleinere FRN Amplituden als Kontrollen. Es zeigten sich keine Unterschiede in der Verarbeitung der Ergebnissequenzen zwischen den Gruppen. Die Unterbrechung einer Sequenz gleicher Ergebnisse führte zu verstärkter Power im Theta Frequenzband. Zusätzlich war die Amplitude der P300 nach zwei vorangegangenen Gewinnen erhöht. Schließlich zeigten Problemspieler im Vergleich zu Kontrollen die Tendenz das gewählte Symbol relativ zum vorangegangenen Ergebnissymbol häufiger zu wechseln.

Zusammenfassend deuten die Ergebnisse auf Unterschiede in der Verarbeitung knapper und voller Ergebnisse hin, die vor allem Gehirnareale und Prozesse umfassen, die mit Aufmerksamkeit und Salienz assoziiert sind. Die Verarbeitung von Ergebnissequenzen umfasst Prozesse der Salienzzuschreibung und Erwartungsverletzung. Außerdem scheinen Problemspieler im Vergleich zu Kontrollen knappe Ergebnisse als gewinnähnlicher zu verarbeiten. Die Ergebnisse und deren Implikationen für problematisches Glücksspielverhalten sowie weitere mögliche Forschungsfragen werden diskutiert.

1 Introduction

> *"No one can say exactly who invented prayer, music, farming, medicine, or money. The same must be said for gambling: It is simply older than history."*
> (Schwartz, 2006, p.5)

Gambling has been a popular activity throughout human history. Its exact origin cannot be traced back. However, the precursors of dice games likely originated from religious practices like divination, which, among others, involved throwing small bones to determine divine answers to peoples' questions (Schwartz, 2006). Throughout the millennia, gambling has lost nothing of its popularity. According to a recent survey, about 80% of Germans aged 16 to 65 have gambled at least once in their lifetime, while 40% reported having gambled in the past year (Bundeszentrale für gesundheitliche Aufklärung, 2014). Compared to surveys on gambling in other countries, the past year prevalence of gambling in Germany is rather low, with other countries showing past year gambling prevalences of 60% to 70% (Williams, Volberg, & Stevens, 2012). While the majority of gamblers does not show problematic behavior related to gambling, a minority develops gambling problems of varying severity over time and can be labelled as pathological or problem gamblers. Problem and pathological gamblers hold stronger misconceptions about gambling than non-problem gamblers. Those misconceptions, called cognitive distortions, refer to the chances of winning and skill involvement in gambling. Two related phenomena are the near miss and fallacies in gambling, especially the gambler's fallacy and the hot hand fallacy. The current work aims at elucidating the biopsychological basis of these phenomena and its modulation by gambling problems.

The following sections first give some theoretical background on pathological gambling and cognitive distortions in gambling, with a special focus on near misses and the gambler's and hot hand fallacies. Then the biopsychological measures and questionnaires used in the current work are introduced. The four studies are presented before their results are integrated in a final discussion, also drawing conclusions for future research.

2 Theoretical background

The following sections first introduce the terms of pathological and problem gambling before several models of pathological gambling are presented and integrated. Next, cognitive distortions in gambling are introduced, with a special focus on near misses, the gambler's fallacy and the hot hand fallacy.

2.1 Pathological gambling in the DSM

In the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev.; *DSM-IV*-TR; American Psychiatric Association, 2000) pathological gambling is defined as "persistent and recurrent maladaptive gambling behavior (…) that disrupts personal, family or vocational pursuits." (American Psychiatric Association, 2000, p.615). The diagnosis of pathological gambling is made if at least five of the A-criteria are met (see Table 1). In addition, the gambling behavior may not solely be caused by a manic episode (B-criterion). The term "problem gambling" is used to describe gambling issues which are not severe enough to warrant a diagnosis of pathological gambling (Raylu & Oei, 2002; Whelan, Meyers, & Steenbergh, 2007). As such, pathological gambling and problem gambling are comparable to substance dependence and substance abuse, respectively (Whelan et al., 2007), where a substance abuser experiences negative consequences of substance consumption but does not meet the criteria for substance dependence.

Both the *DSM-IV-TR* and the *International Statistical Classification of Diseases and Related Health Problems* (10th revision, *ICD-10;* World Health Organization, 1992) list pathological gambling along with other impulse control disorders (*DSM-IV-TR*: Impulse Control Disorders Not Elsewhere Classified, *ICD-10*: Habit and Impulse Disorders). However, past research together with the symptomatology of pathological gambling suggests that it might better be viewed as a behavioral addiction. Table 2 shows a comparison of *DSM-IV-TR* A-criteria for pathological gambling with the *DSM-IV-TR* A-criteria for substance dependence. As can be seen, there is considerable overlap between the A-criteria for substance dependence and pathological gambling. Substance dependences like alcohol dependence also have a high comorbidity in pathological gamblers (Kessler et al., 2008; Petry, Stinson, & Grant, 2005). Furthermore, there is an overlap in the genetic risk factors for developing pathological gambling and alcohol dependence (Slutske et al., 2000). Pathological gamblers and substance dependents also show similar cue reactivity on a subjective and physiological level (Goudriaan, de Ruiter, van den Brink, Oosterlaan, & Veltman, 2010; Kober et al., 2015; Potenza, 2008), as well as similar impairments in decision making (Goudriaan, Oosterlaan, Beurs, & van den Brink, 2005; Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009b) and executive functioning (Goudriaan, Oosterlaan, Beurs, & van den Brink, 2006). Finally, pathological gamblers and

substance dependents also show similar deviations in personality traits compared to healthy persons, for example increased impulsivity (Lawrence, Luty, Bogdan, Sahakian, & Clark, 2009a, 2009b; Leeman & Potenza, 2012).

Due to the considerable similarities between substance dependence and pathological gambling, pathological gambling has been moved to the new diagnostic category "Substance-Related and Addictive Disorders" in *DSM-5* (American Psychiatric Association, 2013). In addition, some further changes have been made to the diagnosis of pathological gambling. First, pathological gambling has been renamed into gambling disorder. Second, the "illegal acts" criterion has been removed from the pool of A-criteria. Third, an explicit time criterion has been added. Disordered gambling can only be diagnosed if the symptoms have been present for at least 12 months. Fourth, the threshold of A-criteria has been lowered from 5 to 4 symptoms which have to be present. Table 1 lists the A-criteria for disordered gambling in the *DSM-5*.

Table 1. A-criteria for pathological gambling/gambling disorder in the DSM-IV-TR and DSM-5

DSM-IV-TR	*DSM-5*
is preoccupied with gambling (e.g. preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble)	is often preoccupied with gambling (e.g. having persistent thoughts of reliving past gambling experiences, handicapping or planning the next venture, thinking of ways to get money with which to gamble)
needs to gamble with increasing amounts of money in order to achieve the desired excitement	needs to gamble with increasing amounts of money in order to achieve the desired excitement
has repeated unsuccessful efforts to control, cut back, or stop gambling	has made repeated unsuccessful efforts to control, cut back, or stop gambling
is restless or irritable when attempting to cut down or stop gambling	is restless or irritable when attempting to cut down or stop gambling
gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g. feelings of helplessness, guilt, anxiety, depression)	often gambles when feeling distressed (e.g., helpless, guilty, anxious, depressed)
after losing money gambling, often returns another day to get even (“chasing” one’s losses)	after losing money gambling, often returns another day to get even (“chasing” one’s losses)
lies to family members, therapists, or others to conceal extent of involvement with gambling	lies to conceal the extent of involvement with gambling
has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling	-
has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling	has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling
relies on others to provide money to relieve a desperate financial situation caused by gambling	relies on others to provide money to relieve desperate financial situations caused by gambling

Table 2. Comparison of the DSM-IV-TR A-criteria for pathological gambling and substance dependence

Pathological Gambling	Substance Dependence
is preoccupied with gambling (e.g. preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble)	a great deal of time is spent in activities necessary to obtain the substance (e.g., visiting multiple doctors or driving long distances), use the substance (e.g., chain-smoking), or recover from its effects
needs to gamble with increasing amounts of money in order to achieve the desired excitement	tolerance, as defined by either of the following: a) a need for markedly increased amounts of the substance to achieve intoxication or desired effect b) markedly diminished effect with continued use of the same amount of the substance
has repeated unsuccessful efforts to control, cut back, or stop gambling	there is a persistent desire or unsuccessful efforts to cut down or control substance use
is restless or irritable when attempting to cut down or stop gambling	withdrawal, as manifested by either of the following: a) the characteristic withdrawal syndrome for the substance b) the same (or closely related) substance is taken to relieve or avoid withdrawal symptoms
gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g. feelings of helplessness, guilty, anxiety, depression)	
after losing money gambling, often returns another day to get even (“chasing” one’s losses)	
lies to family members, therapists, or others to conceal extent of involvement with gambling	
has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling	
has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling	important social, occupational, or recreational activities are given up or reduced because of substance use
relies on others to provide money to relieve a desperate financial situation caused by gambling	
	the substance is often taken in larger amounts or over a longer period than was intended

Pathological Gambling	Substance Dependence
	the substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance

2.2 Prevalence of pathological gambling

Recent past year prevalence estimates for pathological gambling in Germany range from 0.2% to 0.8% (Bühringer, Kraus, Sonntag, Pfeiffer-Gerschel, & Steiner, 2007; Bundeszentrale für gesundheitliche Aufklärung, 2014; Meyer et al., 2015). Compared to other countries, Germany thus has a rather low prevalence of pathological gambling. Williams et al. (2012) computed standardized estimates of pathological gambling corrected for influences of assessment instrument, time frame of gambling symptoms, method of administration, description of the survey to the participants and threshold to administer problem gambling questions. The standardized prevalence rate of pathological gambling ranged from 0.5% in Denmark to 6.4% in South Africa with an average of 2.3%.

2.3 Models on the development of pathological gambling

Several theories and models explaining the development of pathological gambling have been proposed. These range from theories originally developed for substance dependence which are then applied to pathological gambling, to models explicitly developed for pathological gambling. The following section introduces four of these models.

2.3.1 Incentive-sensitization theory

The incentive-sensitization theory (Robinson & Berridge, 1993) is a prominent theory explaining the development of substance dependence. Rømer Thomsen, Fjorback, Møller, and Lou (2014) have applied the incentive-sensitization theory to the development of pathological gambling. The original incentive-sensitization theory proposes that drugs create long lasting changes in the brain, mostly in brain systems involved in incentive motivation (Robinson & Berridge, 2001). These brain systems subsequently become hyperactive and react more strongly to stimuli associated with the drug (Robinson & Berridge, 2001). Importantly, only the system mediating incentive motivation, that is, the “wanting” of the drug, is affected, but not the system mediating the actual pleasure (“liking”) derived from the drug (Robinson & Berridge, 2001). Thus, the development of a substance addiction is characterized by increasing feelings of “wanting” the drug, while the “liking” of the drug stays the same. Robinson and Berridge (1993) proposed the mesotelencephalic dopamine system as

main target of the sensitization process and that people differ in their proneness to sensitization. Rømer Thomsen et al. (2014) reviewed evidence for incentive sensitization in pathological gamblers. They noted that pathological gamblers show an attentional bias towards gambling-related cues (Brevers, Cleeremans, Tibboel et al., 2011; Brevers, Cleeremans, Bechara et al., 2011), as well as signs of neuronal sensitization, both in the form of increased striatal activity (van Holst, Veltman, Büchel, van den Brink, & Goudriaan, 2012) and increased reward positivity (Hewig et al., 2010; Oberg, Christie, & Tata, 2011) following presentation of gambling-related cues.

2.3.2 General theory of addictions

Jacobs (1986) proposed a general theory of addictions, describing the development and necessary predisposing factors of both behavioral and substance-related addictions. According to this model, two kinds of predisposing factors have to be present to put someone at risk for developing an addiction. Those are either a hypotensive or hypertensive resting state of arousal, meaning that the person is either chronically under- or overaroused. The second factor refers to relationship and social problems during childhood and early adolescence. According to Jacobs (1986), children who feel inadequate and rejected by their parents and peers and subsequently indulge in wishful thinking instead of proactively coping with their problems, are at risk of developing an addiction later on. The development of an addiction spans three stages. The first stage is characterized by the first contact with the addictive substance or behavior. This contact results in a positive, stress relieving experience, which is followed by a return to the original negative state, though. By voluntarily seeking out the drug or behavior that created the positive state, the person gradually drifts into stage II of the model, which is characterized by constant ups and downs created by the drug-intake or behavior, which temporarily leads to tension-relief and pleasure, but is always followed by a rebound into the original negative state. The intake of the drug or execution of the behavior is strengthened through both positive and negative reinforcement, with negative reinforcement being the more important mechanism as the addiction progresses, according to Jacobs (1986). The addicted individual gradually spends more and more time on the addictive behavior or substance, at the same time neglecting other behaviors and relationships. This continues until the person reaches stage III, which is characterized by general exhaustion of the individual.

2.3.3 Biopsychosocial model of pathological gambling

Sharpe (2002) proposed a biopsychosocial model of pathological gambling. This model assumes both genetic and early environmental risk factors, in turn leading to increased psychological and biological vulnerability. In accordance with the classic diathesis-stress model, vulnerable people are supposed to more likely develop gambling problems given the respective stressor. According to

Sharpe's (2002) model, the further development of gambling problems is influenced by early gambling experiences. Early wins in the gambling "career" are thought to foster cognitive distortions, like the illusion of control (i.e. a feeling of increased control over the outcomes in gambling) or the gambler's fallacy (i.e. the feeling that a win is more likely following a series of losses), but also increased arousal. Once these distortions have developed, gamblers are at a high risk of developing a problematic gambling behavior, according to Sharpe (2002). At this point, the model acknowledges two different subtypes of gamblers, differing in their motives behind gambling and thus the stressor that finally triggers problematic gambling behavior. According to the model, both groups gamble to cope with unpleasant affective states. For one group, the unpleasant affect consists of boredom, thus these people gamble to increase their arousal and likely prefer gambles like betting on horse racing. The second group, on the other hand, suffers from dysphoric mood and stress, thus gambling to escape these negative feelings. This group is more likely to prefer slot machines/electronic gaming machines. Both groups use gambling as a way to cope with their unpleasant emotions, which are alleviated by gambling. The resulting reinforcement leads to an increase of gambling urges over time. These urges can be controlled by adequate coping strategies. However, if the person does not possess such strategies, he or she will spiral further into problematic gambling behavior.

2.3.4 Pathways model

Blaszczynski and Nower (2002) proposed a model that describes both the development of pathological gambling, as well as different subtypes of pathological gamblers. According to this model, the different groups of pathological gamblers share ecological influence factors and basic mechanisms of acquiring a pathological gambling behavior. The ecological factors increasing the risk for pathological gambling are availability and accessibility of gambling occasions. Increasing the availability and accessibility of gambling, for example by opening new casinos or introducing new lottery systems, leads to an increase of the amount of money spent gambling and also to an increase of excessive amounts of money spent gambling (Grun & McKeigue, 2000). From the first instances of gambling, a path of conditioning, habituation and chasing can lead to the development of pathological gambling. This pathway is common for all subgroups of pathological gamblers proposed by Blaszczynski and Nower (2002). Gambling, especially the intermittently occurring wins while gambling, increase both subjective and physiological arousal and thus reinforce gambling behavior through operant conditioning. Furthermore, the experienced arousal also becomes linked to environmental stimuli, such as the gambling venue, via classical conditioning. With increasing gambling frequency, several cognitive distortions, that is, incorrect beliefs about gambling and the chance of winning, develop, for example an increased illusion of control, referring to the feeling that one has more control over gambling outcomes than is actually the case. The gambler develops a

habit of gambling and in the process also starts losing more and more money. The accumulating losses eventually lead to chasing behavior, that is, gambling even more in order to try to win back the lost money.

Blaszczynski and Nower (2002) proposed that one subgroup of pathological gamblers, labelled “behaviorally conditioned” pathological gamblers, is characterized by only this pathway and neither has any predisposing conditions for developing pathological gambling nor any other psychopathologies before the onset of the pathological gambling. Compared to the other two subgroups of pathological gamblers, the behaviorally conditioned group has the lowest level of gambling problems. The other two subgroups are characterized by additional premorbid risk factors, making these people especially vulnerable for developing pathological gambling.

The second subgroup, labelled “emotionally vulnerable” pathological gamblers, show a range of emotional and biological risk factors for developing pathological gambling. The emotional factors include negative childhood experiences, personality traits like increased risk-taking and boredom proneness, mood and anxiety disorders, as well as a lack of adequate coping skills. The biological risk factors include abnormalities in neurotransmitter systems and general cortical activity. Unlike the “behaviorally conditioned” subgroup, the “emotionally vulnerable” subgroup is thought to initially start gambling as a way to cope with unpleasant emotional states. This includes both gambling to increase arousal levels (for boredom prone people) as well as gambling to reduce negative affect (for depressed and anxious people). Once started, the trajectory of developing a pathological gambling behavior is the same as in the “behaviorally conditioned” subgroup.

The third subgroup, “antisocial impulsivist” pathological gamblers, overall shows the highest amount of gambling problems and general psychopathology. According to the pathways model, they share the same vulnerabilities as the “emotionally vulnerable” group, but are characterized in addition by increased impulsivity, as well as symptoms of antisocial personality disorder and ADHD.

There is empirical evidence for the existence of subgroups of pathological gamblers, corresponding to the three groups proposed by the pathways model (Bonnaire, Bungener, & Varescon, 2009; Gupta et al., 2013; Ledgerwood & Petry, 2010; Turner, Jain, Spence, & Zangeneh, 2008; for a review see Milosevic & Ledgerwood, 2010), although so far no study has explicitly examined the actual development of pathological gambling proposed by the model in a longitudinal design.

2.3.5 Integrated model of pathological gambling

The main points of the four previously introduced models are summarized in Figure 1. In accordance with Blaszczynski and Nower (2002), the main mechanisms behind the development of gambling problems assumed by the integrated model of pathological gambling are classical and operant conditioning. Availability and accessibility of gambling opportunities are the basic prerequisites for starting gambling and developing gambling problems later on. Both have increased in the past years, especially accessibility, due to increased opportunities in internet gambling (Griffiths, 2003). When people gain initial experience with gambling, they are reinforced through operant conditioning. At the same time, the positive experiences of gambling are associated with stimuli and cues in the environment via classical conditioning, such that these cues increase the want to gamble when they are encountered. Depending on individual predisposing and risk factors, people are more or less prone to the consequences of operant and classical conditioning in gambling. Some people are more susceptible to sensitization and related changes in the dopaminergic system, making them more susceptible to classical conditioning of gambling-related cues and subsequent cue induced urges to gamble. Concerning operant conditioning, wins act a as a positive reinforcer for all people. However, for some people gambling itself can act as a positive or negative reinforcer by regulating arousal and emotional states. People with chronic low levels of arousal can use gambling to increase their arousal to subjectively comfortable levels, thus turning the act of gambling into a positive reinforcer. People with chronic high levels of arousal or negative emotional states can use gambling as negative reinforcer, reducing their arousal or escaping from their negative emotions, respectively. The reinforcement gained through gambling subsequently leads to repeated gambling, through which cognitive distortions related to gambling and eventually loss chasing develop, leading to problem and pathological gambling. The integrated model of pathological gambling also takes into account characteristics of the gambling games (e.g. event frequency, light and sound effects, skill elements), since research has shown that certain characteristics are more likely to lead to problematic gambling behavior (for an overview see Parke & Griffiths, 2007). The integrated model proposes that these characteristics interact with individual risk factors, as a result making certain games especially attractive and eventually dangerous for certain groups of people, depending, for example, on their chronic arousal state.

As such, the integrated model of pathological proposed here, focuses on conditioning as the main mechanism behind pathological gambling, as proposed by Blaszczynski and Nower (2002). Furthermore, similar to Jacobs' (1986) proposed stage II, the rewarding effects of gambling are supposed to lead to repeated gambling behavior, eventually resulting in the development of tolerance and decreased effects of gambling. Finally, the integrated model takes a diathesis-stress

perspective, assuming predisposing factors that can lead to the development of gambling problems. Thus, unlike the pathways model, the integrated model does not assume that people without at least a certain amount of risk factors develop gambling problems. Instead, a certain amount of predisposing risk factors are seen as necessary prerequisite to develop problem or pathological gambling, in accordance with Jacobs (1986) and Sharpe (2002). Persons who are not susceptible to gambling as a reinforcer per se (because they have normal levels of arousal and are in no negative emotional state), are not expected to develop gambling problems. Unlike the previously described models, the integrated model explicitly proposes to take game characteristics into account. While Sharpe's (2002) model does take into account different types of gamblers based on their preferred game, the underlying characteristics of those games are not explicitly taken into account in the model.

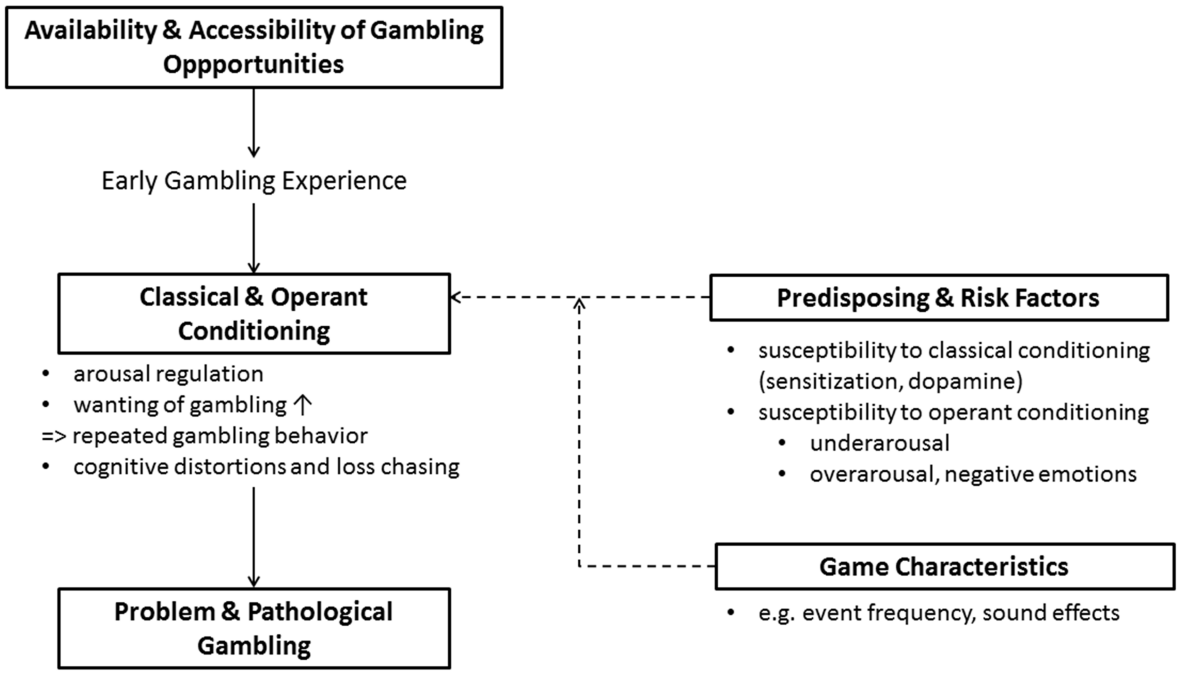

Figure 1. Integrated model of pathological gambling.

2.4 Cognitive distortions

A lot of people show misconceptions about randomness, the chances of winning and the involvement of skill in gambling. These beliefs are summarized under the term cognitive distortions. The work of Toneatto (1999) offers an overview of the broad range of cognitive distortions present in gambling (see Table 3).

Table 3. Cognitive distortions in gambling according to Toneatto (1999)

Category	Explanation
Magnification of gambling skill	Overestimation of one's skill and ability to win
Superstitious beliefs	
Talismanic superstitions	Belief that certain objects increase the probability of winning
Behavioral superstitions	Belief that certain behaviors increase the probability of winning
Cognitive superstitions	Belief that certain mental states increase the probability of winning
Interpretative biases	
Attributional biases	Wins are attributed to skill, losses are attributed to external factors like chance
Gambler's fallacy	Belief that a win is likely to occur after a series of losses
Chasing	Continuing to gamble after a series of losses to win back the money
Anthropomorphism	Attribution of human characteristics to gambling objects
Learning from losses	Reframing of losses as learning experience which will help to win in the future
Hindsight bias	Reevaluation of gambling decisions in the light of the corresponding outcome (if the decision resulted in a win it was correct, if it resulted in a loss it was incorrect)
Temporal telescoping	Belief that a win is close rather than far in time
Selective memory	Selective recall of wins
Predictive skill	Belief in being able to predict gambling outcomes based on salient cues (e.g. gut feeling)
Illusion of control over luck	General belief that luck influences gambling outcomes
Luck as uncontrollable variable	Belief that luck cannot be controlled
Luck as controllable variable	Belief that luck can be controlled
Luck as contagion	Belief that luck in other life domains or of other gamblers influences own gambling behavior
Illusory correlation	Perceiving causal relationships between environmental cues and gambling outcomes

Studies using the speaking aloud method, where gamblers are instructed to verbalize every thought they have during gambling, have shown that a large proportion of gambling-related thoughts reflect cognitive distortions about gambling (Delfabbro & Winefield, 2000; Gaboury & Ladouceur, 1989; Ladouceur, Gaboury, Bujold, Lachance, & Tremblay, 1991; Ladouceur, Gaboury, Dumont, & Rochette, 1988). Furthermore, cognitive distortions are more frequent in pathological compared to non-pathological gamblers (Cunningham, Hodgins, & Toneatto, 2014; Joukhador, Blaszczynski, & Maccallum, 2004; Joukhador, Maccallum, & Blaszczynski, 2003; MacLaren, Ellery, & Knoll, 2015; Myrseth, Brunborg, & Eidem, 2010; Xian et al., 2008). As such, they also provide a leverage point for treatments of pathological gambling. Interventions specifically aimed at correcting cognitive distortions in gambling have proven to be successful both in individual (Ladouceur et al., 2001) and group treatment settings (Ladouceur et al., 2003) compared to a wait-list control group and led to improvements comparable to behavioral therapy (Toneatto & Gunaratne, 2009).

The current work aims at examining three cognitive distortions in particular: the magnification of the skill component in gambling, the gambler's fallacy and the hot hand fallacy.

A phenomenon closely related to the magnification of skill in gambling is the illusion of control. It has first been described by Langer (1975) and refers to "an expectancy of a personal success probability inappropriately higher than the objective probability would warrant" (Langer, 1975, p.313). Langer (1975) hypothesized that this illusion could be evoked in chance situations by adding certain aspects of skill situations. The influence of competition, choice, stimulus familiarity, response familiarity and involvement on the illusion of control was tested. The results showed that all manipulations led participants to behave in a way as though they had control over the chance event. For example, participants' behavior indicated that a self-chosen lottery ticket was deemed more valuable than a lottery ticket chosen by the experimenter, even though both have the same chance of winning.

Another feature that potentially increases the illusion of control in games of chance is the near miss. Relevant research on near misses, the gambler's fallacy and the hot hand fallacy will be discussed in the following two sections.

2.4.1 Near misses

A near miss is a miss outcome which would have almost resulted in a win (Reid, 1986). In games of skill, a near miss does not only convey information about the outcome but also about how to change ones behavior to be successful in the future. For example, a soccer player who barely misses the goal knows that he only has to adjust his technique a bit to be successful next time. However, in games of chance a near miss carries no additional information on how to change ones

behavior to increase the chances of success, as the outcome in a game of chance cannot be influenced by behavior. Yet, several lines of research suggest that near misses in games of chance are distinct from other miss events (henceforth called full misses) and are seen as somewhat reinforcing events.

It has been shown that near misses can prolong the amount of time spent gambling. Kassinove and Schare (2001) demonstrated that a near miss rate of 30% led participants to continue gambling longer than either a 15% or a 45% near miss rate. The authors explain this finding by learning theory. From this point of view near misses are seen as conditioned reinforcers. They acquire this status since they are sometimes followed by wins. Of the three near miss rates tested, 30% seemed to be the best rate for this conditioning process. With a 15% near miss rate there are presumably too few near misses and in case of a 45% near miss rate there are so many near misses that too few of them are actually followed by wins, thus conditioning does not take place (Kassinove & Schare, 2001). The finding that near misses can prolong gambling was replicated by Côté, Caron, Aubert, Desrochers, and Ladouceur (2003), who showed that participants who received near misses in 25% of trials gambled longer than participants who received no near misses.

On a subjective level, some studies have shown that near misses compared to full misses have been rated as being less pleasant but more motivating (Clark, Crooks, Clarke, Aitken, & Dunn, 2012; Clark, Lawrence, Astley-Jones, & Gray, 2009; Qi, Ding, Song, & Yang, 2011), which has already been indicated by informal interview data reported by Reid (1986). However, this effect could not always be replicated (Chase & Clark, 2010; Ulrich & Hewig, 2014). Furthermore, near misses have been rated as being closer to a win than to a miss (Dixon & Schreiber, 2004; Dymond et al., 2014; Habib & Dixon, 2010). However, the post-reinforcement pause, defined as the time between outcome of one trial and initiation of the next trial, which is typically longer following positive reinforcers (e.g. wins) compared to negative reinforcers (Delfabbro & Winefield, 1999; Felton & Lyon, 1966), does not differ between near and full misses, which show shorter post-reinforcement pauses than wins (Dixon, MacLaren, Jarick, Fugelsang, & Harrigan, 2013; Stange, Graydon, & Dixon, 2016).

The processing of near misses has been examined using various biopsychological methods. The following section describes and summarizes studies on the processing of near misses using functional magnetic resonance imaging (fMRI), electroencephalography (EEG), peripheral physiology, magnetoencephalography (MEG) and electromyography (EMG). For each method, studies using non-problem gambling participants will be reported first, followed by studies including problem or pathological gamblers.

The first study to investigate the processing of near misses was an fMRI study by Clark et al. (2009). The paradigm used was a simplified slot machine. Participants had to choose one of six

winning symbols on the first reel. The second reel then spun and stopped. If the winning symbol on the second reel stopped in the same row as the winning symbol on the first reel, the participant won. Otherwise, the participant did not win anything, but also did not lose anything. Near misses were operationalized as outcomes in which the winning symbol on the second reel stopped one position next to the winning symbol on the first reel. The other miss outcomes were full misses. Wins occurred on 1/6 of the trials, near misses on 2/6 and full misses on 3/6 of the trials. The analysis of the fMRI data showed increased signal change in the ventral striatum and the right anterior insula following near compared to full misses. These regions were also more active following wins compared to all miss outcomes. Thus, this study showed that near misses are processed as being somewhat win-like, even though they are not associated with any financial gain. Similar results of an overlap of activated areas in contrasts comparing wins to all misses and near misses to full misses have been reported by Dymond et al. (2014).

A study by Habib and Dixon (2010) reported a somewhat different finding. Pathological and non-pathological gamblers played on a slot machine which delivered equal amounts of wins, near misses and full misses. For non-pathological gamblers, the areas activated following near misses showed a greater overlap with the areas activated following full misses than with the areas involved in processing wins. For pathological gamblers, the reverse result was found. Areas activated by near misses showed a greater overlap with areas activated following wins than with areas activated following misses. According to the authors, these results show that near misses have features of both wins and losses, and that non-pathological gamblers might be more inclined to see the loss-like features while pathological gamblers focus on the win-like features. However, due to some methodological issues, the results should be interpreted cautiously. Firstly, the sample sizes were rather small with only 11 participants per group. Secondly, there was a gender imbalance between groups, with the pathological gambling group consisting of 10 males and 1 female and the non-pathological gambling group consisting of 4 males and 7 females. Finally, the groups were recruited based on their South Oaks Gambling Screen (SOGS, Lesieur & Blume, 1987) scores, with a cutoff of 3 or more points used for the pathological gambling group. This is lower than the originally proposed cutoff of 5 points, thus it might be more appropriate to label the group as problem gamblers.

Chase and Clark (2010) investigated the processing of near misses in a group of 20 regular gamblers, 13 of whom scored at or above the SOGS cutoff for pathological gambling (SOGS >= 5). The analysis of the fMRI data showed greater activity in the ventral striatum following near compared to full misses and an influence of problem gambling severity on the processing of near misses. There was a positive correlation between the SOGS scores and activity following near compared to full misses in the dopaminergic midbrain. A comparison to the sample of non-pathological gamblers

described in Clark et al. (2009) showed weaker activation following wins in reward-related brain areas like the striatum in the current sample. These findings are in line with previous studies showing decreased activity in the striatum following wins in pathological gamblers compared to a control group (Reuter et al., 2005). At the same time, Chase and Clark (2010) found no difference in the activity following near misses between the two samples. Taken together, these results point towards a special role of near misses in pathological gambling.

A reanalysis of the combined fMRI datasets of Clark et al. (2009) and Chase and Clark (2010) assessed functional connectivity following near misses (van Holst, Chase, & Clark, 2014). This analysis specifically targeted neurobiological foundations of the illusion of control. The paradigms used by Clark and colleagues manipulated control as an additional factor. In half of the trials participants chose the winning symbols themselves, whereas in the other half of the trials the computer chose the winning symbol. This manipulation is similar to the choice manipulation in Langer's (1975) original studies on the illusion of control. Thus, participants should have had an increased sense of control over the outcomes in the trials in which they got to choose the winning symbol themselves. Van Holst et al. (2014) analyzed connectivity using the left and right ventral striatum as seed regions. They found that on participant compared to computer chosen trials, near compared to full misses led to increased connectivity between the right ventral striatum and the bilateral insula. This increase in connectivity correlated positively with gambling severity measured via the SOGS. This result is in line with previous research, highlighting the role of the insula in addiction (Naqvi, Rudrauf, Damasio, & Bechara, 2007; Verdejo-Garcia, Clark, & Dunn, 2012) and near miss processing (Clark, Studer, Bruss, Tranel, & Bechara, 2014). The insula has been shown to be involved in craving and urges in substance addiction (Bonson et al., 2002; Brody et al., 2002; Naqvi et al., 2007; Wang et al., 1999). It has been proposed that the insula represents the interoceptive effects of drug consumption in an explicit way, making them available to conscious processing (Naqvi & Bechara, 2010). Similarly, Clark et al. (2014) proposed that the insula processes the peripheral physiological reactions elicited by near misses.

A more recent study comparing cocaine addicts, pathological gamblers and healthy controls could not replicate win-related activity following near misses in pathological gamblers (Worhunsky, Malison, Rogers, & Potenza, 2014). In this study, only the control group showed increased activity in the ventral striatum and the right insula following near misses, while there were no significant activations in the pathological gamblers and the cocaine addicts.

The first EEG studies on the processing of near misses were published in 2011. Most of the studies focused on event-related potentials (ERPs) related to feedback processing, specifically the feedback-related negativity (FRN), indicating an initial evaluation of the feedback concerning its valence (positive versus negative; Hajihosseini & Holroyd, 2013), and the P300, indicating the

motivational salience of the feedback (Johnson, 1986). For a more thorough description of the two potentials see sections 3.1.1.1.1 and 3.1.1.1.2.

Luo, Wang, and Qu (2011) used a slot machine paradigm which delivered wins, near misses and full misses on 1/3 of the trials, respectively. The analysis of the ERPs showed that near misses elicited a smaller (i.e. less negative) FRN compared to full misses. At the same time, there was no difference in P300 between near and full misses. These findings were replicated by Lole, Gonsalvez, Barry, and de Blasio (2013) and Lole, Gonsalvez, and Barry (2015).

A different result was reported by Qi et al. (2011). This study used a simple gambling task where participants had to stop a moving payline at a specific position but actually had no control over the outcome. Wins, near misses and full misses were delivered on 2/7 of the trials, respectively. The remaining 1/7 of trials comprised miss outcomes which were between near and full misses, concerning their distance from the winning position. Contrary to the results of Luo et al. (2011), Qi et al. (2011) found no significant difference between near and full misses in the FRN. However, there was a significant difference in the P300, with near misses eliciting a larger P300 than full misses. The amplitude of the P300 was furthermore related to ratings of motivation to continue gambling across all outcomes. The larger the P300, the higher the motivation rating was. A larger P300 amplitude for near compared to full misses has also been reported by Alicart, Cucurell, Mas-Herrero, and Marco-Pallarés (2015).

Yet another result was obtained by Ulrich and Hewig (2014). The wheel of fortune paradigm used in this study differs from the typical paradigm used to study near misses, as it also delivers near wins (also referred to as narrow wins). Near wins were conceptualized to be counterparts of near misses. Thus, a near win refers to a win that would have almost resulted in a loss. The wheel of fortune delivered four different outcome types (full wins, near wins, full misses, near misses), each occurring on 1/4 of the trials. This paradigm allows examining a general effect of closeness by comparing near to full outcomes irrespective of the valence (win or miss). A main effect of closeness was found for both FRN and P300. Near compared to full outcomes elicited a more negative FRN and a smaller P300.

In addition to ERPs, Alicart et al. (2015) also conducted a time-frequency analysis in the theta (4-8 Hz), alpha (9-13 Hz), lower beta (15-22 Hz) and beta-gamma band (25-35 Hz). The time windows and electrode positions included in the analysis were determined by prior literature and visual inspection, separately for each frequency band. Across all bands, near misses showed increased power relative to full misses, while there were no significant differences between wins and near misses. This result further stresses the win-like features of near misses.

To date, only two studies have compared the processing of near and full misses between pathological gamblers and controls using EEG. Kreussel et al. (2013) compared a group of male pathological gamblers to matched controls on a blackjack task. The goal in this task was to acquire a higher score than the bank while staying below 22 points. A score of 22 or more points led to an automatic loss of the current trial. Near and full misses were determined based on the scores. Trials with a final score of 22 and 23 constituted near misses, while trials with a score of 24-26 constituted full misses. The ERP in the FRN time frame was found to be significantly more negative after near compared to full misses in the control group, while there was no difference between the two outcomes among the pathological gamblers. According to the authors, this finding indicates a more negative evaluation of near misses in the controls, whereas the pathological gamblers did not differentiate the two miss outcomes.

However, Lole et al. (2015) compared pathological gamblers and controls on a slot machine task and found smaller (i.e. less negative) FRN values for near compared to full misses for both pathological gamblers and healthy controls.

One study used MEG as another method for analyzing central nervous processing of near misses (Dymond et al., 2014). The sample consisted of a group of non-problem gamblers and a group of problem and pathological gamblers. Participants gambled on a slot machine paradigm while the MEG was recorded. Near compared to full misses were followed by an increase in theta power in the anterior frontal cortex, orbitofrontal cortex and anterior insula. Furthermore, problem gamblers showed a significantly larger increase in theta power following near misses than non-problem gamblers. Power changes following wins and full misses did not differ between groups.

Some recent studies have investigated the processing of near misses using peripheral physiology measures, more specifically skin conductance and heart rate or heart period. The first to do so aimed at investigating the physiological arousal elicited by near misses and whether there was any relation to gambling severity (Dixon et al., 2011). Participants ranged from non-problem gamblers to problem gamblers, determined by the Canadian Problem Gambling Index, another screening instrument for problem gambling (Ferris & Wynne, 2001). A slot machine paradigm was used, delivering wins on 20% of trials, near misses on 15% of trials and full misses on 65% of trials. Analysis of the skin conductance data showed that near misses elicited larger skin conductance responses (SCR) than both wins and misses, while the latter two did not differ. This suggests that near misses elicit even more physiological arousal than wins. In addition, heart period reactions associated with the gambling outcomes were analyzed. Again, near misses led to the largest reactions. Wins were followed by longer interbeat intervals (IBIs) than full misses. Near misses in turn elicited even longer IBIs. Similar to the SCR results, this effect was not influenced by gambling

problem severity. Another study by Dixon et al. (2013) also showed increased SCRs to near compared to full misses, which again was not related to gambling severity. While the skin conductance results suggest that near misses have something in common with wins, another behavioral measure included by Dixon et al. (2013) argues against this notion. The post-reinforcement pause, referring to the time between outcome delivery and the start of the next gamble (e.g. wheel spin on a slot machine), was also measured in the study. Post-reinforcement pauses have been found to be longer after positive reinforcers (e.g. wins) compared to negative reinforcers (Delfabbro & Winefield, 1999; Felton & Lyon, 1966). Dixon et al. (2013) found longer post-reinforcement pauses following wins compared to near misses, which in turn did not differ from full misses. Thus, the authors conclude that near misses are highly frustrating events, that, however, increase the motivation to continue gambling.

The skin conductance results were partly confirmed in a study by Clark et al. (2012). Near misses again showed increased SCRs compared to full misses. Near misses and wins were not directly compared in the analysis, yet, descriptively the data indicate that near misses elicited smaller SCRs than wins. The heart rate response following outcomes showed a biphasic reaction with an initial decrease and a subsequent increase in heart rate. While there were no differences between the outcomes in the heart rate decrease, the heart rate increase for near misses was larger than that for wins and full misses, with no significant difference between the latter two. There was also no relation to gambling severity, although this result should be interpreted with caution, since only a small proportion of the sample actually scored in a problem gambling range. Clark et al. (2013) could replicate the SCR results in a later study. However, they did not replicate the effect on heart rate increase.

Instead of the mostly used slot machines, Stange et al. (2016) used a scratch card paradigm to investigate near miss processing. Each scratch card contained three separate fields with six symbols each. Participants could either win $ 5.00 by uncovering three $ 5.00 symbols within one field or weekly payments of $ 25.00 dollars for a month, by uncovering three “month” symbols. The scratch cards were rigged, such that every participant experienced a $ 5.00 win, a near miss of the weekly $ 25.00 payment, operationalized as two “month” symbols within one field, and four full misses, operationalized as six non-matching symbols within one field. Participants were instructed to scratch the cards one field at a time. Skin conductance level (SCL) and SCRs were analyzed for two different time frames: SCL was measured in a time frame encompassing the time between starting scratching the first symbol and finishing scratching the last symbol. SCRs to the outcomes were measured in a three seconds long time window which started one second after the last symbol was uncovered. Near misses showed the largest SCL increases while uncovering the symbols, whereas

wins and full misses did not differ on this measure. However, the outcome-related SCR was smaller for near misses than for full misses, with wins scoring in between. The latter finding is not in line with the results previously described. Stange et al. (2016) explain this finding by a potential contamination of the outcome-related SCR by the prior SCL. The SCL measure shows that near misses already had high skin conductance values prior to the outcome, which made a further increase unlikely. Post-reinforcement pause was also assessed and was found to be largest for wins with no significant difference between full and near misses, thus replicating the findings of Dixon et al. (2013).

Wu, van Dijk, and Clark (2015) used facial EMG to analyze emotional responses following two types of near outcomes in gambling: near wins and near losses. Near wins in this case refer to non-win outcomes which would have almost resulted in a win, whereas near losses refer to non-win outcomes which would have almost resulted in a loss. The facial EMG analysis focused on zygomaticus and corrugator activity. Near wins elicited increased activity in the zygomaticus compared to near losses and full misses (non-win outcomes which were not close to wins or losses). Near wins, near losses and full misses did not differ in elicited corrugator activity. Additional analyses showed that near wins and wins elicited the same response pattern of zygomaticus and corrugator activity, even though near wins did not yield any monetary gain.

To sum up, most research on near misses has focused on subjective ratings, fMRI, EEG and peripheral physiology. Near misses share aspects with both wins and full misses. Near misses are rated as being closer to wins than to misses (Dixon & Schreiber, 2004) and also increase the motivation to continue gambling (Clark et al., 2009; Qi et al., 2011). At the same time, near misses are rated as being unpleasant events, even more so than full misses (Clark et al., 2009; Qi et al., 2011). Furthermore, post-reinforcement pauses show the same pattern for near and full misses (Dixon et al., 2013; Stange et al., 2016). The majority of studies using samples of non-problem gamblers, show differences in the processing of near and full misses, that further stress the win-like properties of near misses. Near compared to full misses are followed by increased activity in win-related areas like the ventral striatum and the anterior insula (Clark et al., 2009; Worhunsky et al., 2014), as well as increased physiological arousal as indicated by increased SCRs (Clark et al., 2012; Dixon et al., 2011). Studies using event-related potentials yielded mixed results though, with some finding no difference between near and full misses for FRN (Qi et al., 2011) and P300 (Lole et al., 2015; Luo et al., 2011) while other studies report differences (Kreussel et al., 2013; Lole et al., 2013; Luo et al., 2011; Qi et al., 2011; Ulrich & Hewig, 2014), although the direction of the effect varies between studies. Studies including pathological or problem gamblers also show somewhat mixed results. Some studies suggest that near misses are processed as being more win-like in participants with gambling problems compared to healthy controls (Chase & Clark, 2010; Dymond et al., 2014;

Habib & Dixon, 2010), while other studies failed to find a relation between gambling problems and processing of near misses (Clark et al., 2012; Dixon et al., 2011; Worhunsky et al., 2014).

The inconsistencies in the P300 findings in the EEG studies could be influenced by the different outcome probabilities used. The P300 is larger following stimuli or outcomes occurring more seldom (Duncan-Johnson & Donchin, 1977; Sutton, Braren, Zubin, & John, 1965), an effect that is called "oddball effect". The majority of EEG studies on near misses have used three different outcomes: near misses, full misses and wins. With these three outcomes it is not possible to avoid stimulus probability effects in the P300. If all outcomes occur equally often, that is, every outcome in one third of the trials, one third of the trials consist of wins while two thirds consist of misses in general. In this case, larger P300 outcomes following wins compared to misses are at least partially caused by the smaller probability of wins occurring. The problem of differing stimulus probabilities can best be avoided by adding a fourth outcome category, the near win or narrow win (Ulrich & Hewig, 2014). A near win is a win that would have almost resulted in a miss. Thus, according to this definition, near wins are counterparts of near misses. With near misses, full misses, near wins and full wins, the outcome probabilities can be chosen to avoid confounding probability effects on the P300. If each of the four outcome types occurs in one fourth of the trials, half of the trials will be wins, while the other half will be misses, and half of the trials will be near outcomes while the other half will be full outcomes. Due to the advantage of avoiding confounding probability effects, a paradigm including near wins was chosen for the studies described later on.

2.4.2 Gambler's fallacy and hot hand fallacy

Both the gambler's and the hot hand fallacy refer to a misbelief about randomness in a broad sense. The gambler's fallacy has already been described by Laplace in the 18th century (Laplace, 1902). It refers to the belief that after a run of the same outcome in a binary sequence of equiprobable outcomes, the other outcome is more likely to occur in the next trial. This misbelief thus disregards the independence of the events and implicitly assumes a negative autocorrelation of events (Sundali & Croson, 2006). The hot hand fallacy, on the other hand, refers to the belief that a win is more likely to appear again following a series of wins, even though the series of wins and losses is actually a random sequence, as in pure games of chance, for example. Research on the hot hand fallacy originally started with an analysis of basketball players' performance. Gilovich, Vallone, and Tversky (1985) showed that basketball fans believe that players can become "hot", that is, show streaks of success, where a successful throw more likely follows previous successful throws. However, an analysis of the data of several basketball players showed that the probability of a successful throw was actually independent of previous successes (Gilovich et al., 1985). Thus, the

belief in the hot hand was termed a fallacy, since there is no empirical evidence for such an effect (Avugos, Köppen, Czienskowski, Raab, & Bar-Eli, 2013).

At a first glance, the gambler's fallacy and the hot hand fallacy seem to be complimentary phenomena, with the gambler's fallacy referring to the belief that a run is likely to end, and the hot hand fallacy referring to the belief that a streak is likely to continue. However, as Sundali and Croson (2006) note, these two fallacies are not exact opposites: "The gambler's fallacy describes beliefs about outcomes *of the random process* (e.g., heads or tails), while the hot hand describes beliefs of outcomes *of the individual* (like wins and losses)." (Sundali & Croson, 2006, p. 1). The direct opposite of the gambler's fallacy, that is, the expectation that a run of outcomes of a random process is likely to continue, is termed "hot outcome" (Sundali & Croson, 2006). The opposite of the hot hand fallacy, that is, the belief that a streak of outcomes of the individual is likely to end, is called "stock of luck belief" (Sundali & Croson, 2006). This term stems from the implicit belief that people have a set amount of luck, which is used up after a streak of wins. Thus, a miss is expected to occur more likely in the next trial. In the following, the term "run" is used to denote a sequence of the same outcome in a random process (e.g. heads or tails in a coin toss), while the term "streak" is used to denote a sequence of same outcomes of the individual (wins or losses).

The gambler's fallacy occurs both in laboratory settings (Ayton & Fischer, 2004; Barron & Leider, 2010; Burns & Corpus, 2004; Jarvik, 1951; Marmurek, Switzer, & D'Alvise, 2015), as well as in real world gambling behavior, for example in lotteries (Clotfelter & Cook, 1993; Terrell, 1994) and roulette (Sundali & Croson, 2006)[1]. The hot hand fallacy has been shown for sports (Gilovich et al., 1985)[2] and real world gambling (Sundali & Croson, 2006), as well as in laboratory settings (Ayton & Fischer, 2004).

Most studies on the gambler's and hot hand fallacies have used one of three methods: explanation, prediction or generation (Alter & Oppenheimer, 2006). In explanation studies

[1] A gambler's fallacy like pattern has also been shown for explicit expectations in paradigms linking two stimuli S1 and S2 (e.g. classical conditioning) where S2 follows S1 in 50% of the trials. Following longer runs of S1 without the occurrence of S2, participants expected S2 to occur in the following trial, whereas following longer runs of S1 followed by the occurrence of S2, participants thought it less likely that S2 would occur in the following trial (Perruchet 1985; Perruchet, Cleeremans, & Destrebecqz 2006). Interestingly, the strength of the conditioned eyeblink response (Perruchet 1985) and the reaction time to S2 (Perruchet et al. 2006) show a positive recency effect, that is, the eyeblink response is stronger and the reaction times faster for longer runs of CS-UCS/S1-S2 pairs in the previous trials.

[2] It should again be noted that a hot hand belief in sports is not necessarily a fallacy. Since sports contain a significant amount of skill, it is conceivable that athletes can actually produce streaks of successes, with higher probabilities of another success following previous successes, due to their ability. Thus, a belief in a hot hand in sports should only be termed a fallacy in cases where there is actually no such relationship between previous successes and the probability of another success. This has been shown for basketball (Attali, 2013; Gilovich et al., 1985; Koehler & Conley, 2003). However, for volleyball, for example, another study found evidence for a hot hand in athletes (Raab, Gula, & Gigerenzer, 2012). Hence, the corresponding hot hand belief of volleyball players and fans is actually not a fallacy.

participants see a sequence of binary outcomes and have to indicate which process (e.g. coin toss, basketball throws) likely generated the sequence (e.g. Ayton & Fischer, 2004; Fischer & Savranevski, 2015). In prediction studies participants predict the next outcome in a sequence of binary outcomes (e.g. Barron & Leider, 2010; Burns & Corpus, 2004). Finally, in generation studies participants have to generate a random sequence themselves (Wagenaar, 1972).

Several factors influence the occurrence and strength of the gambler's and hot hand fallacies. Binary outcome sequences that are caused by a random process (e.g. heads and tails in a coin toss sequence) elicit a gambler's fallacy, whereas non-random processes (e.g. hit and miss in basketball throws) elicit a hot hand fallacy (Burns & Corpus, 2004). This phenomenon might be related to the perception of intention in the generator of the outcome sequence. In a series of experiments, Caruso, Waytz, and Epley (2010) showed that participants are more likely to predict the continuation of a run in an outcome sequence produced by an intentional agent (e.g. a gambler trying to obtain a certain number in a dice roll). This tendency was stronger in people who showed an increased predisposition to perceive behavior in terms of the underlying intentions (e.g. locking the door) versus the abstract action (e.g. putting the key in the keyhole and turning it around) (Caruso et al., 2010). The gambler's fallacy is stronger when participants experience the outcomes in a sequential fashion, rather than seeing a sequence of outcomes all at once (Barron & Leider, 2010). However, the simultaneous presentation of outcomes can still elicit a gambler's fallacy, if the attention is drawn towards the last outcomes in the sequence (Barron & Leider, 2010). The gambler's fallacy furthermore depends on the grouping of the outcomes. It only occurs when the run is contained within one block, but disappears if the run occurs over two blocks, that is, around a pause between two blocks (Roney & Trick, 2003). The intertrial interval (ITI) also influences the occurrence of the gambler's fallacy and the hot hand fallacy. While longer ITIs mainly elicited a gambler's fallacy, shorter ITIs elicited equal amounts of gambler's fallacy and hot hand fallacy across participants (Militana, Wolfson, & Cleaveland, 2010). Furthermore, the gambler's fallacy is stronger in binary outcome sequences while adding more possible outcomes dampens the fallacy (Navarrete & Santamaría, 2012). In addition, Matthews (2013) demonstrated context dependency of the gambler's fallacy. Specifically, participants were more likely to show the gambler's fallacy for a run of outcomes of medium length if it was surrounded by runs of shorter length compared to runs of longer length. Thus, a contrast effect emerged, with outcome runs of medium length being perceived as shorter if surrounded by longer outcome runs, but being perceived as longer if surrounded by shorter outcome runs. Gender also influences the gambler's fallacy, with men showing an increased gambler's fallacy compared to women (Marmurek, Switzer, & D'Alvise, 2014, 2015; Suetens & Tyran, 2012). A recent study also showed cultural influences on the two fallacies, with Euro-Canadian participants showing a

stronger tendency towards the hot hand fallacy and Chinese participants showing a stronger tendency towards the gambler's fallacy (Ji, McGeorge, Li, Lee, & Zhang, 2015).

To date, several theories for the explanation of the gambler's and hot hand fallacies have been proposed. The first one builds on the law of small numbers (Tversky & Kahneman, 1971) and the representativeness heuristic (Kahneman & Tversky, 1972). The law of large numbers states that the mean of a random variable approaches the true value in the underlying population, the larger the sample size is. People actually show a belief in the law of small numbers, that is, they expect data from much smaller samples to match properties of the underlying population (Tversky & Kahneman, 1971). According to the representativeness heuristic, an uncertain event is judged based on how similar it is to the underlying population (Kahneman & Tversky, 1972). Applied to a random binary sequence with equal properties of p = .5 of both outcomes, people believe that short sequences should also reflect the overall randomness of the sequence (law of small numbers). Thus, if people observe a run within a short sequence, they expect that the other outcome is now more likely to occur, to balance the previous run and restore the randomness of the sequence, such that the short sequence is representative of the overall randomness in the process (Ayton & Fischer, 2004). Interestingly, the hot hand fallacy has also been explained using the same concepts (Ayton & Fischer, 2004; Gilovich et al., 1985). According to this explanation, a sequence containing long streaks is not representative of randomness and thus people reject the idea that it is generated by a random process and also no longer expect the streak to be balanced out.

Other explanations for the gambler's and hot hand fallacies focus on the perceived cause of the sequence. If the sequence is assumed to be generated by a random process (e.g. coin toss), runs are more likely predicted to end (Burns & Corpus, 2004). With non-random underlying processes (e.g. human success and failure in a skilled task), streaks are more likely predicted to continue (Burns & Corpus, 2004). A similar explanation focusses on the perceived intention of the agent generating the sequence (Caruso et al., 2010). According to this explanation, it is not the distinction random/non-random generating process which causes the gambler's and the hot hand fallacies, but the distinction between non-intentionality and intentionality. If an intentional agent (e.g. human) is assumed to be the generator of the process, streaks are expected to continue, whereas runs are expected to reverse, if generated by an unintentional agent (e.g. coin toss). Oskarsson, van Boven, McClelland, and Hastie (2009) integrated randomness and intentionality into a single mental model of generators of outcome sequences. According to the authors, people generate a mental model of the generator of an observed sequence of outcomes. This model includes information on four continuous dimensions: randomness, intentionality, control, and goal complexity. The gambler's fallacy should occur if the process is seen as random. Attributing intentionality and control to the

generating agent of an outcome sequence should lead to a hot hand fallacy. Goal complexity refers to the relationship between the outcomes of the sequence and the actual goal of the agent generating the sequence. For example, imagine a game in which one wins 50 Cents for every instance of "heads" in a series of coin tosses. One's goal is to win as much money as possible. This translates to throwing as much heads as possible. Thus, there is a simple relation between outcomes and the goal. Oskarsson et al. (2009) use a tennis example to describe a complex outcome-goal relationship. A tennis player likely has the ultimate goal to make points and win the match. This goal is accompanied by several subgoals, for example the subgoal to make the opponent run back and forth and exhaust him (Oskarsson et al., 2009). We can now look at the sequence of where in the court a player plays the ball (right side or left side of the opposing player's court). Which of the two sides is favorable likely changes throughout the game, thus, there is a complex relationship between the outcome and the goal. Oskarsson et al. (2009) hypothesized that for simple outcome-goal relationships, the assumption of an intentional generator behind the outcomes who also has control over the outcomes, should increase the occurrence of the hot hand in predicting the next outcome. On the other hand, a complex outcome-goal relationship should dampen the relation between intentionality, control and the hot hand.

Yet another explanation uses a perceptional approach to the gambler's fallacy. According to this explanation, the gambler's fallacy is caused by a gestalt principle in perceiving the outcomes of a random sequence. As described above, the gambler's fallacy disappears if the outcome to be predicted starts a new block compared to still being contained in the same block as the preceding run (Roney & Trick, 2003). In a recent experiment, Roney and Sansone (2015) specifically tested a gestalt explanation versus the law of small numbers as explanations of the gambler's fallacy. The critical manipulation was in the exact position of a run in a short sequence of binary outcomes (in this case a coin toss). Participants had to predict the next outcome following a series of twelve outcomes. The first eight outcomes in this sequence were identical for all participants. The last four outcomes contained a run of three outcomes, but the position of this run was manipulated between participants. For about half of the participants, the last four outcomes were tails head head head (or head tails tails tails) and thus represented an open run, whereas for the other participants it was head head head tails (or tails tails tails head) and thus represented a closed run. In addition, the occurrence of heads and tails in the outcome sequence was imbalanced. There were eight outcomes of one kind and four outcomes of the other kind with the run at the end of the outcome sequence belonging to the more common outcome. According to the law of small numbers, short coin toss sequences should reflect the overall random nature of coin tosses. Thus, the two outcomes should be expected to even out and participants should more likely predict the more seldom of the two outcomes to occur, irrespective of the run being open or closed. The gestalt explanation, on the

other hand, predicts that participants should only predict the seldom outcome more often in the open run condition, but not in the closed run condition. The results showed that participants were more likely to predict the seldom outcome, and thus break the run, when the run was open, but showed no clear preference for either outcome when the run was already closed. This result is not in line with the law of small numbers explanation of the gambler's fallacy. For the gambler's fallacy to occur, outcome runs still have to be open and ongoing.

Regarding the hot hand fallacy, there exists some evidence showing an evolutionary origin, since acting according to a hot hand belief was likely useful for the human ancestors. Wilke and Barrett (2009) proposed that a belief in the hot hand or more generally speaking a positively autocorrelated sequence is not a fallacy but originally developed as an adaptive mechanism useful for foraging, since the relevant resources (e.g. food) are clumped in time and space. Thus, a mechanism that makes it easier and more likely to detect the clumped resources would be an evolutionary advantage. Furthermore, the authors proposed the hot hand as a default mechanism, as most sequences humans had to deal with during their evolution were not truly random, but showed more or less positive autocorrelation. Random sequences, as they occur in gambling, are evolutionary more recent and were likely less important for survival, such that our perception is not geared towards correctly judging randomness. Accordingly, the correct perception of random processes first has to be learned. Wilke and Barrett (2009) showed a hot hand behavior among their participants in the prediction of actually random binary sequences in various contexts (food, birds' nests, parking lots, bus stops, coin). In a second experiment, members of a South American indigenous tribe, the Shuar (a hunter-horticulture), were compared to American college students in predicting the next outcome of a random binary sequence framed in a fruit or coin context. In both contexts, the Shuar participants showed an increased hot hand fallacy compared to the American college students. Furthermore, while the hot hand fallacy was marginally smaller in the coin context for the Americans, this was not the case in the Shuar sample. Wilke and Barrett (2009) interpreted the occurrence of the hot hand across contexts and cultures as evidence for a universal hot hand tendency in humans which can be (partly) overlearned when dealing with truly random processes.

A study by Scheibehenne, Wilke, and Todd (2011) further supports the hypothesis that humans are more geared towards perception and exploitation of runs in outcome sequences. Participants repeatedly had to put a bet on one of two slot machines. The slot machines produced random binary outcome sequences, each of the two symbols per machine having a probability of $p = .5$. The critical manipulation was the alteration rate of the symbols. One of the machines always had an alteration rate of $p(\text{alteration}) = .5$, whereas the second machine showed varying degrees of positive or negative autocorrelation (manipulated between participants). A negatively autocorrelated

sequence has an alteration rate of p(alteration) > .5 and is thus less likely to produce runs, whereas a positively autocorrelated sequence has an alteration rate of p(alteration) < .5 and is thus more likely to produce runs. Participants were instructed that one of the slot machines was random, while the other produced either positively or negatively autocorrelated sequences. The terms positive and negative autocorrelation were explained, as well as how the autocorrelation allows for better predictions. Thus, in order to maximize their gains, participants should have always bet on the autocorrelated slot machine. The results showed that the participants generally preferred the positively autocorrelated slot machines over the random machine, with the preference being stronger for stronger autocorrelation. In small and medium negative autocorrelation, however, participants preferred the random slot machine[3]. Only when the negative autocorrelation was high, participants preferred this slot machine over the random one. Altogether, participants more often chose the positively autocorrelated compared to the negatively autocorrelated slot machines. This is further evidence for an inert bias in humans to detect runs or streaks in the environment. The hot hand bias is not unique to humans, though. Blanchard and Hayden (2014) show that rhesus monkeys are better at exploiting positively autocorrelated sequences than negatively autocorrelated sequences.

Biopsychological studies directly addressing the gambler's fallacy or the hot hand fallacy are rather scarce. Xue, Lu, Levin, and Bechara (2011) examined the effect of winning or losing a gamble on subsequent risky decision making. On average, participants' choices were more risky following a loss rather than following a win. The authors interpreted this as a sign of the gambler's fallacy. Increased risk-taking following a loss versus a win correlated positively with activation in a fronto-parietal network (left inferior and middle frontal gyrus, right supramarginal gyrus, right orbitofrontal gyrus) and negatively with activation in the amygdala and striatum. Thus, Xue et al. (2011) hypothesized that the gambler's fallacy is characterized by activation in a cognitive decision making system (recruitment of frontal areas) and deactivation of an affective decision making system (amygdala). This assumption was corroborated by the findings of a second study, showing that the gambler's fallacy correlates positively with cognitive ability[4] but negatively with affective decision making ability[5], although the effects were small (cognitive ability r = .170, affective decision making ability r = -.109) (Xue, He et al., 2012). A further study by Xue, Juan, Chang, Lu, and Dong (2012) examined the contribution of the lateral prefrontal cortex (lPFC) to the gambler's fallacy. The gambler's fallacy correlated with increased activation in the left lPFC following long runs of outcomes

[3] A more recent study showed that this tendency is more pronounced in regular gamblers compared to a control group (Wilke, Scheibehenne, Gaissmaier, McCanney, & Barrett, 2014).
[4] A factor consisting of the Wechsler Adult Intelligence Scale, Raven's Advanced Progressive Matrices, a 2-back working memory task and the Stroop task
[5] A factor consisting of the Iowa Gambling Task

(run length > 3). Furthermore, anodal transcranial direct current stimulation (tDCS) over the lPFC compared to a control site (visual cortex) increased the occurrence of the gambler's fallacy following long runs of outcomes (Xue, Juan et al., 2012).

Further evidence for the involvement of the prefrontal cortex in pattern and sequence processing in general comes from a study by Huettel, Mack, and McCarthy (2002). Participants had to respond to a random series of two simple visual stimuli. The results showed increased activation in the prefrontal cortex (middle frontal gyrus, inferior frontal gyrus) and the cingulate gyrus for violations of both repeating patterns (i.e. runs in the sequence) and alternating patterns (i.e. sequences like ABABAB). Furthermore, the violation of alternating patterns showed increased activation in the insula, caudate and putamen. Akitsuki et al. (2003) also found increased activation in the dorsolateral prefrontal cortex for both wins and losses terminating a previous streak (i.e. wins occurring after a streak of losses and losses occurring after a streak of wins).

Osinsky, Mussel, and Hewig (2012) analyzed the effect of previous outcomes on the processing of the current outcome in a gambling task using EEG. They found that the FRN and the P300 were increased when the current outcome terminated a streak in the previous outcomes, that is, following a change in outcomes. Several studies have previously shown this for the P300, with the general result that the P300 is larger following an outcome or event that disrupts the current pattern of outcomes/events (e.g. Jentzsch & Sommer, 2001; Matt, Leuthold, & Sommer, 1992; Sommer, Matt, & Leuthold, 1990; Squires, Wickens, Squires, & Donchin, 1976). In this case, a pattern can refer both to a sequence of repetitions of the same event and a sequence of alterations between two events. If either pattern is disrupted, the P300 is enlarged. Similar results have been found for the FRN. When participants watched the sequential unfolding of a three-reel slot machine outcome, they showed the largest FRN for outcomes where the last symbol did not match the previous two and thus a run of symbols had been disrupted (Donkers, Nieuwenhuis, & van Boxtel, 2005; Donkers & van Boxtel, 2005). This effect could stem from the violation of the expectation of a third matching symbol, as the FRN is larger following unexpected compared to expected outcomes (Hajcak, Moser, Holroyd, & Simons, 2007; Oliveira, McDonald, & Goodman, 2007). Other studies investigating effects of recent outcome sequences on the FRN found that the FRN is only modulated by the previous outcome when the current outcome is a win (Mushtaq, Wilkie, Mon-Williams, & Schaefer, 2016), and that the FRN difference between current wins and losses is larger for previous wins compared to previous losses (Goyer, Woldorff, & Huettel, 2008).

To sum up, several factors have been shown to influence the gambler's fallacy and the hot hand fallacy. The gambler's fallacy is strongest for binary outcome sequences (Navarrete & Santamaría, 2012) caused by a random process (Burns & Corpus, 2004), with the outcomes being

presented in a sequential fashion (Barron & Leider, 2010), with longer ITIs (Militana et al., 2010) and within the same block (Roney & Trick, 2003). Furthermore, there is evidence showing the gambler's fallacy to be stronger in men compared to women (Marmurek et al., 2014, 2015; Suetens & Tyran, 2012). The hot hand fallacy, on the other hand, occurs for outcome sequences caused by a non-random process (Burns & Corpus, 2004) and is stronger if the sequence is caused by an intentional agent (Caruso et al., 2010). Several explanations for the gambler's fallacy and hot hand fallacy have been proposed, focusing on the law of small numbers (Tversky & Kahneman, 1971) and representativeness heuristic (Kahneman & Tversky, 1972), the perceived origin of the outcome sequence (e.g. Oskarsson et al., 2009), a gestalt explanation of the perception of runs (Roney & Sansone, 2015; Roney & Trick, 2003), and an evolutionary advantage of showing a hot hand bias (Wilke & Barrett, 2009). Neuroimaging studies have shown evidence for the involvement of frontal areas in the gambler's fallacy (Huettel et al., 2002; Xue, Juan et al., 2012; Xue et al., 2011). On a related topic, studies have shown that the FRN and P300 are enlarged following breaks in outcome streaks (e.g. Osinsky et al., 2012; Sommer et al., 1990; Squires et al., 1976).

Most research on the gambler's fallacy and the hot hand fallacy has been conducted with student populations or regular gamblers. So far, no study has explicitly examined the gambler's fallacy and hot hand fallacy in pathological gamblers in a behavioral study. Studies showing an increased susceptibility for the gambler's fallacy in pathological gamblers used self-report questionnaires measuring cognitive distortions in gambling, including subscales relating to the gambler's fallacy (Goodie & Fortune, 2013). Thus, the present work contains a behavioral study on the gambler's fallacy and hot hand fallacy in problem gamblers, also assessing the processing of outcome sequences as potential basis for the hot hand and gambler's fallacy.

2.5 Overview of the studies

The aim of the current studies is to examine the neural basis of an illusion of control in gambling by analyzing the processing of near outcomes in gambling, as well as examining the neural basis of the hot hand and gambler's fallacy by analyzing the processing of outcome sequences. Furthermore, all studies investigate the influence of gambling problems on the processing of the respective events.

Studies 1 to 3 were designed to investigate the processing of near outcomes in a multimodal manner. Study 1 analyzes ERPs following near and full outcomes in a sample of problem gamblers and matched controls. Study 2 uses fMRI to investigate the processing of near outcomes in a sample of participants with varying degrees of gambling problems. Finally, study 3 assesses autonomic reactions elicited by near outcomes, also in a sample with varying degrees of gambling problems.

Study 4 looks at the processing of outcome sequences to further investigate the neural basis of the hot hand and gambler's fallacy using ERPs in a sample of problem gamblers and matched controls.

3 Basic methods

The next sections give a brief overview of the biopsychological measures and questionnaires used in the four studies.

3.1 Brief overview of the biopsychological methods

3.1.1 Electroencephalogram

Via EEG, electrophysiological changes in the brain can be measured with a high temporal resolution. The following paragraphs are largely based on Luck (2005) and Cohen (2014).

In an EEG, voltage differences are recorded from the scalp. The voltage is generated by postsynaptic potentials which are caused by the release of excitatory neurotransmitters in the synaptic cleft. These neurotransmitters cause ion channels on the postsynaptic membrane to open and thus allow loaded particles to pass the membrane. This results in a current flow into the neuron. As a result, the neuron can be modelled as a small dipole, with a negativity at the apical dendrites and a positivity at the body of the neuron and the basal dendrites.

A single neuronal dipole is too weak to be measured from the scalp. Signals measurable from the scalp result from the summation of many dipoles, which in turn result from many postsynaptic potentials occurring at the same time. Furthermore, the involved neurons have to be parallel in space. Otherwise the positive and negative parts of the dipoles would cancel each other out. This requirement is met in the layers of the cortex, thus the activity measured in the EEG mainly reflects cortical postsynaptic potentials.

The signal measured in the EEG is not a steady one, but a fluctuating one. These fluctuations are called oscillations and are based on differences in the excitability of the neurons contributing to the EEG signal. The largest part of the oscillations in the EEG goes back to populations of excitatory and inhibitory neurons. As the excitatory neurons become more and more activated, nearby inhibitory neurons also become activated. The increasing activation of the inhibitory neurons in turn inhibits and decreases the activity in the excitatory neurons before the inhibitory neurons themselves decrease in activation again. The now inactive inhibitory neurons allow the excitatory neurons to become active again (Cohen, 2014). This repeating cycle causes oscillations in the EEG signal, which can be categorized according to their frequency (see section 3.1.1.2).

3.1.1.1 Event-related potentials

The EEG has a high temporal resolution, enabling the analysis of voltage changes over the course of a few tens of milliseconds. The average voltage change following an external (e.g. picture

onset) or internal (e.g. button press) stimulus is referred to as ERP. Several distinct components can be discerned in an ERP, with the exact latency and waveform depending on the location the ERP is measured at. Figure 2 shows a generic example of different ERP components.

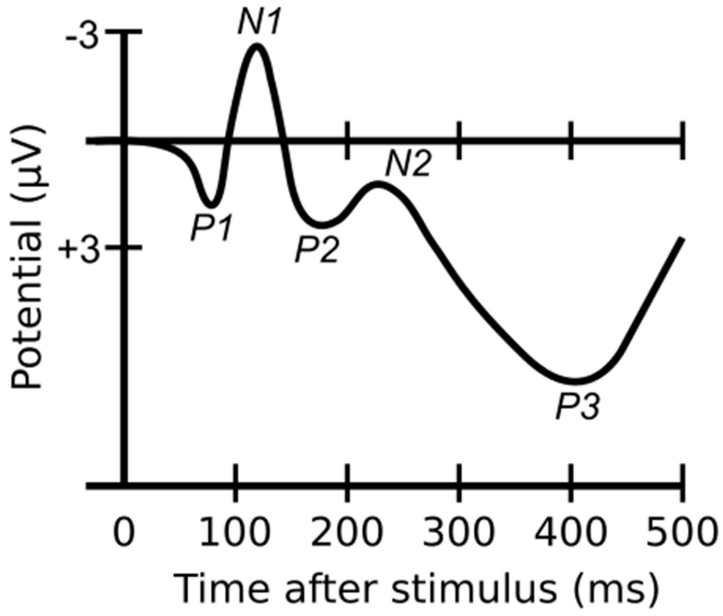

Figure 2. Generic ERP showing different components.
Source: https://commons.wikimedia.org/wiki/File:ComponentsofERP.svg
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Components in the ERP are usually labeled based on their polarity and position in time. "N" stands for a negativity in the ongoing potential, while "P" denotes a positivity. Thus, "P1" refers to the first positivity in the ERP, while "P2" refers to the second positivity in the signal and so on. Sometimes the approximate latency of the ERP component is used instead of the number denoting the position relative to other components. For example, "P200" denotes a positivity occurring about 200ms following stimulus onset.

Two components of the ERP following feedback stimuli, the FRN and the P300 will be discussed in the next sections.

3.1.1.1.1 Feedback-related negativity

The FRN is a negativity in the ERP, occurring about 200-300 ms after feedback onset and is maximal at fronto-central electrode sites. It is more pronounced for negative compared to positive feedback. The FRN has first been described by Miltner, Braun, and Coles (1997). In their study, participants had to correctly estimate time intervals of one second. Participants received auditory, visual or somatosensory feedback indicating whether they were correct or not. The threshold for an answer to be counted as correct was adjusted based on participants' performance, such that overall positive and negative feedback were given in 50% of the trials, respectively. In all feedback

modalities, negative compared to positive feedback elicited a negative deflection. The difference wave resulting from subtracting the ERP following positive feedback from the ERP following negative feedback was larger (i.e. more negative) at central compared to lateral electrode sites, and in the visual modality also larger at anterior compared to posterior sites[6].

The FRN can also be observed following losses in gambling. Gehring and Willoughby (2002) conducted a study in which participants repeatedly had to choose between two amounts of money. There were three different trials regarding the amounts to choose from: 25 Cent vs. 25 Cent, 5 Cent vs. 5 Cent and 25 Cent vs. 5 Cent. Following the choice, the participant received feedback on whether the chosen amount resulted in a gain or a loss and also whether the unchosen amount resulted in a gain or a loss. As such, four different kinds of trials could be analyzed: "gain-and-correct" (choosing 25 Cent over 5 Cent with both options resulting in a win), "loss-and-error" (choosing 25 Cent over 5 Cent with both options resulting in a loss), "gain-and-incorrect" (choosing 5 Cent over 25 Cent with both options resulting in a gain), and "loss-and-correct" (choosing 5 Cent over 25 Cent with both options resulting in a loss). The analysis of the ERPs showed a negative deflection occurring between 200-300ms after feedback onset which was more pronounced for losses compared to gains. Additional analyses showed that this ERP component reflected the overall valence of the obtained outcome (gain vs. loss) but not the correctness of the outcome (i.e.: the component did not differ significantly between the "gain-and-correct" and "gain-and-incorrect" as well as between the "loss-and-correct" and "loss-and-incorrect" conditions). However, the latter observation can likely be explained by the specific paradigm used. Gains and losses could easily be discerned based on the feedback color, while the correctness of the choice had to be determined by comparing the outcome of the chosen alternative to the outcome of the unchosen alternative. Nieuwenhuis, Yeung, Holroyd, Schurger, and Cohen (2004) showed that the FRN can also reflect the correctness of an outcome, with incorrect outcomes eliciting a larger (i.e. more negative) FRN, provided that this dimension is coded in an easily perceptible manner.

Further studies (Hajcak et al., 2007; Moser & Simons, 2009; Nieuwenhuis, Nielen, Mol, Hajcak, & Veltman, 2005; for a meta-analysis see Sambrook & Goslin, 2015) suggest that the FRN is also sensitive to the probability and the expectedness of an outcome, with low-probability or unexpected outcomes generating a larger FRN. However, the valence effect (positive vs. negative feedback, gain vs. loss) is supposed to influence the FRN more strongly than effects of probability (Hajihosseini & Holroyd, 2013).

[6] The difference waves for feedback in the auditory and somatosensory modality did not show significant differences between anterior and posterior electrode sites

Dipole analyses showed that the FRN is likely generated in the anterior cingulate cortex (ACC, Gehring & Willoughby, 2002; Miltner et al., 1997). The same region has been shown to be the generator of a similar ERP component, the error-related negativity (ERN) (Dehaene, Posner, & Tucker, 1994). The ERN is a negative deflection in the ERP peaking about 100ms after the commission of an error (Gehring, Goss, Coles, Meyer, & Donchin, 1993).

Holroyd and Coles (2002) proposed a theory that links both the ERN and FRN to phasic dopaminergic activity as part of an error-processing system. According to this model, the ERN is generated in the ACC following a disinhibition of its neurons caused by a phasic decrease in the dopaminergic activity input from the mesencephalon. This phasic decrease can be observed when an actual outcome is worse than what has been expected. Evidence for the latter comes from animal studies showing a decrease of the firing rate of dopaminergic neurons when an expected reward is not delivered (Schultz, Apicella, & Ljungberg, 1993).

Recently, a new perspective on the FRN has been proposed. Holroyd, Pakzad-Vaezi, and Krigolson (2008) have delivered evidence that the FRN is just a specific form of a more common ERP, the N200. According to the authors, The FRN/N2 is commonly elicited by all kinds of feedback. However, this default reaction to feedback is superimposed by a positive going ERP on correct trials, termed the feedback correct-related positivity. Several other studies have reported supporting evidence for this view on the FRN (e.g. Carlson, Foti, Mujica-Parodi, Harmon-Jones, & Hajcak, 2011; Foti, Weinberg, Bernat, & Proudfit, 2015; Foti, Weinberg, Dien, & Hajcak, 2011; Liu et al., 2014).

3.1.1.1.2 P300

The P300 is a positive component in the ERP occurring about 250-500 ms following a stimulus onset and is maximal at central-parietal electrode sites (Polich, 2007). It has first been reported in 1965 by two independent research groups (Desmedt, Debecker, & Manil, 1965; Sutton et al., 1965). In the series of Sutton et al.'s (1965) experiments, participants were either presented with a light or a sound which was preceded by a cuing stimulus. In one condition, the cue always correctly predicted the second stimulus, thus, participants knew beforehand whether a light or a sound would follow. In a second condition, the cue was no valid predictor, thus, participants could not be certain whether a light or a sound would follow. Sutton et al. (1965) observed a larger positivity peaking about 300 ms following the onset of the second stimulus in the uncertain compared to the certain condition. In a second experiment, two uncertain conditions were used. One cue was followed by light in two thirds of the trials and by sound in one third of the trials. This ratio was reversed for a second cue (light in one third of the trials and sound in two thirds of the trials). Participants showed a larger positive deflection around 300 ms following the stimulus occurring less often. A lot of studies have later replicated the effect of a larger P300 following rare stimuli (e.g. Duncan-Johnson & Donchin, 1977;

Friedman, Hakerem, Sutton, & Fleiss, 1973; Polich, Brock, & Geisler, 1991; Ruchkin, Sutton, & Tueting, 1975; Tueting, Sutton, & Zubin, 1970). This effect is called “oddball-effect”, referring to what has become known as the “oddball paradigm”, which features a rare target stimulus embedded in a series of another more frequently occurring stimulus (Ritter & Vaughan, 1969).

The P300 can also be observed following monetary feedback stimuli (e.g. in gambling paradigms). Here, the P300 has been shown to be influenced by the magnitude of the outcome, with larger amounts leading to an increased P300 (e.g. Kamarajan et al., 2009; Kreussel et al., 2012; Sato et al., 2005; Wu & Zhou, 2009; Yeung & Sanfey, 2004). Regarding the valence of the outcome (positive vs. negative feedback/win vs. loss), most studies showed a larger P300 following positive compared to negative feedback (e.g. Hajcak, Holroyd, Moser, & Simons, 2005; Johnson & Donchin, 1985; Kreussel et al., 2012; Martin & Potts, 2009; Osinsky et al., 2012; Ulrich & Hewig, 2014; Wu & Zhou, 2009; Zhou, Yu, & Zhou, 2010), while a couple of studies reported no valence effect on the P300 (Christie & Tata, 2009; Sato et al., 2005; Yeung & Sanfey, 2004).

The P300 can further be divided into two subcomponents, the P300a and the P300b (also referred to as P3a and P3b, respectively). As described above, the P300b is maximal at central-parietal electrode sites and peaks in the time window 250-500 ms following stimulus onset (Polich, 2007). Compared to the P300b, the P300a has a more frontal topographical distribution, with its maximum at fronto-central electrodes, and also peaks slightly earlier than the P300b (Squires, Squires, & Hillyard, 1975; for a review see Friedman, Cycowicz, & Gaeta, 2001). The P300a is elicited by novel and highly salient stimuli and can thus be observed in what has been called the “novelty oddball”, for example (Friedman et al., 2001). In this oddball variant, there is a third stimulus, next to regularly occurring non-target and irregularly occurring target stimuli. The novel stimuli are also non-targets, that is, participants don’t have to respond to them. However, they occur with the same frequency as targets and thus are rare stimuli. Following these rare non-targets, the P300a can be observed in the ERP.

Two areas generating the P300 have been identified in previous studies: the temporoparietal junction (TPJ) (e.g. Kiss, Dashieff, & Lordeon, 1989; Knight, Scabini, Woods, & Clayworth, 1989; Smith et al., 1990; Verleger, Heide, Butt, & Kömpf, 1994; Yamaguchi & Knight, 1992) and the frontal cortex (e.g. Baudena, Halgren, Heit, & Clarke, 1995; Knight, 1984; Yamaguchi & Knight, 1991). While the TPJ is involved in both the P300b and the P300a, the lPFC is mainly involved in the generation of the P300a (Nieuwenhuis, Aston-Jones, & Cohen, 2005). It has been proposed that the different neural generators are driven by an underlying system, in this case the locus coeruleus-norepinephrine system (LC-NE system). Pineda and colleagues (e.g. Pineda, Foote, & Neville, 1989; Pineda & Westerfield, 1993; Swick, Pineda, & Foote, 1994) were the first to suggest and show evidence for the

LC-NE system as the basis of the P300. This hypothesis has recently been further developed by Nieuwenhuis, Aston-Jones et al. (2005). According to this hypothesis, the P300 is based on phasic activity in the LC-NE system and the ensuing release of norepinephrine. A motivationally salient stimulus (i.e. a target stimulus) leads to a phasic increase in LC-NE activity, which in turn modulates activity in the cortical neurons, generating a larger current source in the apical dendrites of the neurons, which is visible as an enhanced P300 in the scalp EEG (Nieuwenhuis, Aston-Jones et al., 2005). From a functional perspective, the phasic LC-NE response and the ensuing P300 thus facilitate the reaction to stimuli relevant for a given task (Nieuwenhuis, Aston-Jones et al., 2005).

Other theories have linked the P300 to memory-related functions like updating of the mental model of our environment (context-updating hypothesis, Donchin, 1981; Donchin & Coles, 1988) or reactivation of learned stimulus-response links (e.g. Verleger, Baur, Metzner, & Śmigasiewicz, 2014; Verleger, Hamann, Asanowicz, & Śmigasiewicz, 2015).

3.1.1.2 Time-frequency approach

As mentioned above, the signal measured in the EEG is an oscillating one. It can be decomposed into components of different frequencies. Major frequency components in the EEG are the delta (2-4 Hz), theta (4-8 Hz), alpha (8-12 Hz), beta (15-30 Hz) and gamma (30-150 Hz) bands (Cohen, 2014). The boundaries for these frequency bands are not clear-cut and thus may vary slightly from study to study (Cohen, 2014). Using time-frequency analyses, the power in different frequency bands can be analyzed over time (similar to ERPs which show the voltage over time).

3.1.1.2.1 Theta

EEG oscillations with a frequency of 4-8 Hz are referred to as theta oscillations. Theta power has been linked to various cognitive processes like memory formation (Rutishauser, Ross, Mamelak, & Schuman, 2010), working memory (Itthipuripat, Wessel, & Aron, 2013; Raghavachari et al., 2001), recognition (Jacobs, Hwang, Curran, & Kahana, 2006), and cognitive control (Cavanagh & Frank, 2014; Cavanagh, Zambrano-Vazquez, & Allen, 2012). Although task-related theta increase has been shown in various cortical areas (Jacobs et al., 2006; Raghavachari et al., 2001; Raghavachari et al., 2006), midfrontal theta is of special interest for the current work, due to its relation to feedback processing. Previous studies on the ERN and FRN have linked these ERPs to activity in the theta frequency band (Bernat, Williams, & Gehring, 2005; Cohen, Elger, & Ranganath, 2007; Luu, Tucker, Derryberry, Reed, & Poulsen, 2003; Trujillo & Allen, 2007). Accordingly, studies have shown that increased midfrontal theta power is associated with negative or unexpected feedback (e.g. Cavanagh, Figueroa, Cohen, & Frank, 2012; Cavanagh, Zambrano-Vazquez et al., 2012; Cohen, Elger, & Fell, 2009; Cohen et al., 2007; Hajihosseini & Holroyd, 2013).

3.1.2 Functional magnetic resonance imaging

While EEG has a high temporal resolution, it does not provide an equally good spatial resolution of brain activity. Better spatial resolution can be achieved using fMRI, which in turn does not have as much temporal resolution as EEG. The following paragraphs are largely based on Huettel, Song, and McCarthy (2008).

As indicated by its name, magnetic resonance imaging (MRI) uses strong magnetic fields and impulses to acquire images. Image acquisition of human tissues, such as brain tissue, mostly operates on the spin of hydrogen protons. These protons are also called spins, since they possess the nuclear magnetic resonance property. This property consists of two momentums - a magnetic and an angular momentum. Under normal conditions, the axes of the spinning protons are all aligned randomly, thus the overall magnetization of the spin system is near zero. However, if those protons are placed in an external magnetic field, for example in an MRI scanner, their spin axes start a circular movement, which is called precession. Some of the spins are aligned parallel to the magnetic field (low-energy state), some antiparallel (high-energy state). The net magnetization of the system depends on the number of spins in the parallel and antiparallel states. To measure a signal in MRI, a high frequency excitation pulse is used, causing spins in the low-energy parallel state to change to the high-energy antiparallel state. Once the excitation pulse stops, the spins gradually return to their previous low-energy state, thereby emitting photons, which can be detected using radiofrequency coils. The time it takes for the spins to return to their original state are different for different types of tissues, thus MRI can be used to separate different kinds of tissues (e.g. gray and white matter) on an image.

While the just described properties make it possible to gather structural images, another property is exploited to take functional images of the brain. Functional images depend on the blood-oxygenation-level dependent (BOLD) contrast. In short, blood has different magnetic properties, depending on whether it is oxygenated or not, with deoxygenated blood showing a greater magnetic susceptibility. This leads to a stronger MR signal in brain areas with oxygenated blood compared to deoxygenated blood. However, using the BOLD contrast, we cannot measure brain activity directly, but rather measure a correlate of brain activity. When there is neuronal activity in a brain area, more oxygenated blood is transported there. The amount of oxygenated blood is higher than the amount actually needed, thus leading to an overall increase of oxygenated relative to deoxygenated blood in the active area. Based on the BOLD contrast, this brain area now shows a stronger MR signal than surrounding, non-active areas. The typical change in the MR signal following activation in a brain area is referred to as hemodynamic response.

3.1.3 Heart rate and heart period

Using electrocardiography (ECG), we can record the electrical activation generated throughout the cardiac cycle (i.e. the time between two successive heart beats; Berntson, Quigley, & Lozano, 2007). For a basic ECG recording, three electrodes are placed on the limbs of a person according to what is called “Einthoven’s triangle” (Berntson et al., 2007). This means placing one electrode each on the left and right arm of the person and the third electrode on the left leg. Instead of placing the electrodes on the extremities, they can also be placed on the torso near the extremities (e.g. below the left and right collarbone and under the rib cage on the left side of the torso). The normal ECG shows several features which mark distinct events throughout the cardiac cycle (see Figure 3).

The first deflection, the P-wave, is caused by the spread of excitation from the sinoatrial node to the atria of the heart (Berntson et al., 2007). The second prominent deflection, the QRS-complex, goes back to the depolarization of the ventricles, while the third deflection, the T-wave, reflects the repolarization of the ventricles (Berntson et al., 2007).

The time between two successive r-waves is referred to as heart period and is usually measured in milliseconds. Heart period can be transformed to heart rate by the following formula: 60000/heart period. Both measures have been used in psychophysiological research, although Berntson, Cacioppo, and Quigley (1995) have suggested heart period as the preferred measure, since the effects of autonomic innervation are more linear for heart period than for heart rate, and heart period allows for more flexibility, as it can be analyzed both in real time (seconds) as well as in cardiac time (beats).

Heart rate and heart period are influenced by both sympathetic and parasympathetic branches of the autonomic nervous system, although for short-term heart period responses the parasympathetic influence is larger (Berntson et al., 2007).

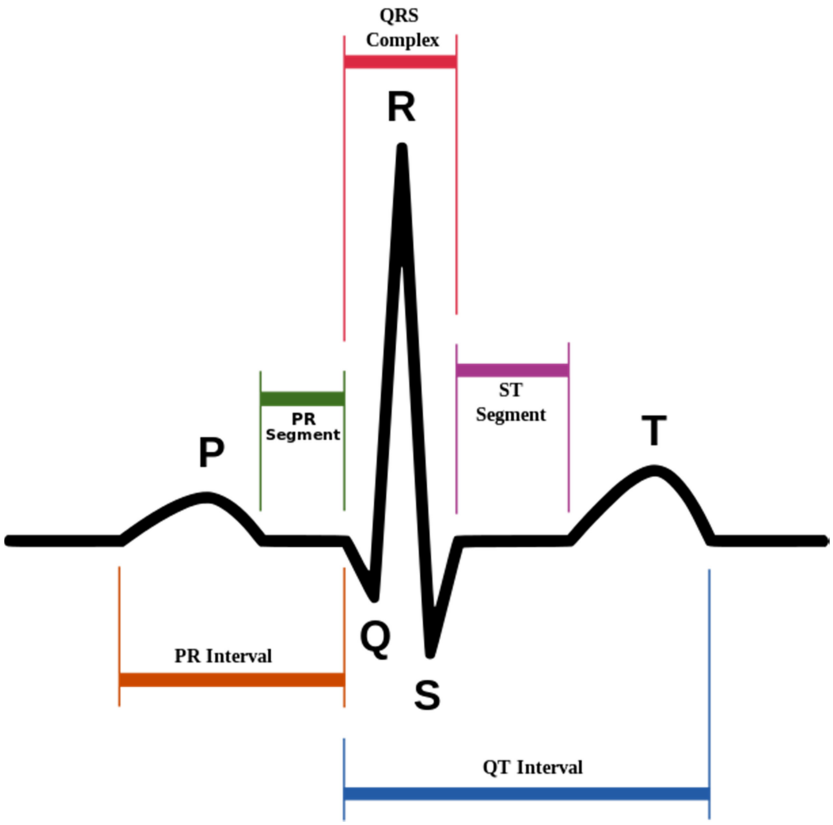

Figure 3. Sample cardiac cycle in a normal ECG.
Source: https://en.wikipedia.org/wiki/File:SinusRhythmLabels.svg

3.1.4 Electrodermal activity

Electrodermal activity (EDA) refers to electric properties of the skin. Depending on the exact measurement used, different measures of EDA can be derived (see Boucsein, 2012 for an overview). The most commonly used measure, skin conductance, uses an exosomatic measurement, applying direct current to the skin using a pair of electrodes usually placed on the hand. The current flow between the two electrodes is measured, from which the skin conductance can be calculated given the constant voltage applied to the electrodes (Dawson, Schell, & Filion, 2007). Figure 4 shows a sample segment from a skin conductance recording. The prominent phasic signal change occurring around 2.5 seconds is called skin conductance response (SCR), while the tonic level of the signal is referred to as skin conductance level (SCL). Several components and measures can be distinguished with regard to an SCR. *Latency* refers to the time between stimulus onset and the onset of the ensuing SCR. *Rise time* refers to the time between onset of the SCR and the peak of the SCR. *Amplitude* refers to the size of the SCR. Finally, *half recovery time* denotes the time between the peak of the SCR and the time point where the signal has returned to half of the peak value. A further distinction can be made between SCR amplitude and magnitude, when averaging SCRs. SCR amplitude uses only the trials in which an SCR has been elicited, while the SCR magnitude includes all available trials, including those with a zero-response.

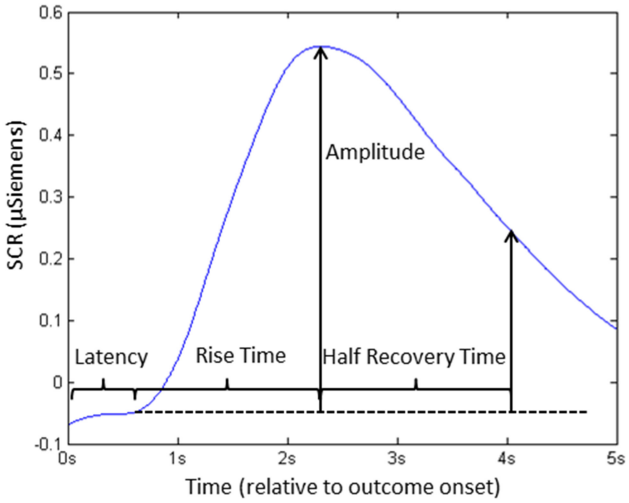

Figure 4. Sample SCR.

Changes in skin conductance are caused by changes in the sweat glands. EDA measurement focuses on eccrine sweat glands, which are thought to react more strongly to psychological than to thermal stimulation (Dawson et al., 2007). Depending on the level of stimulation, there is more or less sweat in the sweat ducts, which in turn causes changes in skin conductance, with more sweat leading to a higher conductance. Sweat glands are mostly innervated by the sympathetic nervous system, more specifically by sudomotor fibers. Thus, skin conductance and SCRs can be used as a measure of sympathetic activity, since SCRs are caused by sudomotor nerve bursts (Benedek & Kaernbach, 2010).

3.2 Questionnaires

Several questionnaires were used repeatedly in the following studies, thus they will be introduced briefly in the following section.

3.2.1 Kurzfragebogen zum Glücksspielverhalten (KFG)

The KFG (Petry, 1996) is a screening questionnaire to assess gambling-related problems. It consists of 20 items which are answered on a 4-point scale (ranging from 0 = “trifft gar nicht zu”/”does not apply at all”, to 3 = “trifft genau zu”/”applies exactly”). The questionnaire contains a broad single scale, consisting of the sum of the item scores, assessing various aspects of problematic gambling behavior (e.g. not being able to control ones gambling behavior, problems at work due to gambling, financial problems due to gambling). A cutoff score of 16 has been proposed and shown to be able to discriminate between pathological and non-pathological gamblers (Petry, 1996).

3.2.2 South Oaks Gambling Screen (SOGS)

The SOGS (Lesieur & Blume, 1987) is another widely used screening questionnaire to assess problematic gambling behavior. It consists of 16 questions, two of which are split up into sub-questions. The questions assess gambling frequency for different kinds of gambling games, money spent on gambling, gambling problems in close friends and relatives, problematic gambling behavior (e.g. loss chasing, playing longer than intended) and borrowing money from different sources in order to gamble. The questions are answered on different scales, with the majority using a dichotomous "yes"/"no" format. A sum score is calculated based on the scoring key provided by Lesieur and Blume (1987). A sum score of 5 or more is indicative of probable pathological gambling (Lesieur & Blume, 1987).

3.2.3 Gambling Related Cognitions Scale (GRCS)

The GRCS (Raylu & Oei, 2004) consists of 23 items which are answered on a 7-point scale (ranging from 1 = "starke Ablehnung"/"strong rejection", to 7 = "starke Zustimmung"/"strong agreement"). The questionnaire assesses several cognitive distortions related to gambling problems. There is one subscale each for the following distortions: "Gambling Expectancies" (4 items; refers to beliefs about the effects of gambling), "Illusion of Control" (4 items; refers to beliefs in being able to influence gambling outcomes by employing specific strategies), "Predictive Control" (6 items; refers to beliefs in being able to predict future gambling outcomes), "Inability to Stop Gambling" (5 items; refers to beliefs in not being able to stop gambling), and "Interpretative Bias" (4 items; refers to ways of interpreting previous gambling outcomes which make the person continue gambling). In addition to mean scores for every subscale, a total sum score can be computed by adding the mean scores of the subscales (Raylu & Oei, 2004).

3.2.4 UPPS Impulsive Behavior Scale (UPPS)

The UPPS (Keye, Wilhelm, & Oberauer, 2009; Whiteside & Lynam, 2001) contains 45 items measuring four different aspects of impulsivity. The responses are given on a 5-point scale (ranging from 1 = "Stimmt gar nicht"/"Is not true at all" to 5 = "Stimmt genau"/"Completely true"). The items are grouped into the four subscales "Premediation", "Urgency", "Sensation Seeking", and "Perseverance". "Premediation" (11 items) assesses to what extent a person carefully plans their actions. "Urgency" (12 items) refers to difficulties in controlling impulses. "Sensation Seeking" (12 items) refers to seeking out and enjoying exciting and risky situations and activities. "Perseverance" (10 items) measures the extent to which a person can stick with unfinished tasks.

3.2.5 Achievement Motives Scale (AMS)

The AMS (Dahme, Jungnickel, & Rathje, 1993; Gjesme & Nygard, 1970; Göttert & Kuhl, 1980) is a 30-item questionnaire consisting of two scales measuring “Hope of Success” and “Fear of Failure”, respectively. Each of the scales consists of 15 items which are answered on a 4-point scale (ranging from 1 = ”is not at all true of me”/”trifft auf mich überhaupt nicht zu” to 4 = ”is very true of me”/”trifft genau auf mich zu”). The subscale “Hope of Success” assesses positive affect in performance situations, whereas the subscale “Fear of Failure” assesses negative affect in performance situations. A “net-hope” scale can be calculated (“Hope of Success” minus “Fear of Failure”) to characterize a person as motivated by success or motivated by the avoidance of failure.

3.2.6 Belief in Good Luck Scale (BIGL)

The BIGL (Darke & Freedman, 1997b) comprises 12 items assessing the extent to which a person beliefs in good luck as an internal and stable trait. The items are answered on a 6-point scale (ranging from 1 = ”strongly disagree”/”stimme überhaupt nicht zu” to 6 = ”strongly agree”/”stimme völlig zu”). A sum score across all items is computed to index the belief in good luck.

3.2.7 Domain-Specific Risk-Taking Scale (DOSPERT)

The DOSPERT (Johnson, Wilke, & Weber, 2004; Weber, Blais, & Betz, 2002) consists of 40 items that can be combined with different instructions to collect self-report measures on either risky behavior, perception of risk, or expected benefits of risky behavior. In the current studies, we used the instructions to measure risky behavior and perception of risk. For risky behavior, participants have to indicate how likely they are to show a certain behavior. For risk perception, they have to indicate how risky they perceive a certain behavior. The German DOSPERT version consists of six subscales. The subscale “Ethical” (8 items) describes situations like breaking the law or morally questionable actions. The subscale “Investment” (4 items) describes situations related to investing ones income. Together with the subscale “Gambling” (4 items), which describes situations where one uses his income for gambling, the “Investment” subscale forms the “Financial” subscale in the English version. The subscale “Health” (8 items) contains situations involving health and safety risks. The subscale “Recreational” (8 items) describes risky recreational activities. The subscale “Social” (8 items) contains risky social situations.

Table 4 shows sample items for each of the questionnaires and corresponding subscales.

Table 4. Sample items of the questionnaires used in the current studies

Questionnaire	Subscale	Sample Item (German translations)
KFG		„Ich habe meistens gespielt, um Verluste wieder auszugleichen“
SOGS		„Haben Sie schon jemals behauptet, Geld beim Glücksspiel gewonnen zu haben, obwohl dies nicht der Wahrheit entsprach? Tatsächlich haben Sie verloren?“
GRCS	Gambling Expectancies	„Glücksspiel macht mich glücklicher.”
	Illusion of Control	„Ich habe spezielle Rituale und Verhaltensweisen, die meine Gewinnchancen erhöhen.”
	Predictive Control	„Ich habe einige Fähigkeit meine Gewinne vorherzusagen.“
	Inability to Stop Gambling	„Ich bin nicht stark genug, um mit dem Spiele auf zu hören.“
	Interpretative Bias	„Meine Verluste auf die Wahrscheinlichkeit zurückzuführen, ermuntert mich weiter zu spielen.“
UPPS	Premediation	„Ich gehöre nicht zu den Leuten, die mit Dingen herausplatzen ohne vorher darüber nachzudenken“
	Urgency	„Ich habe Schwierigkeiten meine Impulse zu kontrollieren.“
	Sensation Seeking	„Im Allgemeinen suche ich nach neuen und aufregenden Erfahrungen und Empfindungen“
	Perseverance	„Wenn ich einmal mit einer bestimmten Sache begonnen habe, hasse ich es damit aufzuhören.“
AMS	Hope of Success	„Ich mag Situationen, in denen ich feststellen kann wie gut ich bin.“
AMS	Fear of Failure	„Situationen, in denen meine Fähigkeiten auf die Probe gestellt werden, mag ich nicht.“
BIGL		„Ich betrachte mich selbst als eine Person, die Glück hat.”
DOSPERT	Ethical	„… illegal Software zu kopieren?“
	Investment	„… 5% Ihres Jahreseinkommens in eine sehr spekulative Aktie investieren?”
	Gambling	„… das Einkommen einer Woche im Casino verspielen?“
	Health	„… ohne Helm Motorrad fahren?“

Questionnaire	Subscale	Sample Item (German translations)
	Recreational	„… einen Tornado mit dem Auto verfolgen, um dramatische Bilder machen zu können?“
	Social	„… bei einem wichtigen Thema anderer Meinung sein als Ihr Vater?“

4 Study 1: Processing of near outcomes in gambling (EEG)

4.1 Introduction

The first study focused on the processing of near outcomes and its potential modulation by gambling problems. In this study, processing of near outcomes was assessed using EEG. More specifically, two feedback-related ERPs (FRN and P300), as well as event-related changes in theta power were analyzed. For an introduction to EEG, FRN, P300 and theta power, see section 3.1.1. The influence of gambling problems was assessed by comparing a group of problem gamblers to a group of control participants.

4.1.1 Summary of previous results

As described in the introduction, previous research on the FRN and P300 following near outcomes has yielded mixed results. Some studies found a smaller (i.e. less negative) FRN following near compared to full misses (Lole et al., 2015; Lole et al., 2013; Luo et al., 2011), while others have found a larger (i.e. more negative) FRN following near misses (Kreussel et al., 2013; Ulrich & Hewig, 2014) or no difference between near and full misses regarding FRN (Qi et al., 2011). Results for the P300 show a similar heterogeneity, with some studies finding no difference between near and full misses (Lole et al., 2015; Lole et al., 2013; Luo et al., 2011) and others finding either a smaller (Ulrich & Hewig, 2014) or larger P300 (Qi et al., 2011) following near compared to full misses.

Concerning theta power, two studies have reported increased theta power for near compared to full misses in the EEG (Alicart et al., 2015) and MEG (Dymond et al., 2014).

Evidence of a modulation of near miss processing, as indexed by ERPs, by gambling problems is mixed. While Kreussel et al. (2013) found differences between pathological gamblers and controls in the closeness effect on the FRN, with pathological gamblers showing no FRN difference between near and full misses and controls showing a more negative FRN to near misses, Lole et al. (2015) found no such differences.

As explained in the introduction, previous research has almost exclusively focused on the processing of wins, near misses and full misses, thus creating possible probability confounds in the P300 results, since the probabilities of wins versus misses and near versus full outcomes cannot be balanced using only these three outcomes. Hence, the wheel of fortune paradigm described in Ulrich and Hewig (2014) was used in the current study to assess the processing of near outcomes in general and its potential modulation by gambling problems.

4.1.2 Pathological gambling and personality

As explained in section 2.4, pathological and problem gamblers show more cognitive distortions related to gambling than non-pathological gamblers (Cunningham et al., 2014; Joukhador et al., 2004; Joukhador et al., 2003; MacLaren et al., 2015; Myrseth et al., 2010; Xian et al., 2008). These cognitive distortions also include beliefs about luck (Toneatto, 1999). Wagenaar and Keren (1988) showed that people distinguish between chance and luck, concerning the cause of events. The extent to which people believe in good luck can be measured by the BIGL (Darke & Freedman, 1997b), for example. Belief in good luck has been shown to influence decision making in gambling. For instance, the betting behavior of people who attribute events to luck versus chance is influenced by previous outcomes of the gamble, while the betting behavior of people of who attribute events to chance versus luck is independent of previous outcomes (Friedland, 1998). Darke and Freedman (1997a) showed that experiencing a lucky event affects high and low believers in good luck differently, with high believers becoming more confident and less risk averse in subsequent tasks, while low believers showed the opposite pattern. Furthermore, previous research has shown that belief in luck is stronger in pathological gamblers relative to non-pathological gamblers (Chiu & Storm, 2010; Wohl, Young, & Hart, 2007).

Pathological gambling has first been included in the *DSM-III* as an impulse control disorder and remained in this section until *DSM-5*. This classification suggests that pathological gamblers should also show increased impulsivity. Several studies have provided corresponding evidence. Compared to controls, pathological gamblers show increased impulsivity measured via behavioral tasks (e.g. Albein-Urios, Martinez-González, Lozano, & Verdejo-Garcia, 2014; Kräplin et al., 2014; Lawrence et al., 2009b; Michalczuk, Bowden-Jones, Verdejo-Garcia, & Clark, 2011; Petry, 2001; Vitaro, Arseneault, & Tremblay, 1999) and trait questionnaires (e.g. Barrault & Varescon, 2013; Kräplin et al., 2014; Lawrence et al., 2009a; Michalczuk et al., 2011; Vitaro et al., 1999). Furthermore, impulsivity correlates positively with symptom severity in pathological gamblers (Alessi & Petry, 2003; Blaszczynski, Steel, & McConaghy, 1997; Steel & Blaszczynski, 1998).

A personality trait which has rarely been investigated in relation to gambling problems is achievement motivation. Achievement motivation can be defined as a "stable disposition to strive for achievement or success" (Atkinson, 1957, p. 359). McClelland, Atkinson, Clark, and Lowell (1953) defined achievement motivation "in terms of affect in connection with evaluated performance" (p. 79). Depending on whether this affect is negative or positive, one can further distinguish between "Fear of Failure" and "Hope of Success" (sometimes also referred to simply as achievement motivation), as measured by the AMS (Dahme et al., 1993; Gjesme & Nygard, 1970; Göttert & Kuhl, 1980). Atkinson (1957) showed that people who are relatively higher in achievement motivation than

fear of failure chose medium risky bets compared to very risky or safe bets more often than people who are relatively higher in fear of failure than achievement motivation. Since gambling usually is a high risk activity, it could be hypothesized that pathological gamblers show an increased fear of failure. However, it is also conceivable that pathological gamblers are actually more achievement motivated and perceive gambling as only moderately risky, due to their alleged skill in gambling. Previous research so far does not support either hypothesis, as there have been no differences between pathological gamblers and controls (Brand et al., 2005) and youths with different amounts of gambling problems (Dickson, Derevensky, & Gupta, 2008) in terms of achievement motivation.

4.1.3 Current study

The aims of the current study were to analyze the processing and evaluation of near outcomes in a sample of problem gamblers and healthy controls. Outcome processing was analyzed via ERPs and time-frequency analysis, while outcome evaluation was assessed using rating data. Furthermore, self-reported cognitive distortions, impulsivity and achievement motivation were assessed and compared between problem gamblers and controls. Finally, choice behavior was analyzed in an exploratory manner to test for gambler's fallacy tendencies.

4.1.4 Hypotheses

Based on our previous results (Ulrich & Hewig, 2014) and the results of Kreussel et al. (2013) we expected near outcomes to elicit an increased (i.e. more negative) FRN and a smaller P300 compared to full outcomes in the control group. Based on the results by Kreussel et al. (2013), we expected problem gamblers to show no difference in the FRN following near versus full misses. Concerning near and full wins we expected problem gamblers to perceive near wins similar to a positive outcome following a risky decision. Such outcomes have been shown to lead to an increased positivity in the FRN time frame in problem gamblers (Hewig et al., 2010), hence a smaller (i.e. less negative) FRN following near compared to full wins was expected for problem gamblers. Concerning P300, problem gamblers were also expected to show smaller amplitudes to near versus full outcomes. Furthermore, based on previous results (Lole et al., 2015; Oberg et al., 2011), problem gamblers were expected to show overall reduced P300 levels.

Concerning the rating data, more negative valence but higher motivation ratings were expected for near compared to full misses in the control group (Clark et al., 2012; Clark et al., 2009; Qi et al., 2011). Problem gamblers were expected to rate near misses as more positive and motivating than control participants, based on the skill acquisition component inherent to near misses (Clark et al., 2009) and previous studies showing increased cognitive distortions related to gambling in pathological and problem gamblers (e.g. Cunningham et al., 2014; Joukhador et al., 2004;

Myrseth et al., 2010). Furthermore, related to the increased cognitive distortions, problem gamblers were also expected to generally rate their chances of winning and control over the outcome higher than the control participants. For the arousal ratings, increased ratings were expected for near compared to full outcomes, based on increased physiological arousal observed following near misses (Clark et al., 2012; Dixon et al., 2011). Problem gamblers were also expected to report generally higher subjective arousal than controls, based on studies showing increased physiological arousal in pathological gamblers compared to controls in gambling situations (Blanchard, Wulfert, Freidenberg, & Malta, 2000; Carroll & Huxley, 1994; Sharpe, Tarrier, Schotte, & Spence, 1995).

Based on previous research, the following hypotheses were derived for personality differences between problem gamblers and controls: Problem gamblers were expected to score higher on a questionnaire measuring general cognitive distortions in gambling (e.g. Cunningham et al., 2014; Joukhador et al., 2004; Myrseth et al., 2010; Raylu & Oei, 2004), a questionnaire measuring belief in good luck as personality trait (Chiu & Storm, 2010; Wohl et al., 2007) and a questionnaire assessing impulsivity (e.g. Barrault & Varescon, 2013; Kräplin et al., 2014; Lawrence et al., 2009a; Petry, 2001; Steel & Blaszczynski, 1998). Furthermore, differences in achievement motivation between problem gamblers and controls were analyzed in an exploratory manner.

4.2 Methods

4.2.1 Participants

Participants were recruited via online advertisement on a local website. The advertisement contained the link to an online questionnaire which every potential participant had to fill in. The questionnaire consisted of demographic questions, as well as the KFG (Petry, 1996). Potential participants scoring at or above the cutoff score (16 points) were invited for an interview session, during which *DSM-IV-TR* A-criteria for pathological gambling were assessed using a German version of a structured interview for pathological gambling (SCID PG, Grant, Steinberg, Kim, Rounsaville, & Potenza, 2004). Furthermore a screening for other mental disorders was conducted (Diagnostisches Kurz-Interview bei psychischen Störungen, Mini-DIPS, Margraf, 1994), and additional questionnaires were filled in by the participants (SOGS, Lesieur & Blume, 1987; GRCS, Raylu & Oei, 2004; UPPS, Keye et al., 2009; AMS, Dahme et al., 1993; BIGL, Darke & Freedman, 1997b). Based on the combined results of the KFG, the SCID PG and the SOGS score, participants were invited to the main part of the study. Two groups of participants were recruited: problem gamblers (PG), who show signs of gambling problems but not to the extent of being diagnosed with pathological gambling (Raylu & Oei, 2002; Whelan et al., 2007) and a control group (nonPG). Participants were included in the PG group if they scored above the respective cutoffs in both the KFG and the SOGS and if they fulfilled at least 2

DSM-IV-TR A-criteria. Participants in the nonPG group were matched to PGs with respect to gender, age, handedness and education as far as possible. Furthermore, nonPG participants had to score below the respective cut off points in both screenings, as well as satisfying less than 2 *DSM-IV-TR* A-criteria. The final sample consisted of 20 PG participants and 20 nonPG participants. Table 5 gives an overview of relevant sample characteristics. All participants received a reimbursement of 18.50 € for taking part in the experiment, which consisted of a fixed reimbursement of 10.00 € and an additional 8.50 € from playing the wheel of fortune and the coin toss paradigms (see below).

Table 5. Demographic and gambling-related data, as well as comorbidities for the sample used in studies 1 and 4

	Problem Gamblers (PG)	Control Group (nonPG)
Group Size	20	20
Age	25.70 (5.72)	25.10 (5.78)
Sex (♂/♀)	18/2	18/2
Handedness (right/left)	19/1	20/0
Education		
„Hauptschulabschluss“	2	1
„Realschulabschluss/Mittlere Reife“	1	0
„(Fach-)Abitur“	12	13
„Berufsausbildung“	3	3
„Hochschulabschluss“	2	3
KFG	29.45 (8.26)	3.20 (3.16)
SOGS	9.10 (3.16)	0.75 (0.97)
DSM-IV-TR A-criteria	3.50 (1.61)	0.20 (0.41)
Comorbidity (Frequency)		
Panic Disorder	1	-
Social Anxiety Disorder	-	1
Specific Phobia	3	1
Generalized Anxiety Disorder	-	1
Post-Traumatic Stress Disorder	1	-
Bipolar Disorder	1	-
Major Depression	1	-
Dysthymia	-	1
Manic Episodes	2	-
(past) Substance Dependence	2	-
Psychosis (Screen)	1	1

Note. Except for group size, sex, handedness and comorbidities, mean values are given. *SD* values are given in parentheses.

4.2.2 Paradigm: Wheel of fortune

The gambling game used in this study consisted of a simple wheel of fortune, adapted from the one used in Ulrich and Hewig (2014). The wheel was shown on a black background and consisted of 8 segments, each spanning 45° and alternately colored orange and turquoise. The wheel spanned a visual angle of about 4.2°. It was programmed and presented via Presentation software (Version 17.1; Neurobehavioral Systems).

On each trial participants chose the color they wanted to bet on, with the bet being fixed to 10 Cents. After the color choice the wheel started spinning[7] and participants had to press the space bar to initiate the stopping of the wheel. The wheel then continued spinning for a variable amount of time (average time until space bar press: 1.18 s, *SD* = 1.18 s; average time between space bar press and wheel stop 1.04 s, *SD* = 0.60 s) before it abruptly stopped. Finally, the outcome of the current trial (+/- 10 Cents) was displayed (see Figure 5 for a sample trial). In one fourth of the trials (distributed evenly across the different outcomes), four rating questions followed, in which the participants had to assess the valence of the outcome ("How pleased are you with the outcome?"), the motivational impact of the outcome ("How motivated are you to continue gambling?"), the arousal elicited by the outcome ("How exciting was the outcome?") and the probability of winning in the next trial ("How likely do you think you will win in the next trial?"). Valence, motivation and arousal ratings were given on 7-point bipolar likert scales, probability ratings were given on an 11-point scale ranging from 0% to 100% in steps of 10%. Participants gambled for 137 trials, with the first three trials being practice trials which were excluded for the analysis. The last six trials consisted of a fixed sequence of outcomes to ensure that participants ended the gamble with a slightly larger amount than they started with, to make them less suspicious about predetermined outcomes in the gamble. These trials were also excluded from the analysis. The remaining 128 trials consisted of 32 full wins, 32 full misses, 32 near wins and 32 near misses. For the full outcomes, the wheel stopped 20° or 25° from the boundary of the next color field. For the near outcomes, the wheel stopped 5° from the boundary of the previous or the next color field (see Figure 6 for examples). After 64 trials participants could take a pause of self-determined length. Participants started with 400 Cents in a virtual account and were instructed that the final balance of the account would be added to their fixed payment of 10.00 €. Since the sequence of the end trials was fixed and wins and losses occurred equally often in the main part of the paradigm, every participant ended the game with 420 Cents in their account. After the game was finished, but before the final account balance was shown,

[7] The spinning motion was created by presenting pictures of the wheel turned by 5° relative to the previous one. Each picture was presented for one frame, corresponding to 16.67 ms at a monitor refresh rate of 60 Hz.

participants were asked to rate their perceived control over the outcomes of the wheel spin on a 7-point bipolar likert scale ranging from “no control at all” to “total control”.

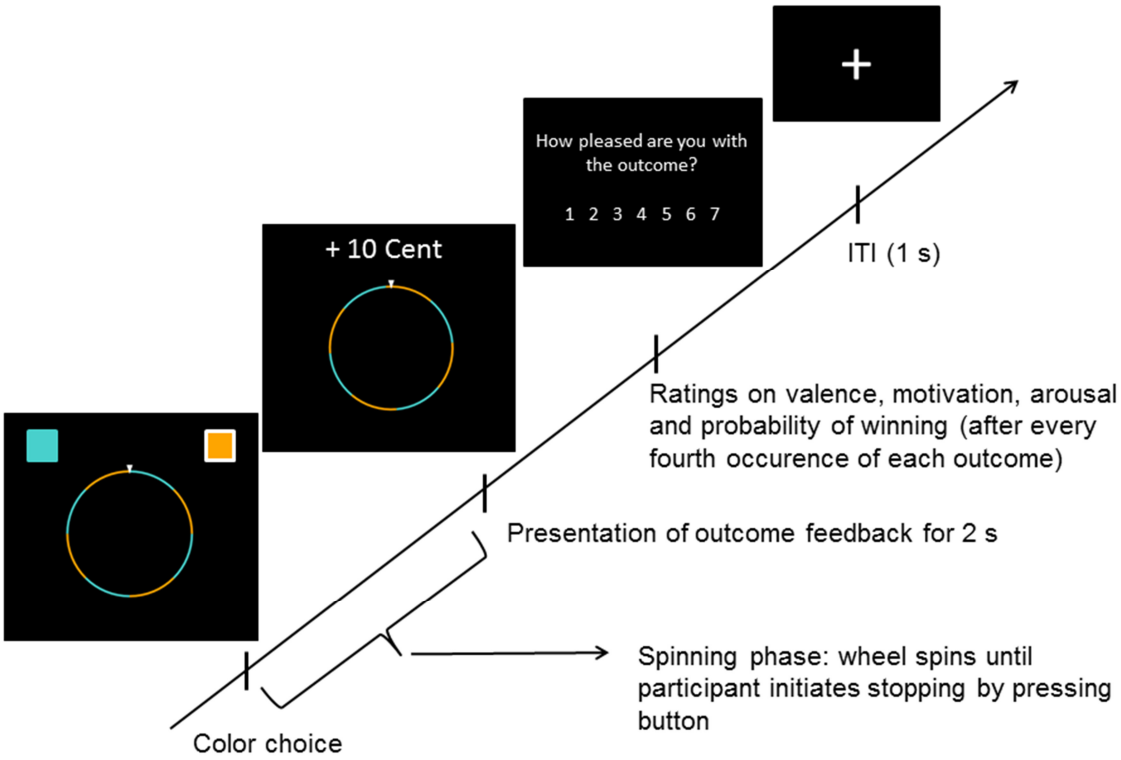

Figure 5. Study 1: Sample trial of the wheel of fortune used in the EEG study.

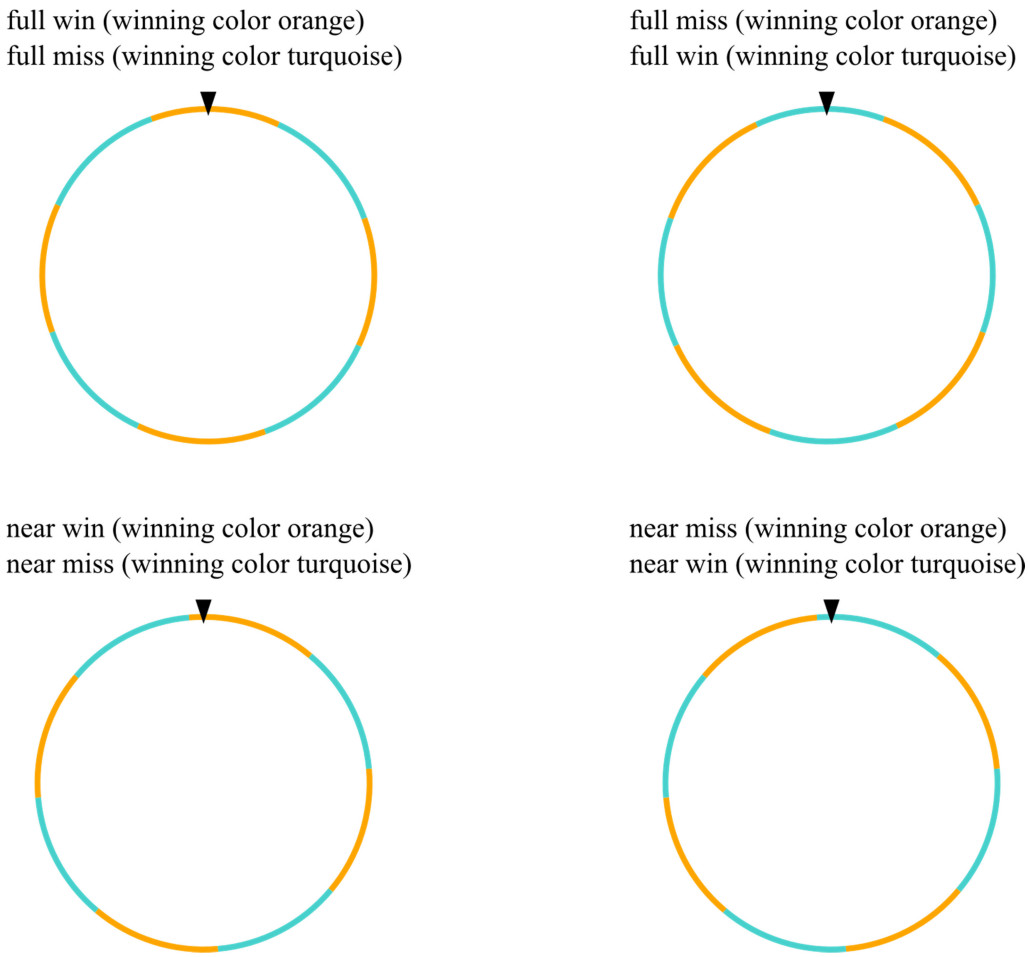

Figure 6. Study 1: Examples of the four different outcome types in the wheel of fortune.

4.2.3 Questionnaires

As mentioned above, participants filled in several questionnaires during the online survey and the interview. The KFG (Petry, 1996) and SOGS (Lesieur & Blume, 1987) were used to assess gambling problems. Gambling-related cognitive distortions were measures via the GRCS (Raylu & Oei, 2004) and the BIGL (Darke & Freedman, 1997b). The UPPS (Keye et al., 2009) was used to assess impulsivity, while the AMS (Dahme et al., 1993) was used to measure achievement motivation. Further information on the questionnaires can be found in section 3.2.

4.2.4 Procedure

Participants gave informed consent after having read information on the experiment and the procedure. Next, the EEG was prepared and participants were seated in an electrically shielded chamber, where they played the wheel of fortune and the coin toss paradigm (see below). 30 participants (15 PG and 15 nonPG) started with the wheel of fortune, while 10 (5 PG and 5 nonPG) started with the coin toss. In the break between the two games, EEG electrode impedances were checked and corrected if necessary.

4.2.5 EEG recordings and quantification

The EEG was recorded using 32 Ag/AgCl electrodes placed according to the 10-20 system (Jasper, 1958), including an electrode below the left eye to correct for blinks and eye movements (electrode positions: Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T7, T8, P7, P8, Fz, FCz, Pz, FC1, FC2, CP1, CP2, FC5, FC6, F9, F10, TP9, TP10, PO9, PO10, IO). The ground electrode was placed at AFz and Cz was used as online reference. The EEG signal was amplified using a BrainAmp DC amplifier (Brain Products GmbH, Gilching, Germany) and the BrainVision Recorder 1.20 (Brain Products GmbH) was used to record the signal with a sampling rate of 250 Hz and an online high-pass filter of 0.016 Hz (10 s), as well as a low-pass filter of 250 Hz. Electrode impedances were kept below 5 kΩ. For offline data processing the BrainVision Analyzer 2.0 software (Brain Products GmbH) was used. Table 6 gives an overview of the processing steps. First, data were rereferenced to linked mastoids (average of TP9 and TP10) and the online reference Cz was reinstated as channel. Next, data were filtered using a 0.1 Hz low-cutoff and a 20 Hz high-cutoff filter (each with 48 dB/octave slope). An automatic raw data inspection was applied using built-in algorithms of the Brain Vision Analyzer. Intervals of 400 ms were marked as artifact when there was either an activity of less than 0.5 µV in time windows of 100 ms, voltage steps exceeding 50 µV/ms within the interval or a maximal amplitude difference above 400 µV in an interval of 200 ms. Channel IO (below the left eye) was excluded from the artifact detection to prevent blinks exceeding the amplitude difference criterion from being discarded as artifacts. For two subjects most of the relevant data was excluded in the raw data inspection. However, visual inspection showed that this was mainly due to blinks being detected as artifacts due to the amplitude difference criterion. For these participants raw data inspection was rerun excluding the amplitude difference criterion. Following raw data inspection, blinks and eye movements were corrected using an independent component analysis (ICA) method implemented in the Analyzer. Next, data were segmented with reference to the outcome onset. For ERP analyses segments started 200 ms before outcome onset and lasted until 1000 ms following outcome onset. For time-frequency analyses the segments lasted from -1000 ms until +1000 ms with respect to outcome onset. An artifact rejection algorithm was applied to all channels, excluding segments with maximal voltage differences above 200 µV in 200 ms windows to exclude potentially remaining blink- and eye-movement artifacts that were not properly corrected by the ICA. For ERP analyses average waveforms were calculated separately for every participant and each of the four outcome types[8]. The averages were then baseline corrected, with the first 100 ms before outcome onset serving as baseline. The peaks of the P2, N2 and P300 were then detected semiautomatically and corrected manually if necessary. P2 and N2 were detected at electrodes Fz, FCz and Cz. The detection time

[8] Averages for the ERPs were based on the following mean numbers of segments (minimum number given in parentheses): Full Win: 31.7 (28), Full Miss: 31.2 (25), Near Win: 31.5 (27), Near Miss: 31.2 (20)

windows were 175 ms to 275 ms for the P2 peak and 275 ms to 450 ms for the N2 peak. The P300 was detected in the time window from 300 ms to 650 ms at electrode Pz. For the statistical analysis the average voltage value in an interval of 24 ms around the detected peaks (+/- 12 ms corresponding to +/- 3 datapoints) was exported. The FRN was then quantified as the difference P2-N2, thus larger positive values denote a larger FRN. For time-frequency analyses complex morelet wavelets were used. The wavelets spanned the frequency range from 1 to 20 Hz in 19 logarithmic steps. A morelet parameter of 5 was chosen. The resulting power values were baseline corrected, using the time between -400 ms to -200 ms before outcome onset as baseline. Following wavelet convolution, averages for every subject and every outcome type were computed[9]. Finally, the mean power from three layers (central frequencies of the exported layers: 4.47 Hz, 5.28 Hz, 6.24 Hz), corresponding to the theta range, was extracted at electrodes Fz, FCz and Cz in the time range 250 ms to 500 ms after outcome onset.

[9] Averages for the wavelets were based on the following mean numbers of segments (minimum number given in parentheses): Full Win: 31.5 (27), Full Miss: 31.1 (24), Near Win: 31.4 (27), Near Miss: 31.0 (20)

Table 6. Study 1: Preprocessing and export for ERP and time-frequency analyses in the wheel of fortune paradigm

ERP	Time-Frequency
Rereferencing linked mastoids	
Filter 0.1 Hz low-cutoff and 20 Hz high-cutoff, 48 dB/octave slope	
Raw data inspection (channel IO excluded) Gradient criterion (≥50 µV/ms) Low Activity criterion (≤ 0.5 µV in 100ms) Difference criterion (≥ 400 µV in 200ms)	
Ocular Correction via ICA	
Segmentation -200 ms to +1000 ms	*Segmentation* -1000 ms to +1000 ms
Artifact Rejection (all channels) Difference criterion (≥ 200 µV in 200 ms)	
Average	*Complex Morelet Wavelets* 1 Hz - 20 Hz 19 layers, logarithmic Morelet parameter 5 -400 ms to -200 ms baseline
Baseline Correction -100 ms to 0 ms	*Average*
Peak Detection (semiautomatic) P2: 175-275 ms N2: 275-450 ms P300: 300-650 ms	*Mean Power Export* Layers 10 - 12 (central frequencies: 4.47 Hz, 5.28 Hz, 6.24 Hz) 250 ms to 500 ms Fz, FCz, Cz
Peak Export P2 + N2: Fz, FCz, Cz P300: Pz 24 ms (+/-12 ms) around peak	

4.2.6 Analysis of choice behavior

To see whether participants showed a gambler's fallacy like behavior, the probability of choosing a color again following a run of this color was analyzed. Since the outcome sequence was random for every participant and the paradigm focused on delivering a certain amount of wins and misses and not a certain amount of outcomes of each color, the color sequence was not controlled and thus varied between participants. For every participant runs of the same color of lengths 1 to 5[10] were extracted and the probability of choosing the same color again in the next trial was calculated (Ayton & Fischer, 2004).

4.2.7 Statistical analysis

The FRN amplitudes and theta power were analyzed with a 2*2*3*2 mixed analysis of variance (ANOVA) with the within-subject factors "outcome" (win vs. miss), "closeness" (near vs. full) and "electrode" (Fz, FCz, Cz) and the between-subject factor "group" (PG vs. nonPG). The P300 amplitudes were analyzed with a 2*2*2 mixed ANOVA with the within-subject factors "outcome" (win vs. miss) and "closeness" (near vs. full) and the between-subject factor "group" (PG vs. nonPG). The mean rating data (valence, motivation, arousal, probability of winning in the next trial) for each outcome type were analyzed separately using 2*2*2 mixed ANOVAs with the within-subject factors "outcome" (win vs. miss) and "closeness" (near vs. full) and the between-subject factor "group" (PG vs. nonPG). The control ratings and questionnaire data were compared between groups with independent samples t-tests. Choice behavior was analyzed with a 5*2 mixed ANOVA with the within-subject factor "run length" (1 vs. 2 vs. 3 vs. 4 vs. 5) and the between-subject factor "group" (PG vs. nonPG). In cases of violation of the assumption of sphericity, the Greenhouse-Geisser correction was applied and corrected degrees of freedom are reported. The *p*-values from the comparison of the questionnaire data were adjusted for multiple comparisons by setting the false discovery rate (FDR) to 0.05 (Benjamini & Hochberg, 1995). For all ANOVAs, partial eta-squared values are reported as measures of effect size. The analyses were run in SPSS 21 (IBM) and R (R Core Team, 2014).

4.3 Results

4.3.1 FRN

The analysis of the FRN yielded a significant main effect of the factor "outcome" ($F(1,38) = 7.87$, $p = .008$, $\eta^2_p = .17$). This effect was qualified by a significant interaction with the factor "electrode" ($F(1.245,47.322) = 9.46$, $p = .002$, $\eta^2_p = .20$). Post-hoc paired t-test revealed, that misses

[10] The runs were overlapping. A run of length 5 (e.g. orange, orange, orange, orange, orange) also contains one run each of lengths 1 to 4.

compared to wins elicited a significantly larger FRN at electrodes Fz ($t(39) = 3.42$, $p = .001$) and FCz ($t(39) = 2.90$, $p = .006$) but not at Cz ($t(39) = 1.63$, $p = .111$). Furthermore, there was a significant main effect of the factor "group" ($F(1,38) = 5.43$, $p = .025$, $\eta^2_p = .13$), with the PG group showing smaller peak-to-peak FRN amplitudes than the nonPG group. Figures 7 and 8 show the ERPs and the mean FRN values at electrodes Fz, FCz and Cz, while Table 7 lists the complete ANOVA results. Figure 9 shows the topography of the difference wave between misses and wins, reflecting the effect of the factor "outcome".

Table 7. Study 1: Results of the ANOVA for the FRN

Effect	*F* (*df*)	*p*	η^2_p
"Outcome"	7.87 (1,38)	.008**	.17
"Closeness"	0.09 (1,38)	.767	< .01
"Electrode"	0.32 (1.277,48.527)	.631	.01
"Group"	5.43 (1,38)	.025*	.13
"Outcome" x "Closeness"	0.01 (1,38)	.917	< .01
"Outcome" x "Electrode"	9.46 (1.245,47.322)	.002**	.20
"Outcome" x "Group"	0.58 (1,38)	.449	.02
"Closeness" x "Electrode"	2.00 (1.181,44.875)	.162	.05
"Closeness" x "Group"	0.85 (1,38)	.362	.02
"Electrode" x "Group"	2.19 (1.277,48.527)	.140	.06
"Outcome" x "Closeness" x "Electrode"	1.71 (1.332,50.598)	.197	.04
"Outcome" x "Closeness" x "Group"	0.34 (1,38)	.564	.01
"Outcome" x "Electrode" x "Group"	0.38 (1.245,47.322)	.588	.01
"Closeness" x "Electrode" x "Group"	0.02 (1.181,44.875)	.930	< .01
"Outcome" x "Closeness" x "Group" x "Electrode"	0.06 (1.332,50.598)	.870	< .01

Note. *$p < .05$. **$p < .01$. ***$p < .001$

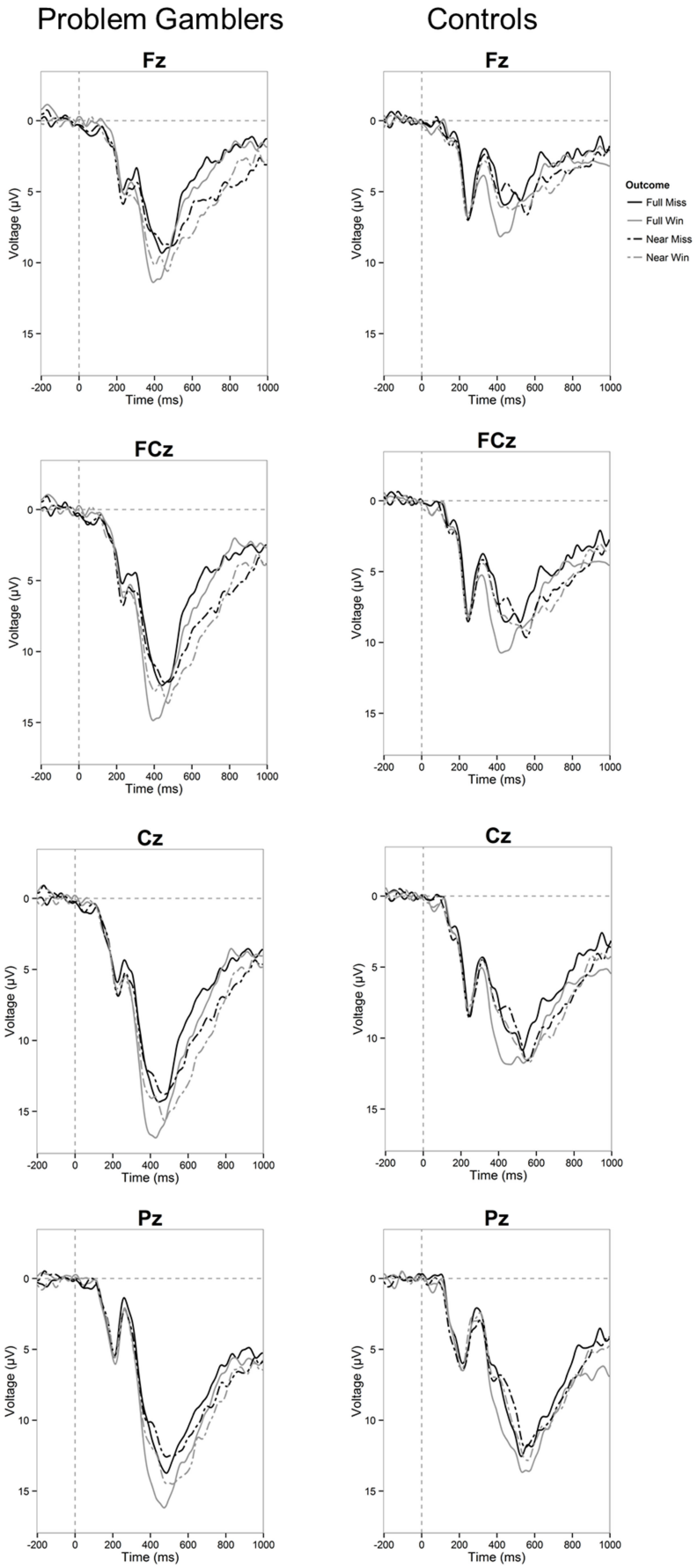

Figure 7. Study 1: ERP waveforms following the four outcome types at electrodes Fz, FCz, Cz, and Pz for problem gamblers and controls.

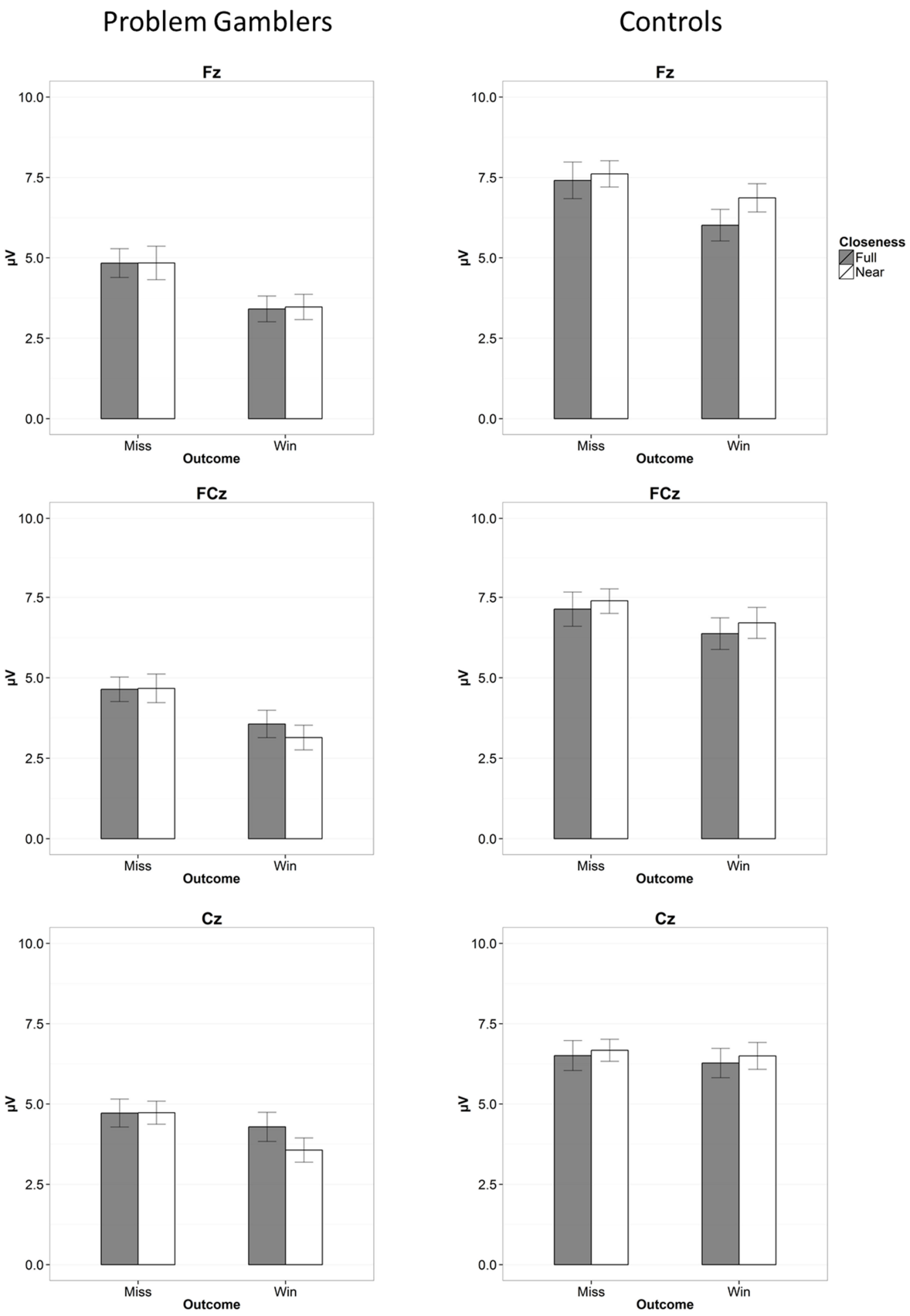

Figure 8. Study 1: Mean FRN values at electrodes Fz, FCz, and Cz for problem gamblers and controls. Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

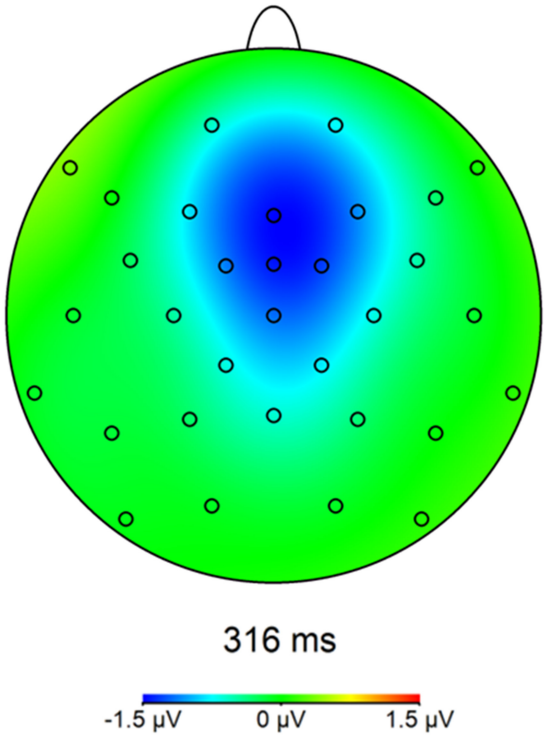

Figure 9. Study 1: Topography of the difference wave miss-win across all participants, reflecting the main effect of outcome for the FRN.

4.3.2 P300

The analysis of the P300 yielded significant main effects of the factors "outcome" ($F(1,38) = 19.41$, $p < .001$, $\eta^2_p = .34$) and "closeness" ($F(1,38) = 14.02$, $p = .001$, $\eta^2_p = .27$). Wins elicited a larger P300 than misses and full outcomes elicited a larger P300 than near outcomes. Figures 7 and 10 show the ERP waveform at Pz and the mean P300 amplitudes. Table 8 lists the complete ANOVA results. Figure 11 shows the topography difference wave between misses and wins, reflecting the effect of the factor "outcome", while Figure 12 shows the topography of the difference wave between near and full outcomes, reflecting the effect of the factor "closeness".

Table 8. Study 1: Results of the ANOVA for the P300

Effect	*F* (*df*)	*p*	η^2_p
"Outcome"	19.41 (1,38)	< .001***	.34
"Closeness"	14.02 (1,38)	.001**	.27
"Group"	0.53 (1,38)	.473	.01
"Outcome" x "Closeness"	0.74 (1,38)	.394	.02
"Outcome" x "Group"	1.16 (1,38)	.288	.03
"Closeness" x "Group"	0.29 (1,38)	.592	.01
"Outcome" x "Closeness" x "Group"	0.53 (1,38)	.472	.01

Note. *$p < .05$. **$p < .01$. ***$p < .001$

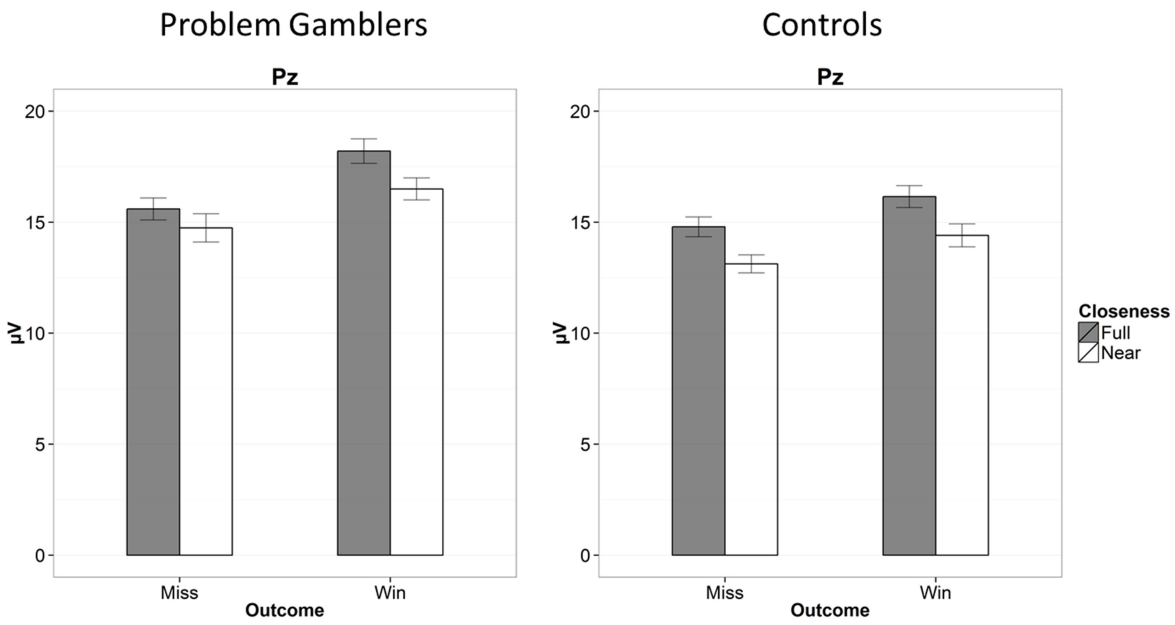

Figure 10. Study 1: Mean P300 values at electrode Pz for problem gamblers and controls. Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

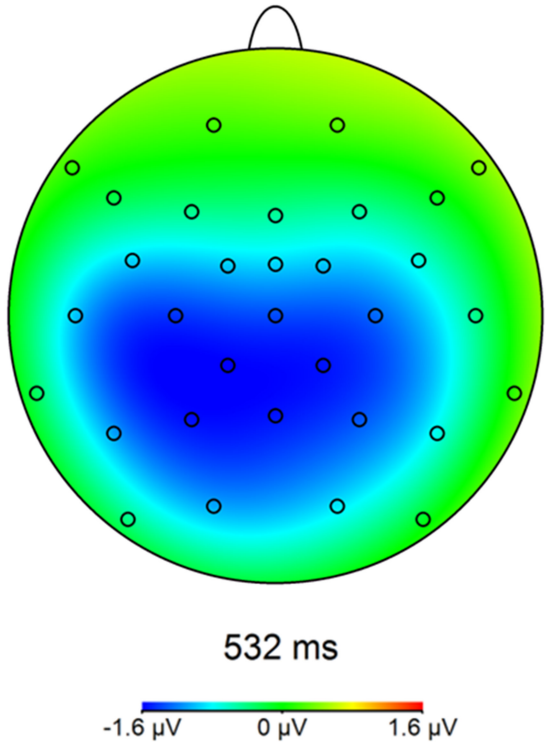

Figure 11. Study 1: Topography of the difference miss-win across all participants, reflecting the main effect of outcome for the P300.

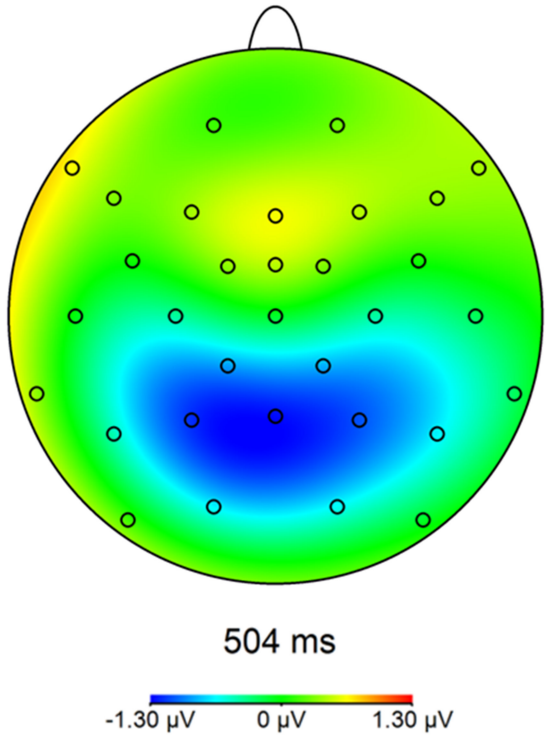

Figure 12. Study 1: Topography of the difference near-full across all participants, reflecting the main effect of closeness for the P300.

4.3.3 Theta

The analysis of theta power yielded a significant main effect of the factor "outcome" ($F(1,38) = 4.42$, $p = .042$, $\eta^2_p = .10$). Furthermore, there was a significant main effect of "electrode" ($F(1.294,49.165) = 5.00$, $p = .022$, $\eta^2_p = .12$). The two main effects were qualified by a marginally significant interaction ($F(2,76) = 3.03$, $p = .054$, $\eta^2_p = .07$). Post-hoc paired t-tests showed that wins and misses differed significantly at electrodes Fz ($t(39) = 2.36$, $p = .024$) and FCz ($t(39) = 2.17$, $p = .037$) but not at Cz ($t(39) = 1.49$, $p = .144$), with misses showing a greater theta power increase relative to baseline compared to wins. Furthermore, for wins, theta power at electrode FCz was significantly larger than at Fz ($t(39) = 2.76$, $p = .009$), while there were no significant differences between electrodes FCz and Cz ($t(39) = 1.61$, $p = .115$) and electrodes Fz and Cz ($t(39) = 0.22$, $p = .830$). For misses, theta power at electrode FCz was significantly larger than at Fz ($t(39) = 2.61$, $p = .013$) and Cz ($t(39) = 3.21$, $p = .003$) while there was no significant difference between electrodes Fz and Cz ($t(39) = 1.37$, $p = .180$) Figures 13 and 14 show the time frequency plots at electrodes Fz, FCz and Cz, and the mean theta values at Fz, FCz, and Cz, separately for both groups. Table 9 lists the full results of the ANOVA. Figures 15 and 16 show the topography of the theta power difference between misses and wins, reflecting the effect of the factor "outcome".

Table 9. Study 1: Results of the ANOVA for theta power

Effect	*F* (*df*)	*p*	η^2_p
"Outcome"	4.42 (1,38)	.042*	.10
"Closeness"	0.39 (1,38)	.539	.01
"Electrode"	5.00 (1.294,49.165)	.022*	.12
"Group"	0.19 (1,38)	.666	.01
"Outcome" x "Closeness"	0.35 (1,38)	.555	.01
"Outcome" x "Electrode"	3.03 (2,76)	.054	.07
"Outcome" x "Group"	1.41 (1,38)	.243	.04
"Closeness" x "Electrode"	0.32 (1.304,49.553)	.632	.01
"Closeness" x "Group"	2.83 (1,38)	.101	.07
"Electrode" x "Group"	0.72 (1.294,49.165)	.436	.02
"Outcome" x "Closeness" x "Electrode"	0.08 (1.280,48.628)	.833	< .01
"Outcome" x "Closeness" x "Group"	0.30 (1,38)	.585	.01
"Outcome" x "Electrode" x "Group"	0.63 (2,76)	.537	.02
"Closeness" x "Electrode" x "Group"	0.16 (1.304,49.553)	.760	< .01
"Outcome" x "Closeness" x "Group" x "Electrode"	1.68 (1.280,48.628)	.203	.04

Note. *p < .05. **p < .01. ***p < .001

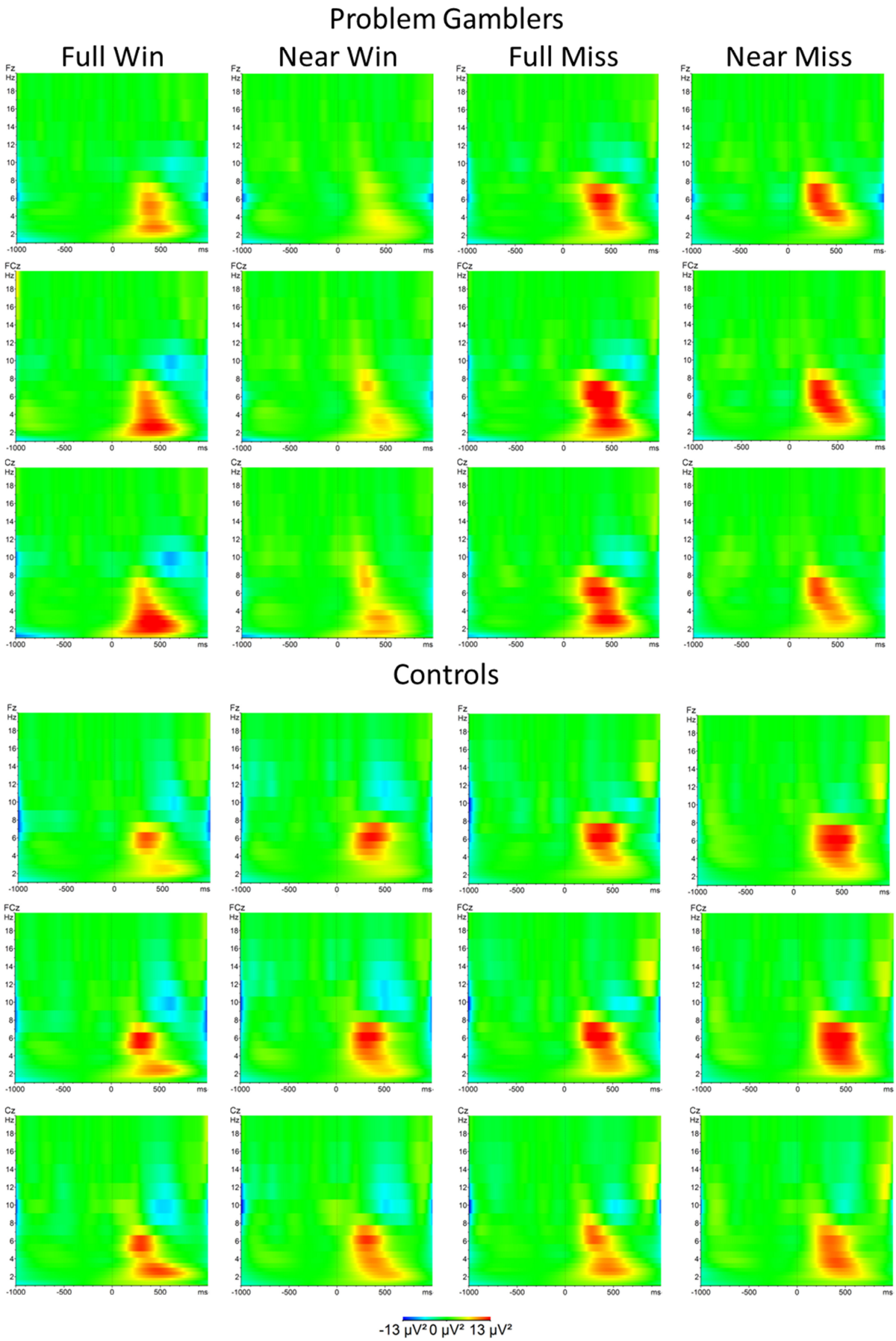

Figure 13. Study 1: Time-frequency plots for the four outcome types at electrodes Fz, FCz and Cz separately for problem gamblers and controls.

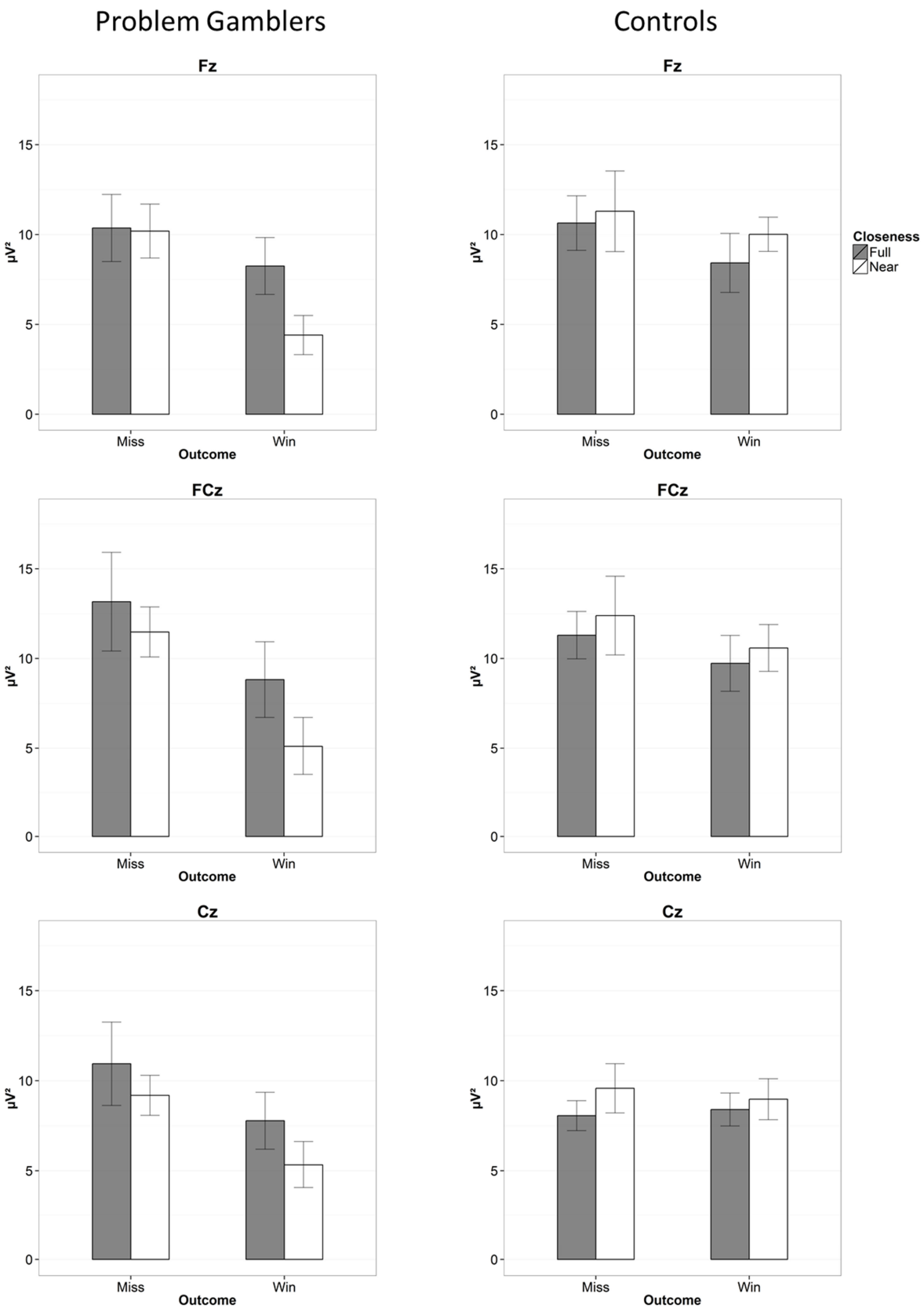

Figure 14. Study 1: Mean theta values at electrodes Fz, FCz, and Cz for problem gamblers and controls.
Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

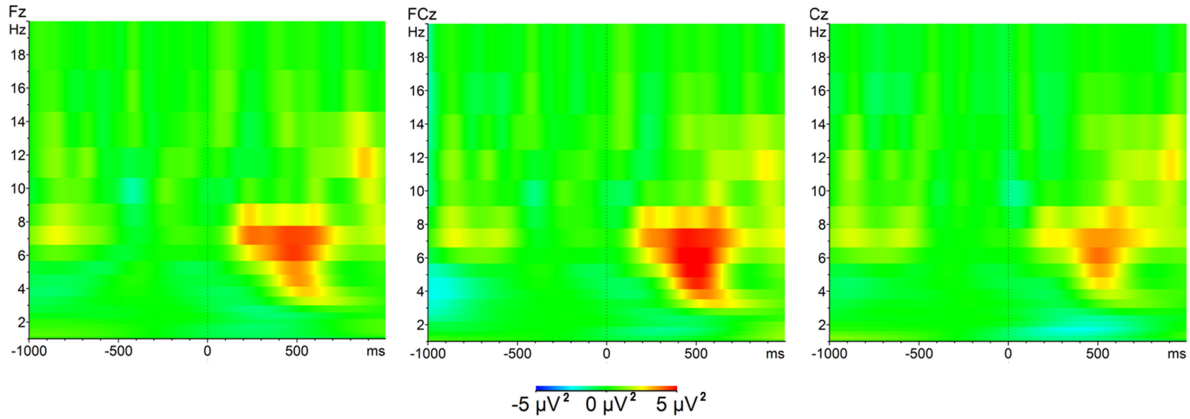

Figure 15. Study 1: Power difference between misses and wins (misses-wins) for electrodes Fz, FCz, and Cz, reflecting the outcome effect in theta power.

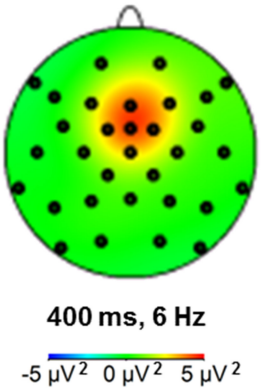

Figure 16. Study 1: Topography of the power difference between misses and wins (misses-wins) at 400 ms post outcome and 6 Hz, reflecting the outcome effect in theta power.

4.3.4 Rating data

The analysis of the valence ratings showed a significant main effect of the factor "outcome" ($F(1,38) = 221.84$, $p < .001$, $\eta^2_p = .85$). Wins were rated as being more pleasant than misses.

The ANOVA of the motivation ratings also yielded a significant main effect of the factor "outcome" ($F(1,38) = 29.52$, $p < .001$, $\eta^2_p = .44$). On average, wins were rated as being more motivating than misses.

The analysis of the arousal ratings showed a significant main effect of the factor "group" ($F(1,38) = 4.32$, $p = .045$, $\eta^2_p = .10$), with the PG group showing higher subjective arousal levels than the nonPG group.

The analysis of the probability of winning ratings yielded a marginally significant main effect of the factor "group" ($F(1,38) = 3.28$, $p = .078$, $\eta^2_p = .08$), with the PG group showing a trend of overall higher probability of winning ratings than the nonPG group.

There were no significant differences between the two groups regarding their average rated control over the outcome ($t(38) = 0.61$, $p = .543$).

Table 10 lists the full ANOVA results for each dependent variable, while Figures 17 and 18 show the mean rating data for both groups.

Table 10. Study 1: Results of the ANOVAs for the rating data

Dependent Variable & Effects	*F* (*df*)	*p*	η^2_p
Valence			
"Outcome"	221.84 (1,38)	< .001***	.85
"Closeness"	0.07 (1,38)	.799	< .01
"Group"	0.03 (1,38)	.854	< .01
"Outcome" x "Closeness"	0.01 (1,38)	.832	< .01
"Outcome" x "Group"	0.13 (1,38)	.722	< .01
"Closeness" x "Group"	0.05 (1,38)	.928	< .01
"Outcome" x "Closeness" x "Group"	0.46 (1,38)	.501	.01
Motivation			
"Outcome"	29.52 (1,38)	< .001***	.44
"Closeness"	0.48 (1,38)	.492	.01
"Group"	1.18 (1,38)	.284	.03
"Outcome" x "Closeness"	0.07 (1,38)	.800	< .01
"Outcome" x "Group"	0.03 (1,38)	.869	< .01
"Closeness" x "Group"	1.08 (1,38)	.305	.03
"Outcome" x "Closeness" x "Group"	0.18 (1,38)	.673	.01
Arousal			
"Outcome"	0.35 (1,38)	.558	.01
"Closeness"	0.15 (1,38)	.705	< .01
"Group"	4.32 (1,38)	.045*	.10
"Outcome" x "Closeness"	0.08 (1,38)	.780	< .01
"Outcome" x "Group"	0.04 (1,38)	.837	< .01
"Closeness" x "Group"	<0.01 (1,38)	1	< .01
"Outcome" x "Closeness" x "Group"	0.20 (1,38)	.655	.01
Probability of Winning			
"Outcome"	2.59 (1,38)	.116	.06
"Closeness"	0.01 (1,38)	.919	< .01

Dependent Variable & Effects	*F* (*df*)	*p*	η^2_p
"Group"	3.28 (1,38)	.078	.08
"Outcome" x "Closeness	0.01 (1,38)	.913	< .01
"Outcome" x "Group"	0.25 (1,38)	.623	.01
"Closeness" x "Group"	<0.01 (1,38)	1	< .01
"Outcome" x "Closeness" x "Group"	0.05 (1,38)	.827	< .01

Note. **p* < .05. ***p* < .01. ****p* < .001

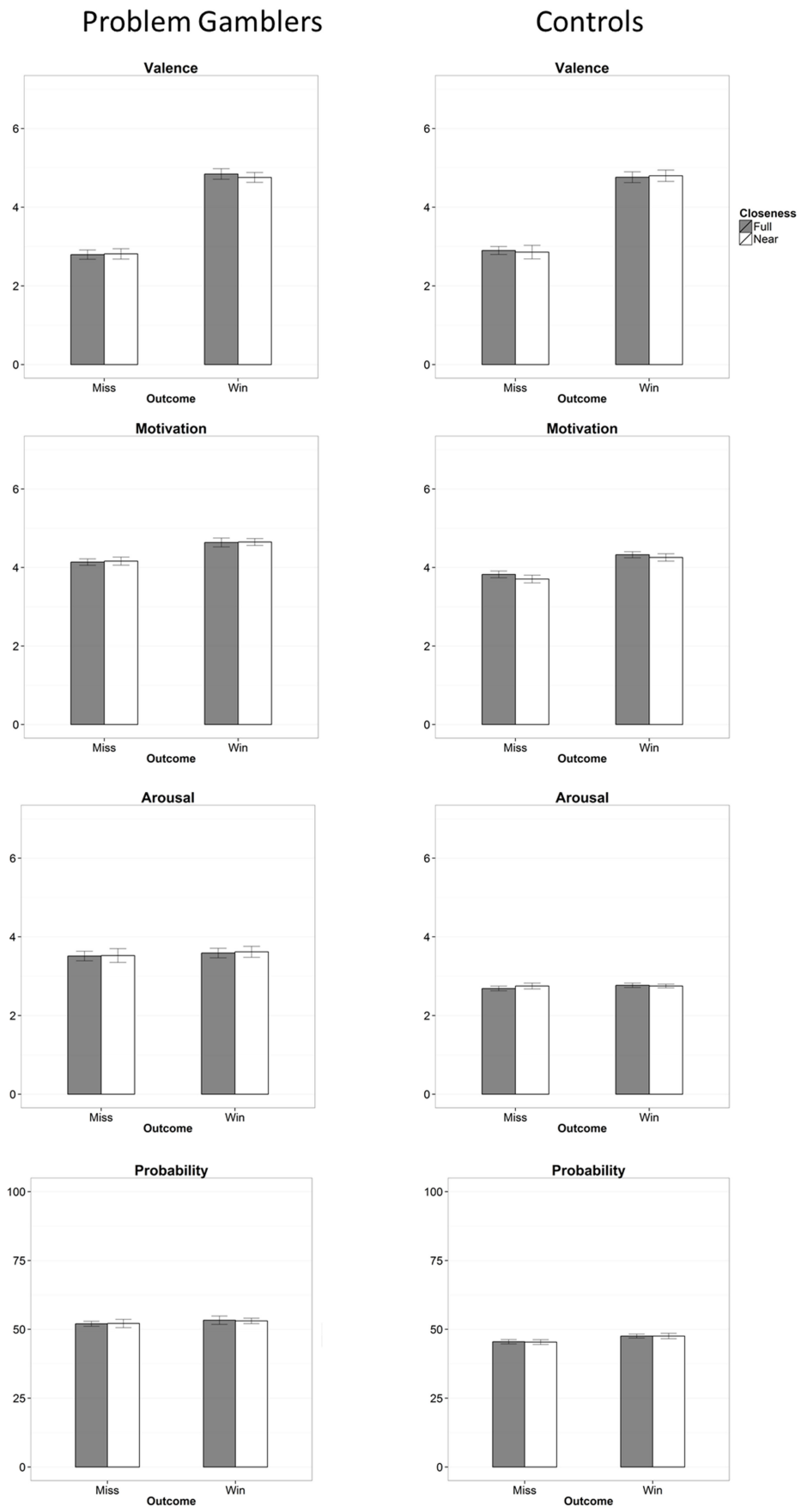

Figure 17. Study 1: Subjective ratings on valence, motivation, arousal and probability of winning in the next trial for problem gamblers and controls.
Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

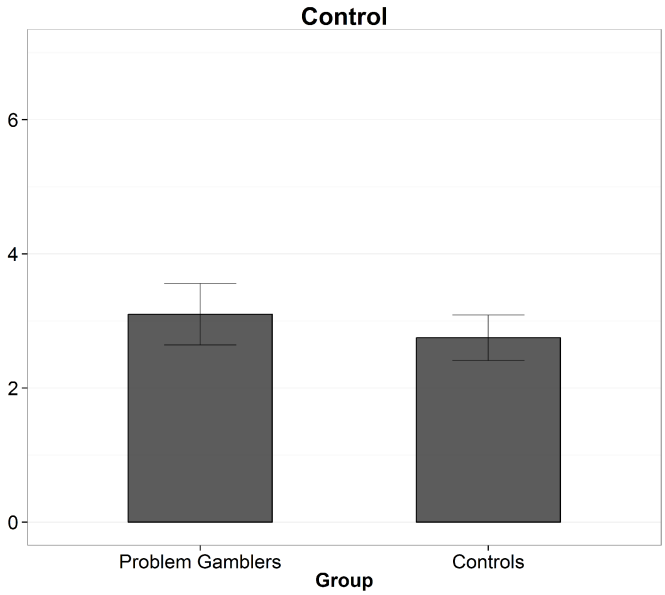

Figure 18. Study 1: Ratings of subjective control over the outcome on the wheel of fortune for problem gamblers and controls.
Error bars denote SEM.

4.3.5 Questionnaires

Compared to the nonPG group, the PG group scored significantly higher on the GRCS sum score ($t(38) = 5.50$, $p < .001$), the GRCS subscales "Gambling Expectancies" ($t(38) = 5.44$, $p < .001$), "Predictive Control" ($t(38) = 5.43$, $p < .001$), "Inability to Stop" ($t(38) = 8.35$, $p < .001$) and "Interpretative Bias" ($t(38) = 6.00$, $p < .001$), the UPPS subscale "Urgency" ($t(38) = 4.97$, $p < .001$), and the BIGL ($t(38) = 4.14$, $p < .001$). There were no significant group differences in the UPPS subscales "Premediation", "Perseverance" and "Sensation Seeking", and the AMS subscales "Hope of Success" and "Fear of Failure". Table 11 shows the mean questionnaire values for PG and nonPG groups as well as the results of the t-tests.

Table 11. Study 1: Comparison of the questionnaire scores between the PG and nonPG group

Questionnaire	Problem Gamblers (PG)	Control Group (nonPG)	*t* (*df*)	*p*
GRCS				
"Gambling Expectancies"	4.10 (1.32)	2.05 (1.05)	5.44 (38)	< .001***
"Illusion of Control"	3.44 (5.51)	1.24 (0.59)	1.77 (38)	0.137
"Predictive Control"	3.28 (1.09)	1.13 (0.32)	5.43 (38)	< .001***
"Inability to Stop"	3.26 (1.09)	1.13 (0.32)	8.35 (38)	< .001***
"Interpretative Bias"	4.75 (1.25)	2.19 (1.44)	6.00 (38)	< .001***
Sum Score	18.83 (7.96)	8.20 (3.35)	5.50 (38)	< .001***

Questionnaire	Problem Gamblers (PG)	Control Group (nonPG)	*t* (*df*)	*p*
UPPS				
"Urgency"	3.33 (0.52)	2.38 (0.69)	4.97 (38)	< .001***
"Premediation"	3.50 (0.46)	3.61 (0.62)	-0.60 (37)	.656
"Perseverance"	3.49 (0.45)	3.51 (0.70)	-0.11 (38)	.915
"Sensation Seeking"	3.64 (0.80)	3.51 (0.90)	0.48 (38)	.688
AMS				
"Hope of Success"	3.19 (0.43)	3.06 (0.39)	0.97 (37)	.441
"Fear of Failure"	2.15 (0.54)	1.94 (0.51)	1.19 (36)	.350
BIGL	45.5 (9.21)	35.3 (6.04)	4.14 (38)	< .001***

Note. Due to missing responses some questionnaire scores could not be computed for all participants, explaining the different number of degrees of freedom. *p*-values were adjusted using an FDR-correction according to Benjamini and Hochberg (1995). *$p < .05$. **$p < .01$. ***$p < .001$

4.3.6 Choice behavior

The ANOVA yielded a marginally significant main effect of the factor "run length" ($F(2.439,92.693) = 2.60$, $p = .068$, $\eta^2_p = .06$). Post-hoc paired t-tests indicated that participants showed a tendency to more often chose a color following run lengths of five compared to lengths of one ($t(39) = 2.16$, $p = .037$), two ($t(39) = 2.03$, $p = .050$) and four ($t(39) = 2.01$, $p = .052$) of the respective color. Table 12 shows the mean number of runs of a given length separately for the PG and nonPG group. Figure 19 shows the mean probability of choosing the last outcome color as a function of previous run length. Table 13 shows the full ANOVA results.

Table 12. Study 1: Mean number of runs of a given length per group

	Run Length				
Group	1	2	3	4	5
PG	64.65 (4.75)	31.30 (3.15)	15.15 (1.95)	8.30 (1.75)	4.05 (1.64)
nonPG	63.60 (5.19)	31.35 (2.32)	15.40 (1.93)	8.10 (2.08)	4.45 (1.10)

Note: *SD* is given in parentheses

Table 13. Study 1: Results of the ANOVA for choice behavior

Effect	*F* (*df*)	*p*	η^2_p
"Run Length"	2.60 (2.439,92.693)	.068	.06
"Group"	0.02 (1,38)	.900	< .01
"Run Length" x "Group"	0.20 (2.439,92.693)	.860	.01

Note. **p* < .05. ***p* < .01. ****p* < .001

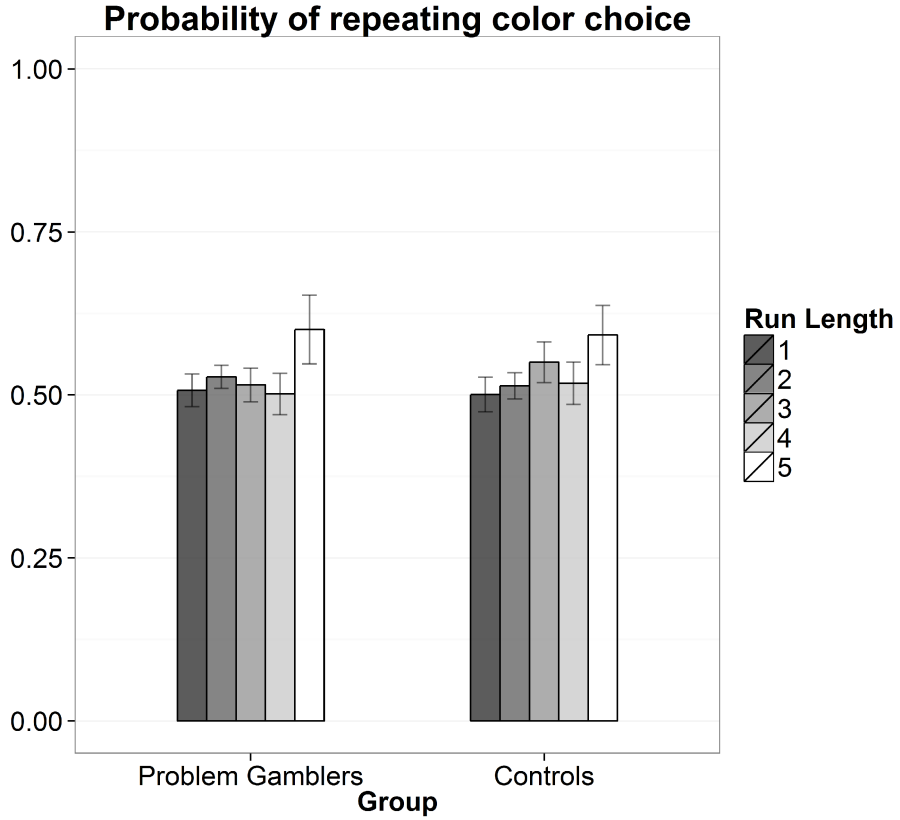

Figure 19. Study 1: Probability of choosing the last outcome color as a function of run length of this outcome color for problem gamblers and controls.
Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

4.4 Discussion

The results showed that misses compared to wins elicited a larger FRN, while near outcomes did not differ from full outcomes in terms of FRN peak-to-peak amplitude. The PG group showed overall smaller FRN amplitudes compared to the nonPG group. For the P300, wins elicited larger amplitudes than misses and full outcomes elicited larger amplitudes than near outcomes. The P300 was not affected by gambling problems. The analysis of theta power showed a significant effect of outcome valence, with misses leading to larger theta power increases than wins. Valence and motivation were rated higher following wins compared to misses, with closeness and gambling

problems having no effect on either rating. However, the PG group showed generally higher subjective arousal ratings and a trend for generally higher ratings of probability of winning in the subsequent trial, even though there was no difference in perceived control over the outcomes compared to the control group. These results will be discussed further in the following sections.

4.4.1 EEG

4.4.1.1 FRN and theta

The results replicated the classic FRN effect of a larger negativity following misses compared to wins (e.g. Gehring & Willoughby, 2002; Hajcak, Moser, Holroyd, & Simons, 2006; Kreussel et al., 2012; Osinsky et al., 2012; Ulrich & Hewig, 2014). Contrary to previous studies using the wheel of fortune (Ulrich & Hewig, 2014; Weiß, 2014), closeness did not have an effect on the FRN. Thus, there was no support for the hypothesis of observing a more negative FRN following near compared to full outcomes in the control group. The corresponding hypothesis of no FRN difference between near and full misses and a smaller (i.e. less negative) FRN following near compared to full wins in problem gamblers was also not supported by results. Methodological constraints might have concealed effects of closeness. The FRN was quantified as peak-to-peak amplitude, requiring the detection of the P2 and N2 peaks separately for every participant and outcome type. The averaged ERPs consisted of 31 segments on average. Thus, a certain amount of noise was still present in the data, complicating the reliable detection of the P2 and N2 peaks. Furthermore, as the grand averages of the PG group show, this group only showed very small N2 peaks. To circumvent this problem, a time-frequency analysis of the theta band was also included, since this analysis does not depend on the manual detection of ERP peaks but yields similar (albeit not identical) information regarding the underlying feedback processing. Theta power was higher following misses compared to wins, replicating previous findings (e.g. Cavanagh, Figueroa et al., 2012; Cavanagh, Zambrano-Vazquez et al., 2012; Hajihosseini & Holroyd, 2013). There was no effect of closeness on theta power. The FRN has been interpreted in terms of indicating the valence of an outcome (good vs. bad; Hajcak et al., 2006; Hajihosseini & Holroyd, 2013). In this light, the lack of a closeness effect on the FRN indicates that near and full outcomes were perceived as equally good or bad, with the main distinction being made between wins and misses, which also showed differences in the FRN. These effects are also mirrored in the valence ratings, which showed a more positive valence for wins compared to misses but also no effect of closeness.

Although problem gambling did not show the expected interaction with closeness on the FRN, a main effect of gambling status was observed, with the PG group showing consistently smaller peak-to-peak FRN amplitudes than the nonPG group. This might indicate an increased reward

positivity to all outcomes in problem gamblers, suppressing the default FRN/N2 component (Holroyd et al., 2008). Thus, compared to the control group, problem gamblers might have generally evaluated the outcomes in the wheel of fortune as more favorably. A different result has been reported by Lole et al. (2015). They quantified the FRN using temporo-spatial principal components analysis. This yielded a component which was negative going following losses and positive going following wins, fitting with previous results on the FRN and a reward-related positivity (Holroyd et al., 2008). Both the negative deflection following losses and the positive deflection following wins were reduced in problem gamblers. Lole et al. (2015) interpreted this finding as a sign of both reward and punishment hyposensitivity in problem gamblers. Similar results of decreased activation in prefrontal areas following both wins and misses in problem gamblers have been reported in fMRI studies (Balodis et al., 2012; de Ruiter et al., 2009). However, ERP studies have also reported a reward hypersensitivity in problem gamblers, indicated by an increased positivity in the P200 and FRN time frames following risky decisions with a positive outcome (Hewig et al., 2010; Oberg et al., 2011). As such, these results are similar to the reduced peak-to-peak FRN amplitudes found in problem gamblers in the current study. The extent to which the occurrence of such a reward hypersensitivity is tied to risky gambling decisions versus all gambling decisions could explicitly be tested in further studies.

4.4.1.2 P300

The hypotheses concerning the P300 were partly confirmed. Both problem gamblers and controls showed an increased P300 following wins compared to misses and full compared to near outcomes, thus replicating previous results using the wheel of fortune paradigm (Ulrich & Hewig, 2014). However, problem gamblers did not exhibit smaller P300 amplitudes compared to controls.

Increased P300 amplitudes following wins compared to misses have been reported in various studies (e.g. Hajcak et al., 2005; Kreussel et al., 2012; Wu & Zhou, 2009) and likely reflect the higher motivational salience of wins (Polezzi, Sartori, Rumiati, Vidotto, & Daum, 2010), which is also reflected in the higher valence and motivation ratings following wins. Following this interpretation, the increased P300 for full compared to near outcomes might also indicate increased motivational salience for these outcomes. However, near and full outcomes did not differ in their subjective valence and motivation ratings, arguing against this interpretation. The P300 has also been interpreted as an index of context updating (Donchin & Coles, 1988). According to Donchin and Coles (1988), larger P300 amplitudes indicate bigger context updates. In this view, the larger amplitude in P300 following full compared to near outcomes might suggest that they are also followed by a larger context update. As part of this context update, the previous action might be stored together with its subsequent outcome. In the case of full outcomes, where the wheel stops in the middle of a color field, it is relatively safe to assume that repeating the previous action will again lead to a stop in the

same field (under the assumption that one has some control over the game). However, for near outcomes this is not the case, since the previous action would have almost resulted in another outcome. Hence, this action cannot yet be classified as one certainly leading to a win or a miss and thus might not yet be stored with its respective outcome. Yet another interpretation, which fits with the fMRI results for the wheel of fortune in section 5.3.1, follows the work of Johnson (1986), who suggested that, among other factors, the P300 is influenced by "equivocation". "Equivocation" refers to a loss of information in stimulus processing, reflected in an ensuing uncertainty of the participant about the stimulus he has just perceived. The P300 has been shown to be smaller when using feedback stimuli that are difficult to discriminate (Johnson & Donchin, 1978). From this perspective, near outcomes might elicit a smaller P300 compared to full outcomes because it is more difficult to detect on which side of the color boundary the wheel stopped.

As noted above, the PG and nonPG groups did not differ in the P300 amplitudes, thus not replicating results indicating smaller P300 amplitudes following feedback in problem gamblers (Lole et al., 2015; Oberg et al., 2011). This points towards similar salience of the feedback stimuli for problem gamblers and controls in the current study. It is conceivable that the discrepancy to previous studies might be related to different levels of problem gambling in the included samples. P300 amplitudes might only be reduced given certain levels of problem and pathological gambling, although the previous studies do not include a detailed enough description of the samples to draw any conclusions regarding this assumption. Thus, this question should be addressed in further studies.

4.4.2 Rating data

4.4.2.1 Valence, motivation, and control

The analysis of the valence and motivation ratings did not support the hypotheses. For both dimensions, significant main effects of the factor "outcome" were found, with wins being rated as more positive and more motivating than misses. Neither closeness, nor group or their respective interactions had an effect on the valence and motivation ratings. As such, the findings largely replicate previous results in the wheel of fortune paradigm (Ulrich & Hewig, 2014; Weiß, 2014). Yet, other studies have repeatedly found near misses to be rated as more motivating and more negative compared to full misses (Clark et al., 2012; Clark et al., 2009; Qi et al., 2011). A possible explanation of this discrepancy might lie in the different outcome probabilities used in the wheel of fortune compared to other paradigms. Clark et al. (2009), who were the first to report the valence and motivational effects, used a slot machine paradigm, which delivered wins in 1/6, near misses in 2/6 and full misses in 3/6 of the trials, whereas closeness and valence (win vs. miss) were fully balanced

in the wheel of fortune. Thus, the valence and motivation effects were observed in paradigms where both wins and near misses occurred less frequently than full misses, while the effects could not be replicated in a paradigm where near and full outcomes as well as wins and misses occurred equally often. Future studies could specifically target this question by systematically varying the probabilities of the outcomes.

Contrary to the expectations, there were no differences between PGs and nonPGs in valence and motivation ratings of near misses, which were expected based on the skill acquisition indicator (Clark et al., 2009) inherent in near misses and the increased gambling-related cognitive distortions in problem gamblers (e.g. Cunningham et al., 2014; Joukhador et al., 2004; Myrseth et al., 2010). PGs and nonPGs also did not differ in their ratings of perceived control. The overall control rating indicated that both groups (correctly) felt that they had little to no control over the outcomes. Thus, PGs did not seem to show an increased sense of control over the gamble, which could explain why the expected group differences for the near misses were not observed.

4.4.2.2 Arousal

Contrary to the hypotheses, no effect of closeness on subjective arousal was found, suggesting that near outcomes might have different effects on subjective and physiological arousal. This idea is supported by previous research from different areas (e.g. gambling, phobia, and anxiety research), which also failed to find a relation between measures of subjective and physiological arousal (Coventry & Constable, 1999; Diemer, Lohkamp, Mühlberger, & Zwanzger, 2016; Morrow & Labrum, 1978). As stimulus probability has also been shown to influence the SCR (Ben-Shakhar, Lieblich, & Kugelmass, 1982), previous results showing increased SCRs following near misses (Clark et al., 2012; Dixon et al., 2011) might have been party influenced by this effect, as near misses occurred less frequently than full misses in the paradigms used.

As expected, the PG group generally showed higher subjective arousal than the nonPGs. Thus, we replicated previous findings suggesting increased physiological arousal during gambling in pathological gamblers relative to controls (Blanchard et al., 2000; Carroll & Huxley, 1994; Sharpe et al., 1995) for subjective arousal. Yet, the current study cannot address the question whether the two effects are related.

4.4.2.3 Probability of winning

PGs were expected to show generally higher ratings for the probability of winning ratings than nonPGs. The corresponding analysis only yielded a marginally significant effect in the expected direction. PGs showed a trend of higher ratings of the probability of winning than the controls, with the ratings descriptively also being larger than 50%, the number that would be expected if the

outcomes were perceived as being determined by chance alone. Thus, the PG group seemed to be slightly too optimistic regarding their chances of winning, fitting with an "optimism" bias in problem gamblers, reported by Atkins and Sharpe (2003), and in a broader sense also increased cognitive distortions in problem and pathological gamblers (e.g. Cunningham et al., 2014; Joukhador et al., 2004; Myrseth et al., 2010), as an increased belief in one's ability to win is part of these distortions (Toneatto, 1999).

4.4.3 Questionnaires

As expected, problem gamblers scored higher on gambling-related cognitive distortions (with the exception of the GRCS subscale "Inability to Stop") and showed an increased belief in good luck compared to controls. This replicates previous studies showing increased cognitive distortions in general (e.g. Cunningham et al., 2014; Joukhador et al., 2004; Myrseth et al., 2010) and increased belief in good luck as a specific cognitive distortion (Chiu & Storm, 2010; Wohl et al., 2007) in pathological and problem gamblers.

Concerning impulsivity, only the UPPS subscale "Urgency" showed increased scores for problem gamblers compared to controls. "Urgency" as measured in the UPPS indicates the occurrence of strong impulses which are difficult to control (Whiteside & Lynam, 2001), which can be seen as a core aspect of impulsivity. Thus, the results replicate previous findings showing increased impulsivity in pathological gamblers (e.g. Barrault & Varescon, 2013; Kräplin et al., 2014; Lawrence et al., 2009a; Petry, 2001; Steel & Blaszczynski, 1998). Furthermore, this result also fits with a previous study showing the "Urgency" subscale to be the only UPPS subscale significantly predicting SOGS scores in a combined sample of pathological gamblers, controls and individuals with borderline personality disorder and alcohol abuse problems (Whiteside, Lynam, Miller, & Reynolds, 2005).

Concerning achievement motivation, no significant differences between problem gamblers and controls were found. As such, this finding is similar to the results reported by Dickson et al. (2008) who found no differences in achievement motivation between youths with different levels of gambling problems. This suggests that general achievement motivation is not suited to distinguish between problem gamblers and controls. Instead, more specific aspects of achievement motivation might have to be taken into account, for example concerning the means through which people strife for achievement. One study showed that pathological gamblers show less motivation for achievement through conforming to rules compared to the male norm sample in the California Psychological Inventory (Taber, Russo, Adkins, & McCormick, 1986).

4.4.4 Choice behavior

The exploratory analysis of the choice behavior in the wheel of fortune paradigm showed a marginal effect of run length. However, contrary to the classic gambler's fallacy effect, the tendency of choosing the last color did not decrease with increasing run lengths, but rather increased. As such, this pattern rather resembles the belief in a hot outcome (Sundali & Croson, 2006). Maybe the ability to press a button to initiate the stopping of the wheel of fortune created the impression that is was not a random game of chance, but a game involving a skill element. Previous research has shown that the gambler's fallacy is predominant for outcome sequences generated by random processes, whereas the hot hand fallacy, which is conceptually related to the hot outcome, as they both refer to an expected positive autocorrelation, occurs more often for sequences generated by non-random processes (Burns & Corpus, 2004).

4.4.5 Limitations

Some limitations concerning the sample and the paradigm have to be kept in mind when interpreting the current results. First of all, the current results only apply to problem gamblers. While it can be hypothesized that the reported effects might be stronger in pathological gamblers, this needs to be tested in further studies. Furthermore, one can argue about what constitutes a good control group for problem and pathological gamblers. In the current study, the main aim regarding the control group was to recruit participants without gambling problems who mere matched on age, sex, handedness and education. However, the actual gambling behavior, that is, hours and money spent gambling, was not taken into consideration. Thus, the control group consists of participants who gamble more or less regularly (but without showing any related problems) and participants who do not gamble or gamble only infrequently. The best control group to answer questions related to the addictive properties of gambling might be a control group of regular gamblers, though, who do not show gambling problems. Regarding the comorbidities in the present sample, one could argue that this might have increased error variance in the current study. However, pathological gambling is often comorbid with other disorders (Kessler et al., 2008; Petry et al., 2005), making the present sample ecologically valid. Finally, the wheel of fortune uses equal probabilities of the four outcome types to rule out confounding effects of different outcome probabilities, especially on the P300. At the same time, this likely decreases external validity of the paradigm, since most real world gambles (e.g. slot machines) do not have a probability of winning outcomes and near outcomes of 50%, respectively.

4.5 Summary

To sum up, the current study showed that near outcomes elicited smaller P300 amplitudes compared to full outcomes, while the FRN amplitude, theta power and subjective evaluation did not differ between the two outcome types. Concerning color choice behavior, both groups of participants showed a tendency towards a hot outcome behavior, choosing the same color again following longer runs of this color. The results also point towards differences between problem gamblers and controls. Relative to controls, problem gamblers generally rated their subjective arousal caused by the outcomes higher, while at the same time showing reduced peak-to-peak FRN amplitudes, suggesting an increased reward-like response to all outcomes in problem gambling. Furthermore, the probability ratings and questionnaire data indicated increased cognitive distortions in the problem gambling group, replicating previous studies.

5 Study 2: Processing of near outcomes in gambling (fMRI)

5.1 Introduction

The second study used fMRI to analyze the processing of near outcomes in gambling. Furthermore, compared to the first study, the modulation by gambling problems was not analyzed by comparing groups, but by adding gambling problems as a continuous variable. For an introduction to fMRI see section 3.1.2.

5.1.1 Summary of previous results

As described earlier, brain imaging studies on near misses have shown that near compared to full misses activate areas involved in the processing of winning outcomes, like the ventral striatum and the anterior insula (Clark et al., 2009; Dymond et al., 2014). So far, no study has investigated brain activation following near wins. Hence we let participants gamble on a wheel of fortune while recording functional brain scans. We further assessed participants' gambling problems to test whether they correlate with near win processing. Previous studies have shown that near miss processing is influenced by gambling problems. Chase and Clark (2010) found a positive correlation between scores on the SOGS and activation in the dopaminergic midbrain following near miss outcomes. Habib and Dixon (2010) showed that pathological gamblers activated win-related brain networks, while control participants rather activated loss-related brain networks following near miss outcomes.

5.1.2 Current study

The current study analyzed the processing and evaluation of near and full outcomes using fMRI and rating data. Gambling problems were assessed and integrated into the analysis as a continuous variable to analyze potential influences on the processing of near outcomes. Furthermore, the relation between gambling problems and self-reported impulsivity, cognitive distortions and achievement motivation was assessed.

5.1.3 Hypotheses

In a previous study we showed that near outcomes in general (i.e. both near misses and near wins) elicit a larger FRN and smaller P300 compared to their full counterparts (Ulrich & Hewig, 2014). Thus, we also expected a main effect of outcome closeness for brain activation. Specifically, we expected increased activation following near compared to full outcomes in the ventral striatum, anterior insula and ACC. The latter area is derived from our FRN observations, as the FRN is likely generated in the ACC (Gehring & Willoughby, 2002; Miltner et al., 1997). We further expected a

correlation between gambling problems and reaction to near outcomes, with participants with higher scores on gambling screenings showing greater activation in the mentioned areas.

Based on previous results (Clark et al., 2012; Clark et al., 2009; Qi et al., 2011) it was expected that near compared to full misses would be rated as more negative but also more motivating.

Similar to the EEG study described in section 4, it was expected that gambling problems should correlate positively with self-reported cognitive distortions, belief in good luck, and impulsivity. There was no clear hypothesis for achievement motivation, hence the relation was analyzed in an exploratory manner.

5.2 Methods

5.2.1 Participants

Participants were recruited via a local website. Potential participants first had to fill in an online questionnaire, consisting of demographic questions as well as screening questions regarding pathological gambling. The latter comprised the SOGS (Lesieur & Blume, 1987) and the KFG (Petry, 1996). We invited 31 participants according to their score in the screenings with the aim of having a broader range of scores in the final sample. Due to technical problems or excessive movement during scanning, five participants had to be excluded from the fMRI analysis, leaving a final sample of 26 right-handed participants (16 male, 10 female, overall mean age = 26.65 *SD* = 6.78), the majority of which had a high-school diploma. The mean KFG score was 10.85 (*SD* = 11.35, *Min* = 0, *Max* = 51), the mean SOGS score 3.65 (*SD* = 3.09, *Min* = 0, *Max* = 11). There were no differences between males and females regarding age, education, KFG and SOGS scores. Five participants had previously taken part in an EEG experiment using the same paradigms (see section 4).

For the analysis of the rating data only one subject had to be excluded, who could not complete the wheel of fortune task due to technical problems, leaving a sample of 30 subjects for the rating data (18 male, 12 female, overall mean age = 26.33 *SD* = 6.42), the majority of which had a high-school diploma. The mean KFG score was 10.53 (*SD* = 10.67, *Min* = 0, *Max* = 51), the mean SOGS score 3.30 (*SD* = 3.02, *Min* = 0, *Max* = 11). There were no differences between males and females regarding age, education, KFG- and SOGS scores. Five participants had previously taken part in an EEG experiment using the same paradigms.

All participants received a reimbursement of 18.50 € for taking part in the experiment, consisting of a fixed amount of 10 € and an additional 8.50 € from playing the wheel of fortune and a coin toss game.

5.2.2 Paradigm: Wheel of fortune

The task consisted of the same wheel of fortune version as used in the EEG study described in section 4, except for a longer ITI (randomly varying between 2 and 6 seconds) adapted to the longer duration of the hemodynamic response in fMRI studies compared to ERPs in EEG studies. Figure 20 shows a sample trial. As in the EEG study, there were 32 trials of each of the four outcome types.

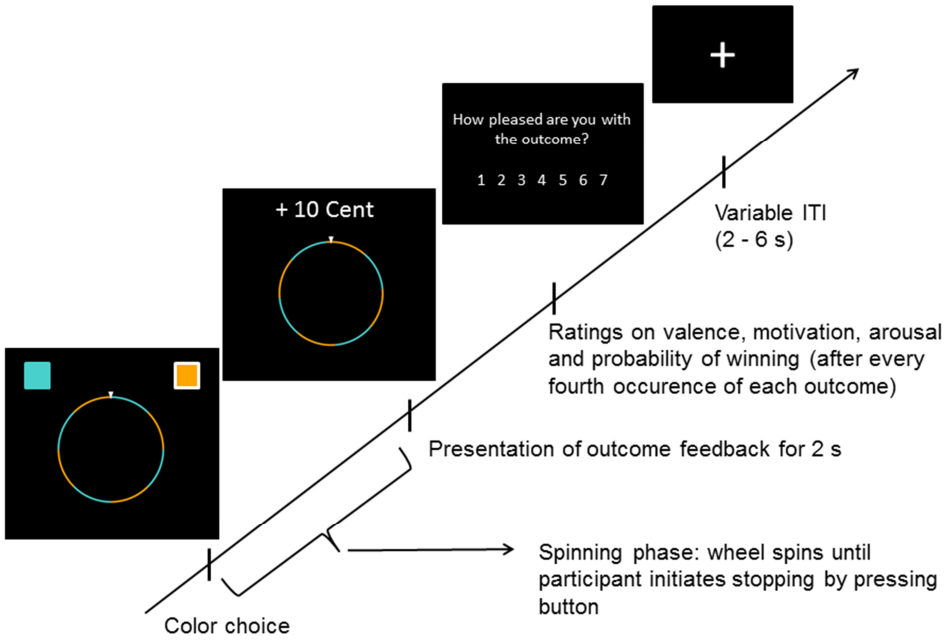

Figure 20. Study 2: Sample trial of the wheel of fortune used in the fMRI study.

5.2.3 Questionnaires

In the online survey, potential participants filled in the KFG (Petry, 1996) and the SOGS (Lesieur & Blume, 1987). After the scanning procedure participants filled in the GRCS (Raylu & Oei, 2004), the UPPS (Keye et al., 2009), the AMS (Dahme et al., 1993) and the BIGL (Darke & Freedman, 1997b). Further information on the questionnaires can be found in section 3.2.

5.2.4 Procedure

Upon entering the lab, participants received written information on the procedure of the experiment and the MRI measurement, before they signed an informed consent form. Next, participants were prepared for entering the MRI scanner. They then played the wheel of fortune paradigm and a coin toss paradigm. Two participants started with the coin toss paradigm, the rest started with the wheel of fortune paradigm. The results of the coin toss paradigm are not part of the

current work and will be reported elsewhere. Upon leaving the scanner, participants filled in the questionnaires listed above, before they were paid.

5.2.5 fMRI data acquisition and processing

Scanning was performed on a 3T MR scanner (Magnetom Trio A, Tim System, Siemens Medical Systems, Erlangen, Germany). 900 Functional images, comprised of 30 axial slices each, were acquired using a T2*-weighted sequence (Repetition Time TR = 2.0 s, echo time TE = 30 ms, flip angle = 90°, voxel size = 3.59x3.59x3.8 mm, matrix size = 64 x 64, field of view FOV = 230 mm x 230 mm). An additional T1 weighted sequence was used to capture high resolution structural images (voxel size 1x1x1 mm).

fMRI data were analyzed using SPM 12 (Wellcome Trust Centre for Neuroimaging, London). The first 10 scans were excluded from the analysis, to ensure equilibrium magnetization. Preprocessing steps included slice time correction, realignment of the functional scans, coregistration to the anatomical scan, normalization of the scans to an MNI template and smoothing. Realignment showed that three participants had to be excluded from further analysis due to excessive movement (more than 5 mm in any direction or rotation of more than 5°). During normalization, voxel size was set to 2x2x2 mm. For smoothing, an 8 mm Gaussian kernel was used.

For the first level analysis, data were high-pass filtered (128 s high-pass filter). A canonical hemodynamic response function (HRF) was modelled to the onset of the color choice, onset of the spinning of the wheel, onset of the four different outcome types and onset of the rating questions. For all events, the actual duration was extracted from the logfiles and entered into the design matrix. In addition, the movement parameters obtained during realignment were entered as regressors of no interest. The analysis focused on the four outcome types, the other events were included to account for as much variance as possible in the design matrix. The first level design matrices thus comprised 13 regressors (color choice, wheel spin, full wins, full misses, near wins, near misses, rating, 6 movement parameters).

Data were analyzed on the group level using a random effects flexible factorial 2x2 design (factors “outcome” and “closeness”). Figure 21 shows the design matrix. T-Contrasts were computed to analyze increased activation following wins compared to misses (Wins > Miss), misses compared to wins (Miss > Win), near compared to full outcomes (Near > Full), full compared to near outcomes (Full > Near) as well as a possible interaction between the factors “outcome” and “closeness” (Full Win + Near Miss > Full Miss + Near Win; Full Miss + Near Win > Full Win + Near Miss). Even though activation in specific areas was expected in the Near > Full contrast, whole brain analyses were conducted, to detect further activations which might otherwise be overlooked. To correct for

multiple testing, the significance threshold was set to $p < .05$, family-wise-error (FWE) corrected, with a cluster extent of 10 voxels (Chase & Clark, 2010). As this is a rather conservative correction (Lieberman & Cunningham, 2009), analyses were rerun using a less conservative correction of $p < .0005$ uncorrected and a cluster extent of 100 voxels (Hewig et al., 2009). To analyze the influence of gambling problems on the processing of near outcomes in the wheel of fortune, KFG scores were entered as continuous predictor in a separate regression using the contrast Near > Full as criterion. One subject had to be excluded from this analysis, since their KFG score was an outlier (KFG score = 51, more than 3 *SDs* above the mean). WFUPickatlas (Maldjian, Laurienti, & Burdette, 2004; Maldjian, Laurienti, Kraft, & Burdette, 2003) and Talairach Deamon (Lancaster et al., 1997; Lancaster et al., 2000) were used to locate significant activations in terms of anatomical landmarks and Brodmann areas (BA).

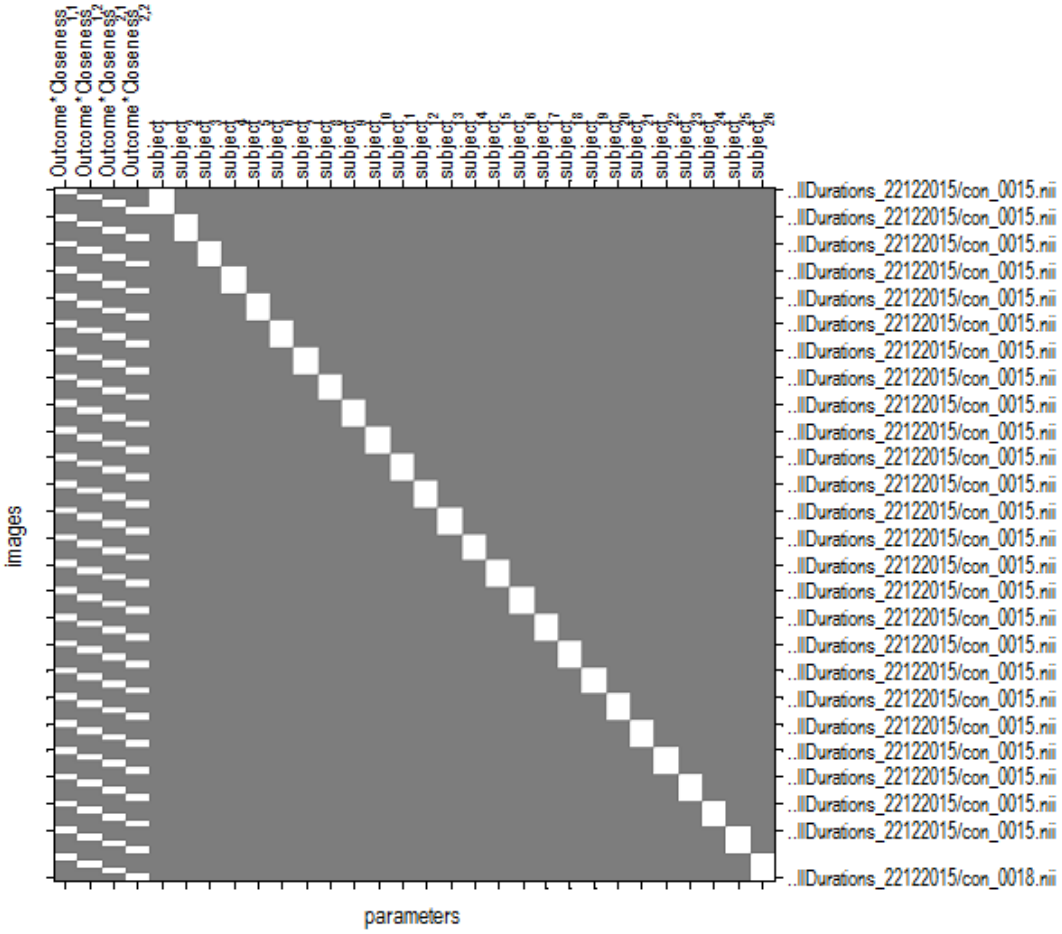

Figure 21. Study 2: Design matrix of the flexible factorial design used in the second level analysis. Parameters were included for full wins, near wins, full misses, near misses and each of the 26 subjects entered into the analysis.

5.2.6 Further statistical analysis

Rating data on valence, motivation, arousal and probability of winning in the next trial were analyzed using 2x2 repeated measures ANOVAs with the factors "outcome" (win vs. miss) and "closeness" (near vs. full). Partial eta-squared values are reported as measures of effect size. To analyze the relation between gambling problems and the other questionnaires, Pearson correlations between the KFG scores and the other questionnaires were computed. The corresponding *p*-values were adjusted for multiple comparisons by setting the FDR to 0.05 (Benjamini & Hochberg, 1995). One subject had to be excluded from this analysis, since their KFG score was an outlier (KFG score = 51, more than 3 *SDs* above the mean). SPSS 21 (IBM) and R (R Core Team, 2014) were used to run the analyses.

5.3 Results

5.3.1 fMRI data

Wins compared to misses led to significant activations in the bilateral caudatus (peak voxel left: *x, y, z* = -12, 14, -2, *Z* > 8, p_{FWE} < .001, 122 voxels; peak voxel right: *x, y, z* = 10, 10, -6, *Z* = 7.69, p_{FWE} < .001, 285 voxels; see Figure 22), the bilateral superior frontal gyrus (BA 8, peak voxel left: *x, y, z* = -14, 26, 52, *Z* = 6.00, p_{FWE} < .001, *x, y, z* = -24, 28, 52, *Z* = 5.20, p_{FWE} = .005, 157 voxels; peak voxel right: *x, y, z* = 18, 30, 54, *Z* = 6.36, p_{FWE} < .001, 155 voxels; see Figure 23) and the left angular gyrus (peak voxel: *x, y, z* = -40, -60, 34, *Z* = 5.71, p_{FWE} < .001, *x, y, z* = -50, -66, 30, *Z* = 5.05, p_{FWE} = .009, 176 voxels; see Figure 24). Further regions were activated. Table 14 lists all significant activations for the Win > Miss contrast.

Table 14. Study 2: Significant activations in the Win > Miss contrast at p_{FWE} < .05 and cluster extent 10

Region		MNI coordinates			Max *Z*-value	peak p_{FWE}	*n* Voxel
		x	y	z			
Caudatum	left	-12	14	-2	> 8	< .001	122
Caudatum	right	10	10	-6	7.69	< .001	285
Superior Frontal Gyrus (BA 8)	right	18	30	54	6.36	< .001	155
Superior Frontal Gyrus (BA 8)	left	-14	26	52	6.00	< .001	157
		-24	28	52	5.20	.005	
Angular Gyrus (BA 39)	left	-40	-60	34	5.71	< .001	176
		-50	-66	30	5.05	.009	

Region		MNI coordinates			Max *Z*-value	peak p_{FWE}	*n* Voxel
		x	y	z			
Cingulate Gyrus (BA 31)	left	-2	-34	38	5.34	.002	81
Anterior Cingulate (BA 32)	right	6	44	-2	5.10	.007	11
Superior Frontal Gyrus (BA 9)	right	14	46	36	4.99	.012	14
Anterior Cingulate (BA 32)	left	-4	40	14	4.82	.024	20

Note: MNI coordinates are given for peak voxels

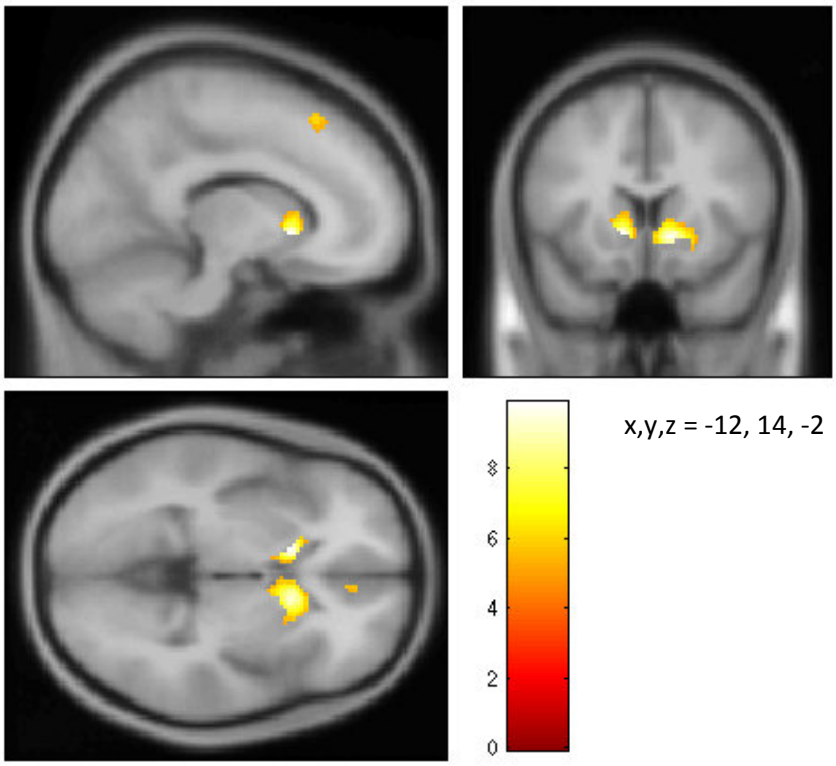

Figure 22. Study 2: Activity in the bilateral caudatus in the Win > Miss contrast.

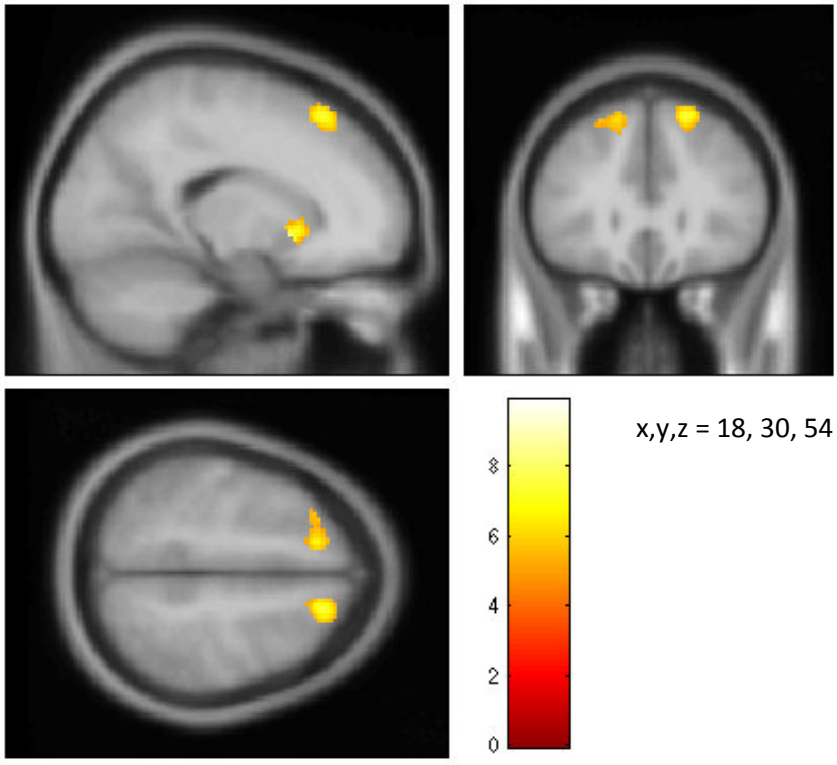

Figure 23. Study 2: Activity in the bilateral superior frontal gyrus in the Win > Miss contrast.

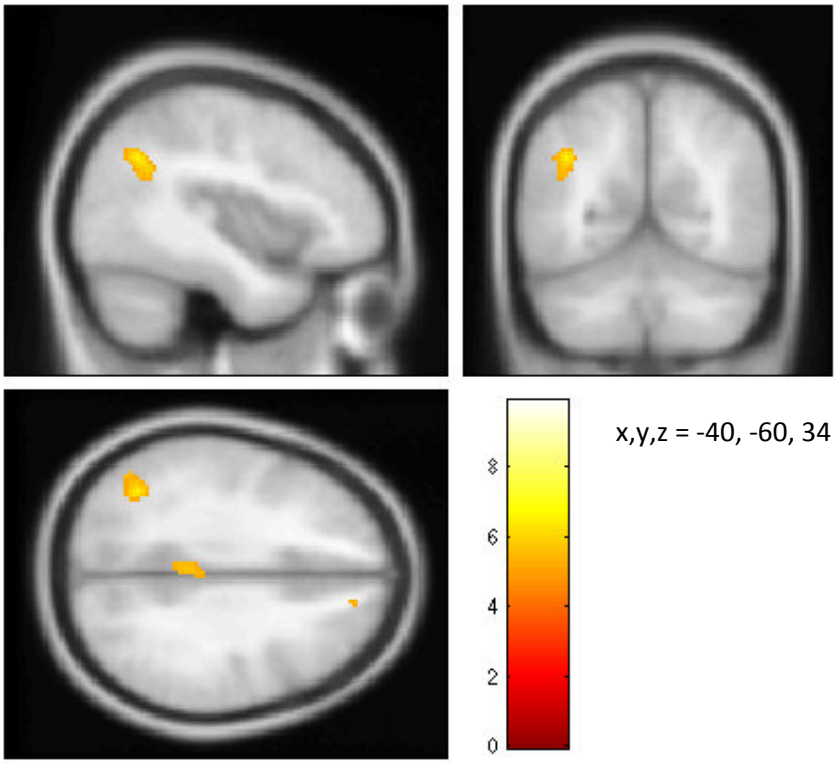

Figure 24. Study 2: Activity in the left angular gyrus in the Win > Miss contrast.

The Near > Full contrast yielded significant activations in the bilateral superior parietal lobule (peak voxel left: *x, y, z* = -20, -54, 64, *Z* = 6.01, p_{FWE} < .001, 86 voxels; peak voxel right: *x, y, z* = 20, -56, 64, *Z* = 5.47, p_{FWE} = .001, 50 voxels; see Table 15 and Figure 25).

Table 15. Study 2: Significant activations in the Near > Full contrast at p_{FWE} < .05 and cluster extent 10

Region		MNI coordinates			Max *Z*-value	peak p_{FWE}	*n* Voxel
		x	y	z			
Superior Parietal Lobule (BA 7)	left	-20	-54	64	6.01	< .001	86
Superior Parietal Lobule (BA 7)	right	20	-56	64	5.47	.001	50

Note: MNI coordinates are given for peak voxels

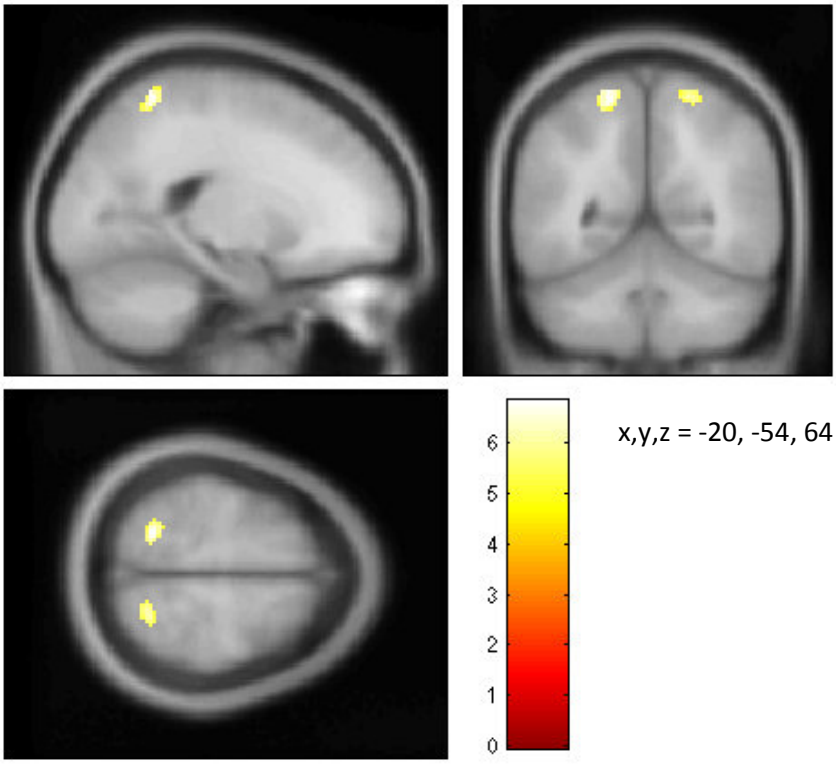

Figure 25. Study 2: Activity in the bilateral superior parietal lobule in the Near > Full contrast.

Neither the Miss > Win, Full > Near nor the interaction contrasts yielded significant activation at p_{FWE} < .05 and cluster extend 10.

At a more liberal threshold of $p_{uncorrected}$ < .0005 and cluster extend of 100 voxels, additional active areas were found in the Near > Full contrast. One cluster was located in the right inferior

parietal lobule and gyrus postcentralis and a second cluster in the right inferior frontal gyrus (see Table 16). In addition, significant clusters emerged in the Full > Near contrast (see Table 17).

Table 16. Study 2: Significant activations in the Near > Full contrast at $p_{uncorrected} < .0005$ and cluster extent 100

Region		MNI coordinates			Max *Z*-value	peak p_{FWE}	*n* Voxel
		x	y	z			
Superior Parietal Lobule (BA 7)	left	-20	-54	64	6.01	< .001	303
Superior Parietal Lobule (BA 7)	right	20	-56	64	5.47	.001	549
Precuneus (BA 7)	right	18	-72	50	4.28	.198	
Superior Parietal Lobule (BA 7)	right	24	-64	48	4.19	.269	
Postcentral Gyrus (BA 2)	right	60	-20	34	4.60	.063	543
Inferior Parietal Lobule (BA 40)	right	40	-40	48	4.58	.066	
Postcentral Gyrus (BA 2)	right	54	-30	46	4.01	.445	
Inferior Frontal Gyrus (BA 9)	right	52	6	28	4.46	.107	391
Inferior Frontal Gyrus (BA 9)	right	56	10	36	4.39	.136	

Note: MNI coordinates are given for peak voxels

Table 17. Study 2: Significant activations in the Full > Near contrast at $p_{uncorrected} < .0005$ and cluster extent 100

Region		MNI coordinates			Max *Z*-value	peak p_{FWE}	*n* Voxel
		x	y	z			
Cuneus (BA 18)	right	18	-98	8	4.43	.119	167
Middle Occipital Gyrus (BA 18)	right	24	-90	16	4.15	.298	
Extra-Nuclear	right	24	-14	24	4.37	.144	130
Caudate	left	-14	10	14	4.15	.304	107
Caudate	left	-12	18	2	4.05	.403	
Lingual Gyrus (BA 18)	left	-2	-82	-6	4.14	.306	136

Region		MNI coordinates			Max *Z*-value	peak p_{FWE}	*n* Voxel
		x	y	z			
Cuneus (BA 18)	left	-4	-100	4	4.10	.344	
Lingual Gyrus (BA 17)	left	-4	-94	-4	3.57	.911	

Note: MNI coordinates are given for peak voxels

The regression analysis of the Near > Full contrast with the KFG scores yielded no significant results at either the p < .05 FWE corrected level nor the p < .0005 uncorrected level. An additional exploratory analysis at an uncorrected threshold of p < .001 yielded several smaller clusters in which participants with higher KFG scores showed increased activation in the Near > Full contrast (see Table 18). The two largest clusters were located in the superior frontal gyrus (BA 8/BA 9, peak voxel left: *x, y, z* = -12, 28, 48, *Z* = 4.04, p_{FWE} = .560, 61 voxels; peak voxel right: *x, y, z* = 16, 42, 50, *Z* = 3.61, p_{FWE} = .955, 72 voxels; see Figure 26 and Figure 27).

Table 18. Study 2: Significant activations in the regression of the Near > Full contrast on KFG scores (positive correlation between KFG scores and activation in the Near > Full contrast) at $p_{uncorrected} < .001$

Region		MNI coordinates			Max *Z*-value	peak p_{FWE}	*n* Voxel
		x	y	z			
Superior Frontal Gyrus/Medial Frontal Gyrus (BA 8)	left	-12	28	48	4.04	.560	61
Postcentral Gyrus (BA 3)	left	-20	-36	50	3.79	.835	11
Middle Temporal Gyrus	right	48	-50	4	3.77	.853	11
Superior Frontal Gyrus (BA 6)	left	-12	28	62	3.71	.904	20
Extra-Nuclear	right	8	2	-6	3.70	.907	25
Superior Frontal Gyrus (BA 8)	right	16	42	50	3.61	.955	72
Superior Frontal Gyrus (BA 9)	left	-14	52	42	3.51	.983	29
Parahippocampal Gyrus (BA 19)	right	18	-52	-6	3.38	.997	13

Note: MNI coordinates are given for peak voxels. Only clusters comprising more than 3 voxels are shown.

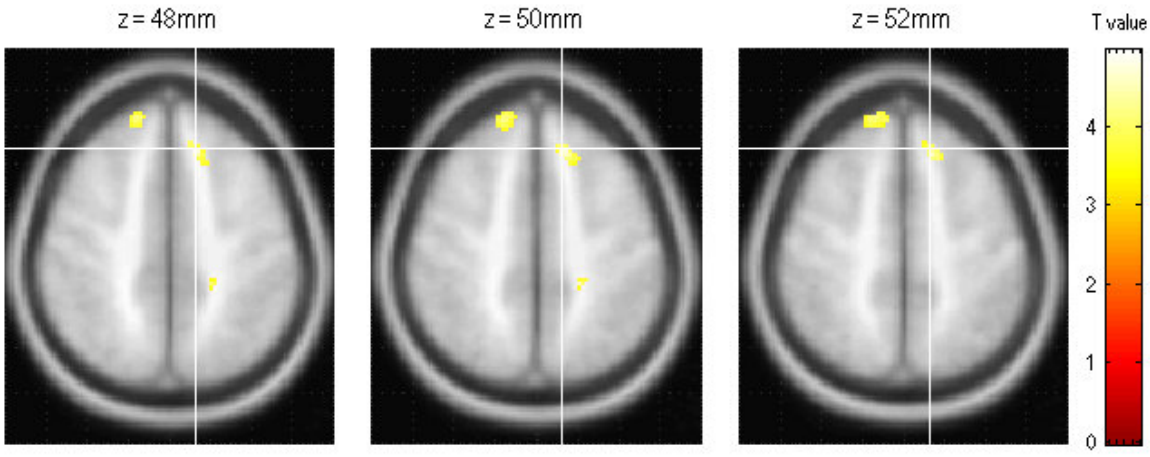

Figure 26. Study 2: KFG as positive predictor of activation in the Near > Full contrast in the bilateral superior frontal gyrus.

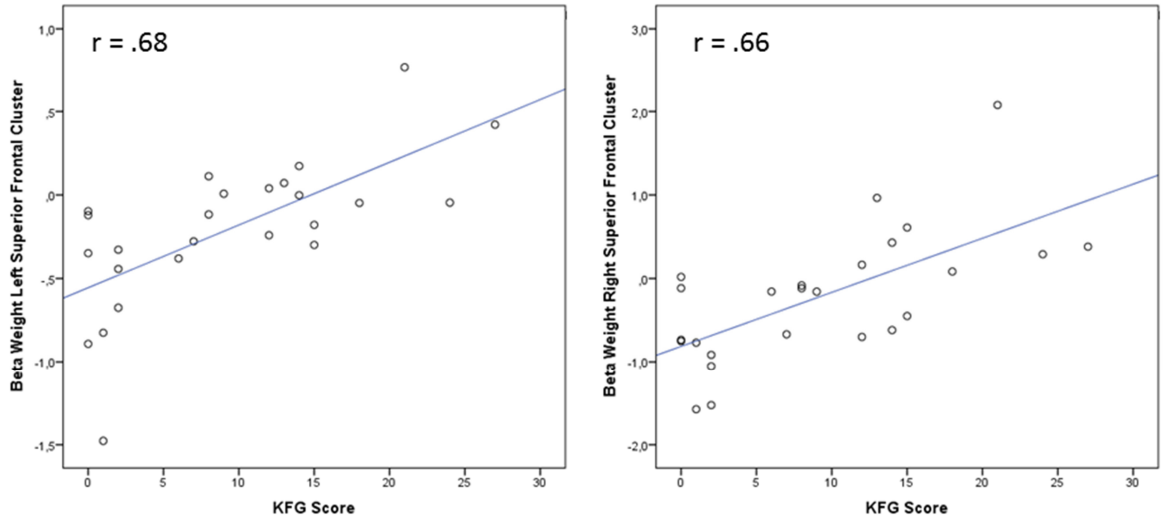

Figure 27. Study 2: Correlation of the beta weights extracted from the bilateral superior frontal clusters with the KFG scores.
Beta weights were extracted using the MarsBar-Toolbox (Brett, Anton, Valabregue, & Poline, 2002) for SPM.

5.3.2 Rating data

The analysis of the valence ratings yielded a significant main effect of „outcome“ ($F(1,29) = 170.49$, $p < .001$, $\eta^2_p = .86$), with wins being rated as more positive than misses. For the motivation ratings, a significant main effect of “outcome” ($F(1,29) = 38.23$, $p < .001$, $\eta^2_p = .57$) as well as a marginally significant main effect of “closeness” ($F(1,29) = 4.15$, $p = .051$, $\eta^2_p = .13$) were found. Wins were rated as more motivating than misses and full outcomes were rated as slightly more motivating than near outcomes. The analysis of the arousal ratings yielded no significant effects. For the probability ratings a significant main effect of “outcome” ($F(1,29) = 14.33$, $p = .001$, $\eta^2_p = .33$) was present. Participants rated their chance of winning higher following a win compared to a miss. Table

19 shows the full ANOVA results, while Figure 28 shows the mean value of each rating per outcome type.

Table 19. Study 2: Results of the ANOVAs for the valence, motivation, arousal and probability ratings

Dependent Variable & Effects	*F*(*df*)	*p*	η^2_p
Valence			
"Outcome"	170.49 (1,29)	< .001***	.86
"Closeness"	1.01 (1,29)	.324	.03
"Outcome" x "Closeness"	0.01 (1,29)	.910	< .01
Motivation			
"Outcome"	38.23 (1,29)	< .001***	.57
"Closeness"	4.15 (1,29)	.051	.13
"Outcome" x "Closeness"	0.22 (1,29)	.646	.01
Arousal			
"Outcome"	0.77 (1,29)	.389	.03
"Closeness"	1.09 (1,29)	.306	.04
"Outcome" x "Closeness"	1.28 (1,29)	.267	.04
Probability			
"Outcome"	14.33 (1,29)	.001**	.33
"Closeness"	0.16 (1,29)	.690	.01
"Outcome" x "Closeness"	0.08 (1,29)	.786	< .01

Note. *p < .05. **p < .01. ***p < .001

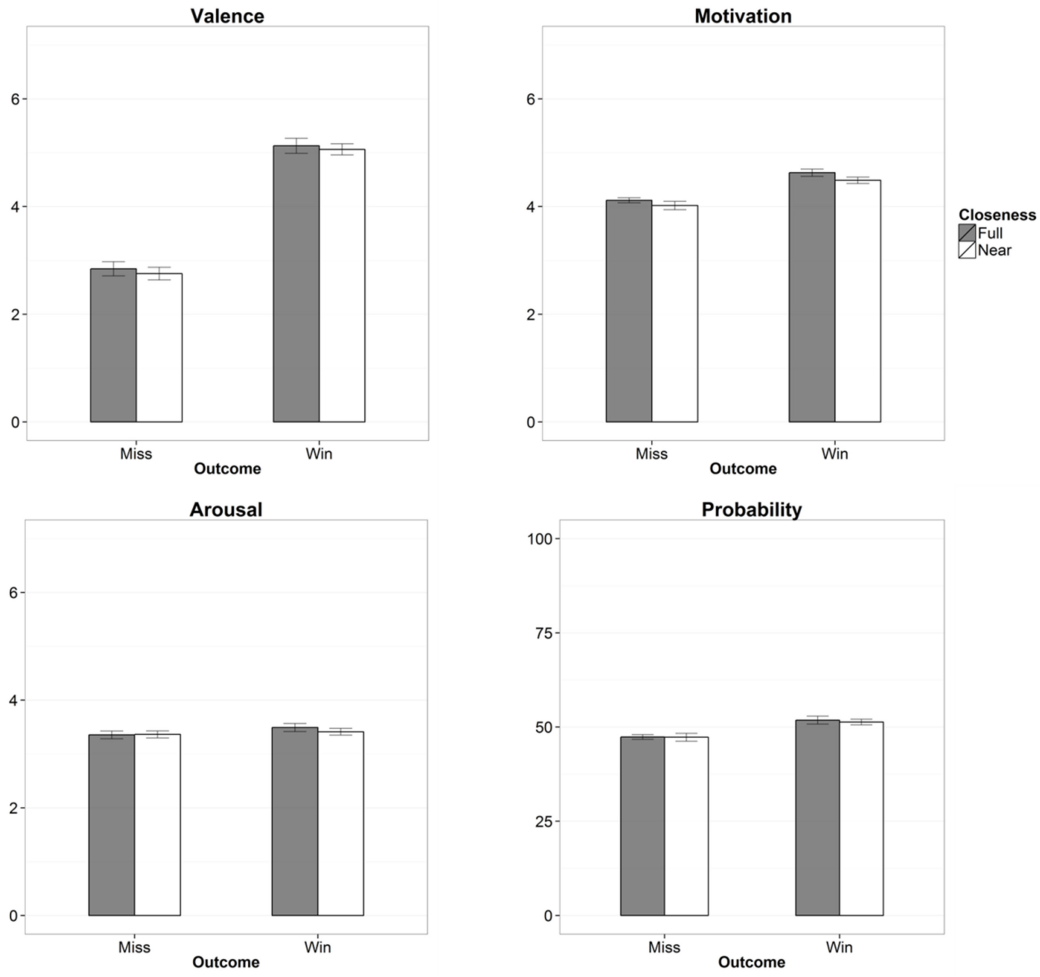

Figure 28. Study 2: Subjective ratings on valence, motivation, arousal and probability of winning in the next trial.
Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

5.3.3 Questionnaires

Table 20 shows the correlations between the KFG and the questionnaires assessing cognitive distortions, impulsivity and belief in good luck. KFG scores correlated (marginally) significantly with the GRCS sum score (r = .57, p = .014) as well as the subscales “Gambling Expectancies” (r = .50, p = .020), “Inability to Stop” (r = .49, p = .021), “Interpretative Bias” (r = .53, p = .019) and “Predictive Control” (r = .41, p = .054). Furthermore, the KFG scores correlated significantly with the UPPS subscale “Urgency” (r = .43, p = .047) and marginally significantly with the UPPS subscale “Perseverance” (r = -.36, p = .093). There were no significant correlations with achievement motivation and belief in good luck.

Table 20. Study 2: Correlations of the KFG scores with cognitive distortions, impulsivity, achievement motivation and belief in good luck

Questionnaire	Correlation coefficient (*p*-value)	*n*
GRCS		
"Gambling Expectancies"	.50 (.020)*	30
"Illusion of Control"	.22 (.315)	30
"Predictive Control"	.41 (.054)	30
"Inability to Stop"	.49 (.021)*	30
"Interpretative Bias"	.53 (.019)*	30
Sum	.57 (.014)*	30
UPPS		
"Urgency"	.43 (.047)*	30
"Premediation"	-.32 (.138)	30
"Perseverance"	-.36 (.093)	30
"Sensation Seeking"	.27 (.208)	30
AMS		
"Hope of Success"	-.05 (.845)	30
"Fear of Failure"	.03 (.891)	27
BIGL	.05 (.845)	30

Note. Due to missing responses some questionnaire scores could not be computed for all participants, explaining the different sample sizes for the correlations. *p*-values were adjusted using an FDR-correction according to Benjamini and Hochberg (1995). *$p < .05$. **$p < .01$. ***$p < .001$

5.4 Discussion

The analysis of the fMRI data on the wheel of fortune paradigm showed significantly greater activation following wins compared to misses in several clusters including the bilateral caudatum, the bilateral superior frontal gyrus (BA 8) and the left angular gyrus (BA 39). The activity following near compared to full outcomes was significantly greater in the bilateral superior parietal lobule (BA 7) and at a less stringent type I error correction also in the right postcentral (BA 2) and inferior frontal gyrus (BA 9). An exploratory analysis revealed that KFG scores predicted increased activity in the Near > Full contrast in the bilateral superior frontal gyrus (BA 8). The rating data mainly showed effects of the factor "outcome", with wins being rated as more pleasant and more motivating and leading to a stronger expectation of winning again in the following trial.

5.4.1 fMRI results

5.4.1.1 Contrast Win > Miss

Monetary wins compared to misses led to increased activity in the bilateral caudate, which has been shown to be involved in monetary reward processing in previous studies (Delgado, Nystrom, Fissell, Noll, & Fiez, 2000; Miedl, Fehr, Meyer, & Herrmann, 2010; Reuter et al., 2005). Other studies have shown the caudate to be involved in reward anticipation (Knutson, Adams, Fong, & Hommer, 2001; Knutson, Fong, Adams, Varner, & Hommer, 2001) and reward processing in general (Schultz, Tremblay, & Hollerman, 2000). Activity in the superior frontal cortex has also been associated with reward processing in previous research (Nieuwenhuis, Slagter, Alting von Geusau, Heslenfeld, & Holroyd, 2005), but also with decisions under uncertainty (Volz, Schubotz, & Cramon, 2005), craving (McClernon, Hiott, Huettel, & Rose, 2005; Rose et al., 2011) and processing of conflict (Ridderinkhof, Ullsperger, Crone, & Nieuwenhuis, 2004; Usami et al., 2013). The angular gyrus has also been shown to be involved in reward processing (Fliessbach et al., 2007; Liu, Hairston, Schrier, & Fan, 2011).

5.4.1.2 Contrast Near > Full

Contrary to the expectations, there was no significant activation in the ventral striatum, anterior insula or ACC in the Near > Full contrast. Thus, previous results (Chase & Clark, 2010; Clark et al., 2009; Dymond et al., 2014) showing activation in ventral striatum and anterior insula following near misses could not be replicated. One might argue that these results might only apply to misses and that near compared to full wins activate other brain areas. If that was the case, however, there should have been significantly activated voxels in the interaction contrasts, which was not the case. As such, the lack of differential activation in the ventral striatum by near and full outcomes fits with the rating data, which also did not show valence differences for near and full outcomes.

Near compared to full outcomes were followed by increased activity in the bilateral superior parietal lobule. This region encompasses the secondary somatosensory cortex (Trepel, 2008), and has been shown to be activated by looking at and reaching for objects and manipulating them (Hyvärinen & Poranen, 1974). Electrical stimulation in BA 7, temporarily inactivating the neurons, leads to deficits in visuospatial attention, as shown by marked shifts in a line bisection task (Vallar et al., 2014). This is in line with previous research showing the superior parietal lobule to be involved in visuospatial attention, together with frontal areas (for a review, see Kanwisher & Wojciulik, 2000). For example, Kastner, Pinsk, Weerd, Desimone, and Ungerleider (1999) found increased activation in the bilateral superior parietal lobule, together with activity in other parietal (intraparietal sulcus, inferior parietal lobule) and frontal areas (frontal eye field, supplementary eye field, middle frontal

gyrus) when participants covertly had to direct their attention to a space where a visual stimulus would later appear. With respect to near outcomes, the activity in the bilateral superior parietal lobule could indicate increased attention towards near compared to full outcomes, presumably because they require more attention to be processed correctly. Full outcomes, with the wheel stopping in the middle a color segment, can likely be identified very easily, whereas participants might have to take a closer look at near outcomes to figure out on which side of the color boundary the wheel stopped.

The superior parietal lobule and BA 7 have also been found to be involved in decision making in general (Huettel, 2006), as well as decision making under risk and ambiguity (Krain, Wilson, Arbuckle, Castellanos, & Milham, 2006) and perception of ambiguous conditioned stimuli (Bach, Seymour, & Dolan, 2009).

Another activation cluster was found in the right postcentral gyrus and inferior parietal lobule. The inferior parietal lobule has also been suggested to be involved in spatial perception (Rizzolatti & Matelli, 2003). However, as Husain and Nachev (2007) note in their review, the inferior parietal lobule is also involved in non-spatial processes. For example, Adler et al. (2001) found an increase in activation in the bilateral inferior parietal lobule during two different continuous performance tasks, suggesting a contribution of this area to vigilance. Similar results have previously also been reported in other studies (Pardo, Fox, & Raichle, 1991; Sturm et al., 1999).Furthermore, the inferior parietal lobule and the adjacent temporoparietal lobule have been shown to be involved in the processing of stimulus sequences, showing increased activity following the rare stimulus in an oddball sequence (Clark, Fannon, Lai, Benson, & Bauer, 2000; Downar, Crawley, Mikulis, & Davis, 2002).

Finally, an activation cluster was present in the right inferior frontal gyrus. This area, as well as adjacent frontal areas, has been implicated in counterfactual thinking. For example, De Brigard, Addis, Ford, Schacter, and Giovanello (2013) conducted a study where participants had to remember autobiographic events and create positive (i.e. envision a better outcome) or negative (i.e. envision a worse outcome) counterfactuals to the recollected events. Among other areas, counterfactual episodic thinking evoked activity in the right inferior frontal gyrus, irrespective of the valence of the counterfactual. In another study, Henderson and Norris (2013) analyzed brain activity following outcomes in a gambling task. In each trial, participants had to choose between two cards. They were also informed which outcomes were possible in the current trial, but not which outcome was behind which card. Every trial included either two possible wins or two possible losses. Following the choice of a card, the outcomes behind both cards were revealed, so participants did not only see their chosen outcome, but also the unchosen outcome. Four different outcome types were analyzed:

"Outright Wins" (i.e. winning the larger of the two possible wins), "Disappointing Wins" (i.e. winning the smaller of the two possible wins), "Relieving Losses" (i.e. losing the smaller of the two amounts), and "Outright Losses" (i.e. losing the larger of the two amounts). Outcomes involving mixed emotions, that is, "Disappointing Wins" and "Relieving Losses" elicited greater activity in the bilateral posterior parietal cortex (BA 40) and the right dorsolateral prefrontal cortex (BA 6/8). Similar patterns of activation have been found in the Near > Full contrast, indicating that near outcomes might also initially elicit mixed emotions. However, no difference between the valence of near and full outcomes was found in the rating data, suggesting that the potential mixed emotional state is only short lived in this paradigm.

In sum, the analysis showed activated areas in the parietal cortex, extending into frontal areas in the right hemisphere. These areas were more activated following near outcomes in general, thus there was no brain area that reacted specifically to only near misses or near wins. As such, these findings mirror previous EEG results (Ulrich & Hewig, 2014; Weiß, 2014) which also showed main effects for close outcomes but no interaction with their valence. The balanced probabilities used in the wheel of fortune paradigm (50% wins, 50% misses, 50% full outcomes, 50% near outcomes) might have led people to specifically pay attention to the valence and closeness categories and their dichotomous structure, thus contributing to the observed main effects. Future research could test the reactions to near wins and misses which occur more seldom.

5.4.1.3 Correlation of brain activity following near outcomes with gambling problems

The regression analysis with the KFG scores as predictor variable and the Near > Full contrast as criterion only yielded results at a liberal significance threshold and thus should be interpreted with caution and tested in further studies. The two largest clusters found in this analysis were located bilaterally in the superior frontal gyrus (BA 8). With increasing KFG scores, participants showed increased activity to near comparted to full outcomes in these areas. This areas are also adjacent the frontal cluster found for mixed outcomes by Henderson and Norris (2013), indicating that participants with signs of gambling problems might show more ambivalent emotions after near outcomes than those without gambling problems.

5.4.2 Rating data

As in the current EEG study and a previous study (Ulrich & Hewig, 2014), there was a significant difference in self-reported valence of wins and misses, with wins being rated as more pleasant. However, there was no effect of closeness on the valence ratings. This result does not support the hypothesis derived from previous studies, which have reported lower valence ratings for near compared to full misses (Clark et al., 2009; Qi et al., 2011). However, the lack of a closeness

effect on the valence ratings in the wheel of fortune seems to be pretty robust and previous studies reporting valence differences between near and full misses have used different paradigms. Hence, the observed difference between studies could be due to different paradigms, specifically due to different probabilities of the outcome types (see below).

For the motivation ratings, a significant effect of outcome was present, with wins being rated as more motivating than misses. In addition, there was a marginally significant main effect of closeness, with near outcomes being rated as slightly less motivating than full outcomes. Previous research on near misses has overall shown them to be rated as more motivating compared to full misses (Clark et al., 2009; Qi et al., 2011). Since previous studies using the wheel of fortune paradigm (current EEG study described in section 4; Ulrich & Hewig, 2014) have not shown an effect of closeness on motivation ratings, the current result should be interpreted with caution. Roese (1997) suggested that close outcomes are more likely to elicit counterfactual thinking. Counterfactual thinking refers to imagining alternatives to past experienced outcomes (Markman, Gavanski, Sherman, & Mcmullen, 1993; Roese, 1997). Counterfactual thinking can be further divided into upward and downward counterfactual thinking, depending on whether the imagined alternative outcome is better or worse than the one actually experienced (Markman et al., 1993). Both kinds of counterfactuals have been shown to lead to decreases in motivation under specific circumstances. For example, Dyczewski and Markman (2012) could show that upward counterfactuals induce a decrease in motivation when people generally have the feeling of not being able to influence the outcome through their actions. McMullen and Markman (2000) showed that downward counterfactuals can also yield decreased motivation when they result in positive affect. The motivating effect of near misses in previous studies (e.g. Clark et al., 2009) might also be influenced by the outcome probabilities used. For example, in the study by Clark et al. (2009) wins occurred on 1/6 of the trials and as such were more seldom than misses. In such a paradigm, a near miss might be motivating, because it supposedly indicates that the win (the relatively rare event) is about to occur again. However, in the wheel of fortune paradigm, wins occur in half of the trials and constitute a common outcome. Thus, in this case near misses might no longer have the motivational property reported in previous studies.

For probability ratings there was a significant effect out outcome, with the chance of winning being rated higher following wins compared to misses. This is similar to a hot hand belief (e.g. Burns & Corpus, 2004), where winning again is thought to be more likely following a streak of previous wins. Similar results have also been reported in previous studies (Porchet et al., 2013; Studer, Limbrick-Oldfield, & Clark, 2015).

5.4.3 Questionnaires

As expected, there were positive correlations between a screening questionnaire for gambling problems and a questionnaire assessing gambling-related cognitive distortions. This is in line with previous results showing increased cognitive distortions in problem and pathological gamblers (e.g. Joukhador et al., 2004; MacLaren et al., 2015; Myrseth et al., 2010).

However, there was no significant correlation between gambling problems and belief in good luck. As such, this result is not in line with the literature (Chiu & Storm, 2010; Wohl et al., 2007) and the results obtained in the EEG study. However, these studies involved group comparisons of pathological and problem gamblers, and controls while the present study used a correlational design with the participants mostly being in the non-problematic range of gambling behavior. It is conceivable that an increased belief in good luck is not related linearly to gambling problems, but can only be observed at certain higher levels of gambling problems. This could be tested in future studies[11].

As in the EEG study, the current results fit with previous results of increased self-reported impulsivity in pathological gamblers (e.g. Barrault & Varescon, 2013; Kräplin et al., 2014; Lawrence et al., 2009a; Michalczuk et al., 2011; Vitaro et al., 1999) and again replicates the specific association of the UPPS "Urgency" subscale with gambling problems (Whiteside et al., 2005).

Finally, there was no correlation between gambling problems and achievement motivation as measured by the AMS. The current sample might not have included enough participants in the upper range of gambling problems to detect any relations. However, as the group comparison in the EEG study also yielded no differences in achievement motivation between problem gamblers and controls, the combined results of the two studies suggest that the amount of gambling problems is not related to achievement motivation.

5.4.4 Limitations

Some limitations have to be kept in mind when interpreting the study results. Firstly, the sample included a somewhat restricted range of problem gamblers, since there were only five participants out of the 26 analyzed who had a KFG score above the cutoff score for probable pathological gambling (KFG score of 16), with one participant being an outlier and thus being removed from the regression analysis. Thus, further relations between gambling problems and near outcome processing might have been missed in the analysis due to restricted variance. Finally, as is

[11] First evidence in support of this hypothesis can be gained from an exploratory analysis of the data in study 1. In fact, within the control group, the correlation between the KFG and BIGL is very small (r = -.10 p = .683), whereas it is moderate in the problem gambling group (r = .38 p = .100).

the case in many fMRI studies, the current results were interpreted using reverse inference (Poldrack, 2006), that is, based on the activity of certain brain areas, certain cognitive states were assumed to be present. As such, the activation of a brain area is no absolute proof that a certain cognitive process is involved. However, hypotheses can be derived from such observations, which can then be tested in further experiments.

5.5 Summary

To sum up, the fMRI results showed that near compared to full outcomes elicited increased activity in the superior parietal lobule, an area linked to visual attention, suggesting that near outcomes draw more attention than full outcomes. Gambling problems did not correlate strongly with the processing of near outcomes. However, an exploratory analysis showed increased activity in the bilateral superior frontal cortex for near compared to full outcomes with increasing gambling problems, potentially indicative of increased counterfactual thinking following near outcomes with increasing gambling problems. The questionnaire results showed increased cognitive distortions and urgency with increasing gambling problems.

6 Study 3: Processing of near outcomes in gambling (peripheral physiology)

6.1 Introduction

The third study analyzed the processing of near outcomes using measures of peripheral physiology, specifically heart period and skin conductance. As in the second study, problem gambling was included as a continuous variable. An introduction to heart period and skin conductance can be found in sections 3.1.3 and 3.1.4.

6.1.1 Summary of previous results

As described in the general introduction, a couple of studies have used SCR and heart-related measures to assess autonomous reactions to near misses in gambling. Taken together, those studies suggest that near compared to full misses lead to increased physiological arousal as indexed by increased SCRs and differences in phasic heart rate responses (Clark et al., 2012; Clark et al., 2013; Dixon et al., 2011; Dixon et al., 2013). Concerning heart rate, the studies showed a more or less clear biphasic response to the gambling outcomes, with an initial heart rate decrease and a subsequent heart rate increase. One study found an increased heart period increase (corresponding to an increased heart rate decrease) following near compared to full misses (Dixon et al., 2011), whereas another study found an increased subsequent heart rate increase (Clark et al., 2012) and a third study reported no difference in either art of the response (Clark et al., 2013).

No studies have included near wins so far. Thus, as with the EEG and fMRI studies, an experiment using a balanced paradigm, containing both near misses and near wins, was used to further elucidate the processing of near outcomes in general. Similar to the P300 in the EEG, SCRs have been shown to be sensitive to stimulus properties, with larger SCRs being caused by rare stimuli (Ben-Shakhar et al., 1982). Using the wheel of fortune paradigm thus allows an analysis of SCRs following outcomes that is not confounded by stimulus probability.

6.1.2 Gambling and risk-taking

Risk and risky decisions refer to decisions with uncertain outcomes - there is a certain probability to obtain a positive outcome and a certain probability to obtain a negative outcome. As such, gambling itself is a risky decision. Several studies have investigated the relation between pathological gambling and risk-taking.

Mishra, Lalumière, and Williams (2010) showed that self-reported risk-taking across several domains is positively related to the severity of gambling problems. Descriptively, this relation was largest for risk-taking in the gambling domain and smallest for risk-taking in the social domain. A

similar result has been reported by Powell, Hardoon, Derevensky, and Gupta (1999), who found increased self-reported risk-taking in pathological gamblers. Further studies showed impaired decision making of problem and pathological gamblers in the Iowa Gambling Task (Kertzman, Lidogoster, Aizer, Kotler, & Dannon, 2011; Lorains et al., 2014) and a loss aversion task (Lorains et al., 2014), as well as a general shift towards choosing more risky options, irrespective of the associated winning probabilities (Ligneul, Sescousse, Barbalat, Domenech, & Dreher, 2013). These deficits might change once gamblers are in treatment, as Brañas-Garza, Georgantzís, and Guillen (2007) showed less risk-taking in a lottery task in pathological gamblers undergoing treatment. However, most behavioral studies showing deficits in decision making and increased risk-taking in pathological gamblers have focused on gambling-related decisions. Hence, it is still unclear, whether pathological and problem gambling is related to generally increased risk-taking or increased risk-taking related to the gambling domain.

6.1.3 Current study

The current study analyzed the processing and evaluation of near and full outcomes using heart period and skin conductance. Gambling problems were assessed and integrated into the analysis as a continuous variable to analyze potential influences on the processing of near outcomes. Furthermore, the relation between gambling problems and self-reported cognitive distortions, impulsivity and risk-taking was evaluated.

6.1.4 Hypotheses

It was hypothesized that near outcomes should elicit stronger physiological reactions than full outcomes. More specifically, the heart period response and the SCR should be increased relative to full outcomes. Based on the heterogeneous results of previous research, no clear hypothesis on which part of the biphasic heart rate response would be affected by closeness of the outcomes could be derived. Furthermore, a relation to gambling problems was expected, with participants with higher scores on the gambling problem screening questionnaires showing increased physiological reactions to near outcomes. Finally, a self-reported risk-taking questionnaire was analyzed, to answer the question whether participants with increased gambling problems show generally increased risk-taking behavior or whether the increased risk-taking is restricted to the gambling domain.

6.2 Methods

6.2.1 Participants

For this experiment 50 male participants were recruited. The sample comprised both participants with potential problem gambling and without gambling problems. As problem gambling has been shown to be more prevalent in males (Bundeszentrale für gesundheitliche Aufklärung, 2014; Erbas & Buchner, 2012), we focused on males only in the current study. Participants were recruited via online advertisement on a local website. Interested participants then filled in an online survey consisting of demographic questions (age, gender, handedness, education) and two screening questionnaires for gambling problems, the KFG (Petry, 1996) and the SOGS (Lesieur & Blume, 1987). Based on their score on the gambling screenings, participants were then invited to take part in the experiment. We aimed at including participants with a broader range of scores on the screening questionnaires, thus we approximately recruited half of the participants from above and below the questionnaires' cutoff points for problem gambling. The cutoff for problem gambling in the KFG is set at a score of 16 (Petry, 1996). The corresponding score in the SOGS is set at 5 (Lesieur & Blume, 1987). The final sample consisted of 50 participants (mean age = 27.78, *SD* = 9.12, Range: 18-56) with 20 and 21 participants scoring at or above the cutoff score in the SOGS and KFG respectively. The mean scores for SOGS and KFG were 3.82 (*SD* = 3.10) and 13.38 (*SD* = 8.05). The correlation between the two questionnaires was r = 0.652. Participants received a reimbursement of 10.00 € plus an extra 2.00 € for their performance in the wheel of fortune (see below).

6.2.2 Paradigm: Wheel of fortune

The wheel of fortune paradigm used is similar to the one in the EEG and fMRI studies. However, some changes were implemented. First of all, the wheel already spun while the participants were making their color choice. Secondly, the amount of the fixed bet was raised to 40 Cents. Thirdly, the wheel did not stop abruptly, but decelerated to a standstill. Finally, during the ITI, the running total was shown. Figure 29 summarizes the sequence of a trial. The participants first had to choose either orange or turquoise as the color to place a bet on while the wheel of fortune was spinning[12]. After the choice, the wheel kept spinning at a constant speed for a variable time (2.13-2.42 s) before decelerating to a standstill. The deceleration phase always lasted 4.47 s, the entire phase from color choice to outcome presentation was 6.75 s long on average across all participants (6.60 - 6.89 s). The outcome was presented for 4 seconds and was followed by an ITI that randomly lasted for 6-8 s.

[12] As in studies 1 and 2 the spinning motion was created by rapid serial presentation of 72 pictures, each showing a 5° change in the position of the wheel of fortune, relative to the previous picture. Each picture was presented for 16.7 ms, corresponding to the 60Hz refresh rate of the screen.

Participants started with an account balance of 200 points and were told that they received an additional 2.00 € bonus payment if they ended the game with more than 320 points. Altogether, participants played 91 trials on the wheel of fortune, the first three of which served as practice trials, after which the account balance was reset to 200 points. The final eight trials were not analyzed and always consisted of a fixed sequence of outcomes to ensure that participants ended the game with 360 points, thus entitling them for the bonus payment. The 80 trials in between practice and end sequence consisted of 20 full wins, full misses, near wins and near misses each. The near outcomes were each subdivided into 10 outcomes stopping before or after the color boundary (thus resulting in 10 near wins before boundary, near wins after boundary, near misses before boundary and near misses after boundary, respectively). The sequence of outcomes was determined randomly for every participant. As in the EEG and fMRI versions of the wheel of fortune, two different stopping positions for the wheel were used for full outcomes- one 20° away from the previous color boundary, the other 25° away from the previous color boundary.

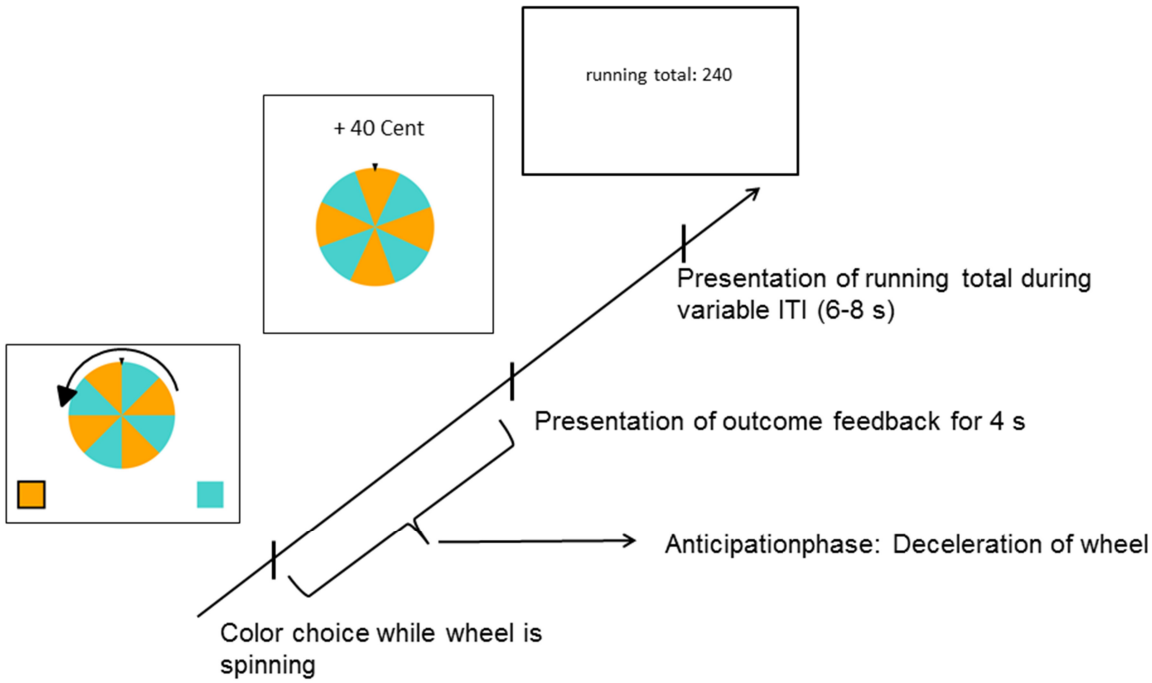

Figure 29. Study 3: Sample trial of the wheel of fortune used in the peripheral physiology study.

6.2.3 Questionnaires

The questionnaires included a set of follow-up questions (see Annex C), which referred to the wheel of fortune. Participants were asked whether they had had a specific strategy in the game, whether they had preferred one of the colors over the other, whether they had participated in a

similar experiment before, what they thought was the aim of the study, and whether they had noticed that some of the outcomes were close, that is, stopped close to a color boundary.

Additionally, the following questionnaires were filled in: GRCS (Raylu & Oei, 2004), UPPS (Keye et al., 2009) and the DOSPERT (Johnson et al., 2004).

6.2.4 Procedure

Upon entering the lab, participants first received written information on the procedure of the experiment and then filled in an informed consent form, before ECG and skin conductance electrodes were applied. Then participants played on the wheel of fortune and finally filled in several questionnaires at the end of the experiment.

6.2.5 Heart period and skin conductance measurement

Heart period and skin conductance were measured using a BrainVision BrainAmp ExG amplifier (Brain Products GmbH, Gilching, Germany) and the BrainVision Recorder 1.20 software (Brain Products GmbH). The sampling rate was set to 250Hz. Three disposable Ag/AgCl electrodes (Kendall ECG Electrodes H98LG, Covidien, Neustadt, Germany) were used to measure heart period. The electrodes were placed according to a modified Einthoven II lead, with the ground electrode placed below the left collarbone, the negative electrode placed below the right collarbone and the positive electrode placed on the left side below the rib cage. Skin conductance was measured via two Ag/AgCl electrodes (diameter of contact area between skin and electrode paste: 7mm) placed on the fingertips of the left index and middle finger respectively. The electrodes were filled with TD-246 Skin Conductance Electrode Paste (0.5% saline in neutral base, Discount Disposables, St. Albans, Vermont).

6.2.6 Heart period analysis

First, R-peaks in the raw ECG signal were detected using QRS-Tools (Allen, Chambers, & Towers, 2007). The automatically detected R-peaks were then visually inspected for every participant and corrected for ectopic beats, if necessary. Then, the time-series of R-peaks was exported.

A custom-built Matlab script was used to compute event-related IBIs linked to outcome onset. For every outcome type, this script extracted the IBI in which the outcome onset occurred (in the following denoted as “IBI around”), as well as six IBIs before and after this particular IBI (labelled “IBI-6” to “IBI-1” and “IBI+1” to “IBI+6”), resulting in 13 IBIs per outcome. For each person the average IBI values were computed per outcome type.

To assess the relation between heart period reaction and gambling problems, a single measure of IBI reactivity was computed. This measure quantifies the IBI change around outcome

onset and consisted of the average difference (maximum-minimum) between the minimum IBIs before and after outcome onset and the maximum IBI in between the detected minima [((maximum-$minimum_{before}$)+(maximum-$minimum_{after}$))/2]. This measure was extracted on a single trial basis for every participant and then averaged for every outcome type.

6.2.7 Skin conductance analysis

Skin conductance data were analyzed using a custom-built Matlab script. First, the data were filtered with a 5^{th} order 2.525 Hz lowpass Butterworth filter and a 5^{th} order 0.05 Hz highpass Butterworth filter (Figner & Murphy, 2011). The magnitude of the SCR was evaluated as the largest difference between a local minimum and a subsequent local maximum in the time window 0 to 5 post outcome onset for every outcome[13]. For trials without a local minimum in this time window, SCR was scored as the difference between the maximum in the time window minus the skin conductance value at time 0. For trials without a local minimum followed by local maximum (e.g. trials with a monotone decline of the skin conductance) the SCR was scored as 0. Those magnitudes were then transformed using the natural logarithm of the magnitudes + 1 (ln(x+1)) (Dawson et al., 2007). For every participant the ln(x+1) SCR values were then averaged across trials for each outcome type.

6.2.8 Statistical analysis

The event-related IBIs were analyzed using a three factorial 2*2*7 repeated measures ANOVA with the factors “outcome” (win vs. miss), “closeness” (full vs. near) and “IBI-time” (IBI-around vs. IBI+1 vs. IBI+2 vs. IBI+3 vs. IBI+4 vs. IBI+5 vs. IBI+6). Where necessary, a Greenhouse-Geisser correction was applied to the *p*-values. Post-hoc paired t-tests were used to analyze significant interactions. The corresponding *p*-values were adjusted to an FDR of 0.05 (Benjamini & Hochberg, 1995). Partial eta-square values are reported as estimates of effect size.

Ln(x+1) transformed SCRs were entered into a two factorial 2*2 repeated measures ANOVA with the factors “outcome” (win vs. miss) and “closeness” (full vs. near).

In addition, for both IBIs and SCRs exploratory analyses were run to test whether there were any differences between near outcomes that stopped before vs. after the color boundary, as previous research has shown that this might be the case for near misses (Clark et al., 2013; Clark et al., 2009). First, a 2*2*7 repeated measures ANOVAs including only near outcomes was run with the factors “outcome” (win vs. miss), “position” (before vs. after color boundary) and “IBI-time” (IBI-around vs. IBI+1 vs. IBI+2 vs. IBI+3 vs. IBI+4 vs. IBI+5 vs. IBI+6) and IBI as dependent variable. Then, a

[13] We chose a longer time window compared to commonly used 1-3 s or 1-4 s poststimulus (Dawson et al., 2007), since the wheel of fortune paradigm used in this study included a deceleration phase. This creates the possibility that participants realized what their outcome was at different times throughout the trial.

2*2 repeated measures ANOVA with the factors “outcome” (win vs. miss) and “position (before vs. after color boundary) with SCR as dependent measure was run. Where necessary, a Greenhouse-Geisser correction was applied to the *p*-values. Additionally, the initial analyses for event-related IBIs and SCRs were repeated separately with near outcomes before and after the color boundary.

To assess the relation between gambling problems and physiological reactivity to gambling outcomes, the heart period reaction measure described above and SCR for every outcome type were entered in two ANCOVAs with the factors “outcome” (win vs. miss) and “closeness” (near vs. full), including the z-standardized KFG-scores as covariate.

To analyze the relation between gambling problems and cognitive distortions, impulsivity, and risk-taking, Pearson correlations between the KFG scores and the other questionnaires were computed. The corresponding *p*-values were adjusted for multiple comparisons by setting the FDR to 0.05 (Benjamini & Hochberg, 1995).

The computation of the ANCOVAs was done in SPSS 21 (IBM), while all other computations were done in R (R Core Team, 2014).

6.3 Results

6.3.1 Heart period

Table 21 shows the results of the ANOVA with IBIs as dependent variable. Figure 30 depicts the time course of the event-related IBIs around outcome onset. The analysis of the IBIs around and following outcome onset revealed significant main effects of “outcome” ($F(1,49) = 11.19$, $p = .002$, $\eta^2_p = .19$), and “IBI number” ($F(6,294) = 88.88$, $p < .001$, $\eta^2_p = .64$), as well as a significant interaction of “IBI number” and “closeness” ($F(6,294) = 4.55$, $p < .001$, $\eta^2_p = .08$). Follow-up paired t-tests with adjusted *p*-values according to Benjamini and Hochberg (1995) showed that full and near outcomes differed significantly at IBI+1 ($t(49) = 3.39$, $p = .010$) and IBI+2 ($t(49) = 2.72$, $p = .032$) with near outcomes showing larger IBIs. Furthermore, misses elicited larger IBIs than wins and IBIs decreased over time.

The ANOVA including only near outcomes before and after the color boundary yielded significant main effects of “outcome” ($F(1,49) = 4.06$, $p = .049$, $\eta^2_p = .08$) and “IBI number” ($F(6,294) = 85.98$, $p < .001$, $\eta^2_p = .64$) as well as a marginally significant interaction of “position” and “IBI number” ($F(6,294) = 2.69$, $p = .05$, $\eta^2_p = .05$). The full ANOVA results are depicted in Table 22. Descriptively, near outcomes which stopped directly after the color boundary, elicited slightly larger IBIs around outcome-onset than IBIs stopping just before the boundary. However, running the main ANOVA

separately for near outcomes before and after the color boundary did not qualitatively change the results compared to the ANOVA with overall near outcomes.

Table 21. Study 3: Results of the main ANOVAs for the dependent measures IBI and SCR

Dependent Variable & Effects	*F* (*df*)	*p*	η^2_p
IBI			
"Outcome"	11.19 (1, 49)	.002**	.19
"Closeness"	3.13 (1, 49)	.083	.06
"IBI Number"	88.88 (6, 294)	< .001***	.64
"Outcome" x "Closeness"	0.20 (1, 49)	.657	< .01
"Outcome" x "IBI Number"	0.95 (6, 294)	.399	.02
"Closeness" x "IBI Number"	4.55 (6, 294)	.004**	.08
"Outcome" x "Closeness" x "IBI Number"	1.17 (6, 294)	.324	.02
SCR			
"Outcome"	0.18 (1, 49)	.671	< .01
"Closeness"	0.22 (1, 49)	.641	< .01
"Outcome" x "Closeness"	3.44 (1, 49)	.070	.07

Note. p-values for the following effects in the IBI ANOVA were Greenhouse-Geisser corrected: IBI Number, Outcome x IBI Number, Closeness x IBI Number, Outcome x Closeness x IBI Number. *$p < .05$. **$p < .01$. ***$p < .001$

Table 22. Study 3: Results of the additional ANOVAs concerning near outcomes before and after the color boundary for the dependent measures IBI and SCR

Dependent Variable & Effects	*F* (*df*)	*p*	η^2_p
IBI			
"Outcome"	4.06 (1, 49)	.049*	.08
"Position"	0.02 (1, 49)	.881	< .01
"IBI Number"	85.98 (6, 294)	< .001***	.64
"Outcome" x "Position"	0.08 (1, 49)	.783	< .01
"Outcome" x "IBI Number"	0.12 (6, 294)	.920	< .01
"Position" x "IBI Number"	2.69 (6, 294)	.050	.05
"Outcome" x "Position" x "IBI Number"	0.49 (6, 294)	.607	.01
SCR			
"Outcome"	1.92 (1, 49)	.172	.04
"Position"	0.20 (1, 49)	.658	< .01
"Outcome" x "Position"	0.10 (1, 49)	.756	< .01

Note. p-values for the following effects in the IBI ANOVA were Greenhouse-Geisser corrected: IBI Number, Outcome x IBI Number, Position x IBI Number, Outcome x Position x IBI Number.
*$p < .05$. **$p < .01$. ***$p < .001$

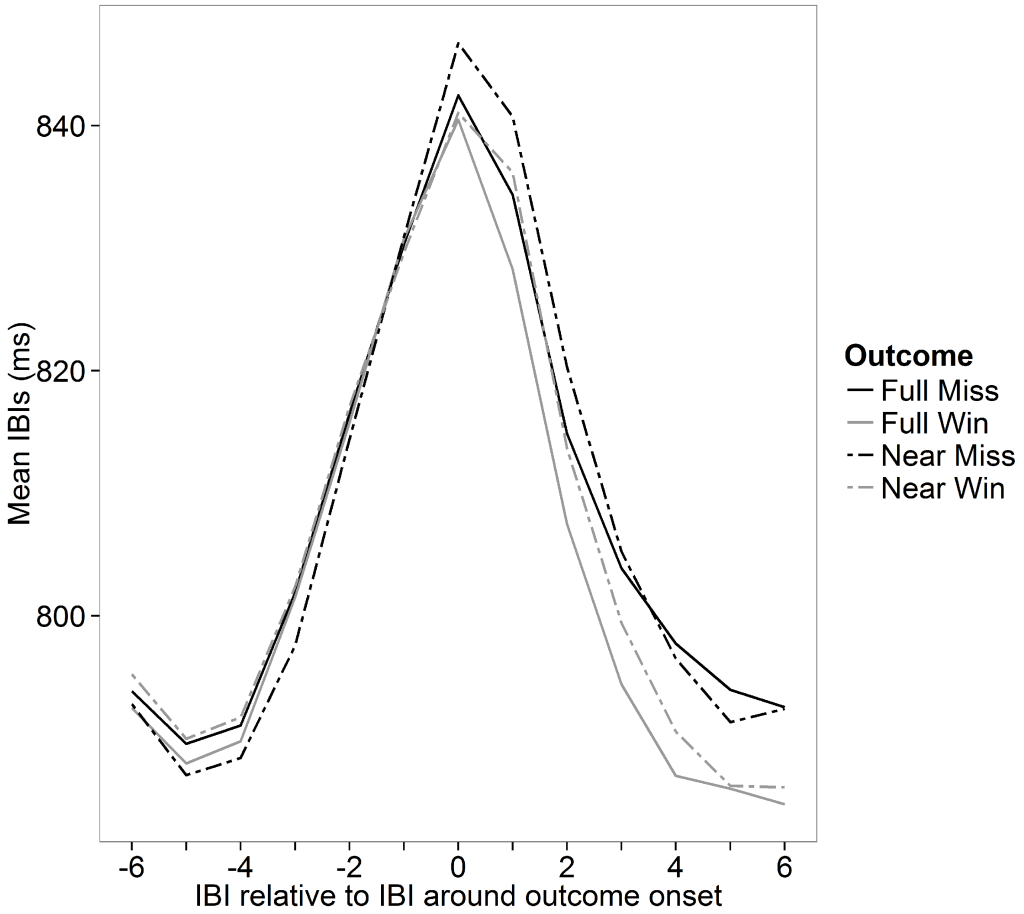

Figure 30. Study 3: Mean IBI course around outcome onset.

6.3.2 Skin conductance

Table 21 depicts the results of the main ANOVA with ln(x+1) SCR as dependent variable, while Figure 31 shows the mean ln(x+1) SCR. Except for a marginally significant interaction of "outcome" and "closeness" ($F(1,49) = 3.44$, $p = .070$, $\eta^2_p = .07$), the ANOVA yielded no significant results. Descriptively, near misses ($M = 0.046$) elicited larger SCRs than full misses ($M = 0.040$), whereas near wins ($M = 0.040$) elicited smaller SCRs than full wins ($M = 0.043$).

The exploratory analysis of differences between near outcomes before and after the color boundary yielded no significant effects (see Table 22). Rerunning the original ANOVA to analyze SCRs separately with near outcomes before and after the color boundary, yielded a significant interaction of "outcome" and "closeness" " ($F(1,49) = 4.56$, $p = .038$, $\eta^2_p = .09$) when only near outcomes stopping before the boundary were included. However, post-hoc paired t-tests did not show any significant differences between the outcome types (all corrected $p > .282$). Figure 32 shows the mean ln(x+1) SCR including only near outcomes stopping before the color boundary. Rerunning the ANOVA with near outcomes stopping after the color boundary yielded no significant effects.

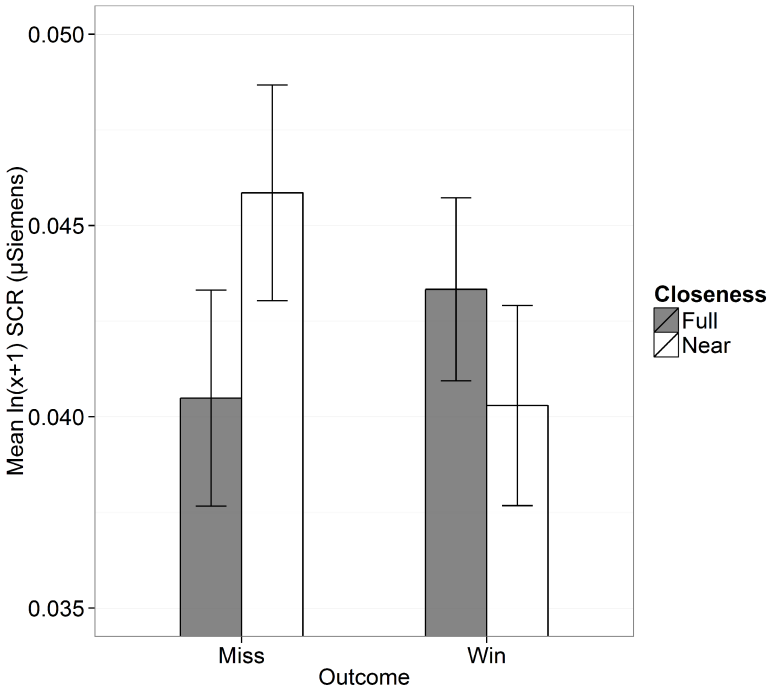

Figure 31. Study 3: Mean ln(x+1) SCR following full wins, full misses, near wins and near misses, respectively.
Error bars denote standard errors of the mean adjusted for within-subject designs according to Cousineau (2005) and Morey (2008).

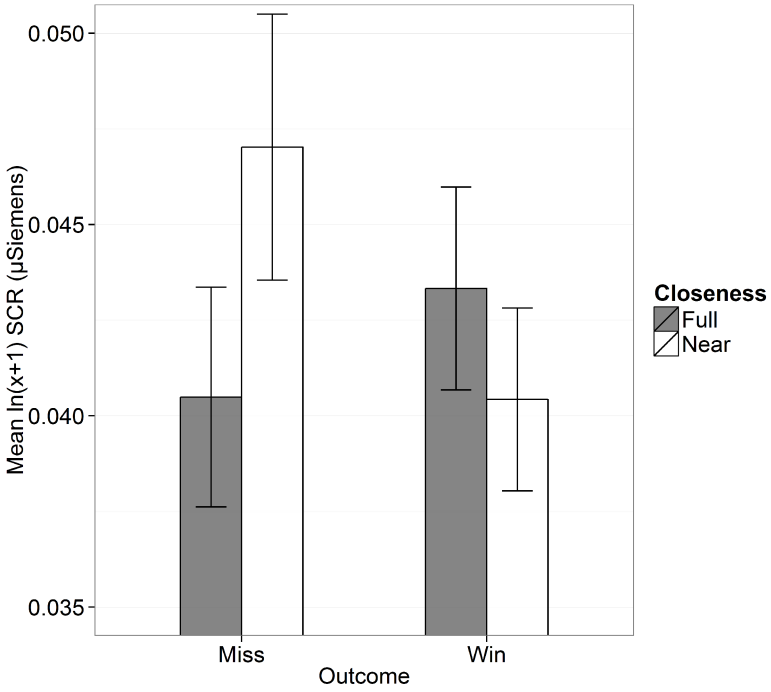

Figure 32. Study 3: Mean ln(x+1) SCR values including only near outcomes stopping before the color boundary.
Error bars denote standard errors of the mean adjusted for within-subject designs according to Cousineau (2005) and Morey (2008).

6.3.3 Correlations with gambling status

The results of the ANCOVAs for IBI reaction and SCR are depicted in Table 23. For mean IBI reaction there was a significant three-way interaction between "outcome", "closeness", and KFG ($F(1,48) = 4.20$, $p = .046$, $\eta^2_p = .08$), a marginal main effect of the covariate KFG ($F(1,48) = 3.28$, $p = .077$, $\eta^2_p = .06$), and a marginal interaction between "outcome" and KFG ($F(1,48) = 3.68$, $p = .061$, $\eta^2_p = .07$). To further analyze the significant three way interaction, we computed scores to quantify the effect of closeness separately for wins and misses and to quantify the effect of outcome separately for full and near outcomes. Specifically, we subtracted the mean IBI reaction following full outcomes from the mean IBI reaction following near outcomes for wins and misses separately and we subtracted the mean IBI reaction following miss outcomes from the mean IBI reaction following win outcomes for full and near outcomes separately. We then correlated each of the four difference measures with the KFG score; *p*-values were adjusted according to Benjamini and Hochberg (1995). Figure 33 depicts the four respective scatterplots. There were significant correlations between the closeness effect for misses and the KFG ($r = -.352$, $p = .024$) as well as the outcome effect for full outcomes and the KFG ($r = -.361$, $p = .024$). Participants with higher KFG scores showed decreased differences between near and full misses. As the scatterplot (see Figure 33A) indicates, this difference becomes negative for participants with higher KFG scores, indicating that they show a smaller IBI reaction to near compared to full misses. Regarding the correlation with the outcome effect for full outcomes, participants with higher KFG scores showed a decreased difference between full wins and misses. The scatterplot (see Figure 33C) shows that this difference also becomes negative for participants with high KFG scores, indicating that they show a smaller IBI reaction to full wins compared to full misses. Figure 34 descriptively shows the mean IBI reaction for each outcome type for participants with high (>= 1) and low (<= -1) KFG z-scores (n high = 8, n low = 10). As can be seen, participants with low KFG scores react most strongly to full wins, whereas participants with high KFG scores react most strongly to full misses, with little difference between the other outcomes.

The ANCOVA with SCR as dependent variable yielded a significant interaction of "closeness" and KFG ($F(1,48) = 4.43$, $p = .041$, $\eta^2_p = .09$), as well as a marginally significant interaction between "outcome" and "closeness" ($F(1,48) = 3.48$, $p = .068$, $\eta^2_p = .07$). As Figure 35 shows, there is a positive linear correlation between KFG scores and the difference in SCR between near and full outcomes ($r = .29$, $p = .041$), with participants with higher KFG scores showing increased SCR responses to near vs. full outcomes.

Table 23. Study 3: Results of the ANCOVAs for the dependent measures mean IBI reaction and SCR

Dependent Variable & Effects	*F* (*df*)	*p*	η^2_p
Mean IBI Reaction			
"Outcome"	0.21 (1, 48)	.649	< .01
"Closeness"	0.44 (1, 48)	.512	< .01
KFG (covariate)	3.28 (1, 48)	.077	.06
"Outcome" x "Closeness"	0.76 (1, 48)	.389	.02
"Outcome" x KFG	3.68 (1, 48)	.061	.07
"Closeness" x KFG	1.33 (1, 48)	.254	.03
"Outcome" x "Closeness" x KFG	4.20 (1, 48)	.046*	.08
SCR			
"Outcome"	0.18 (1, 48)	.670	< .01
"Closeness"	0.24 (1, 48)	.629	< .01
KFG (covariate)	1.73 (1, 48)	.195	.04
"Outcome" x "Closeness"	3.48 (1, 48)	.068	.07
"Outcome" x KFG	1.40 (1, 48)	.242	.03
"Closeness" x KFG	4.43 (1, 48)	.041*	.09
"Outcome" x "Closeness" x KFG	1.51 (1, 48)	.226	.03

Note. *$p < .05$. **$p < .01$. ***$p < .001$

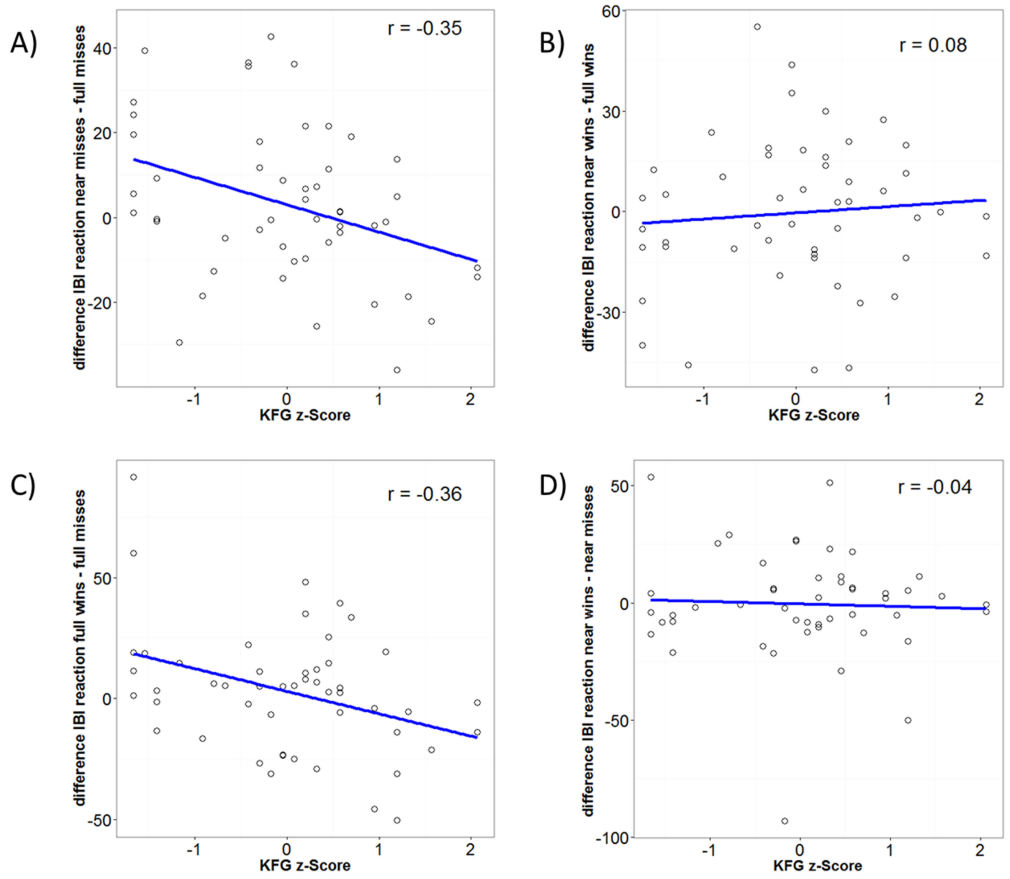

Figure 33. Study 3: Scatterplots of the KFG z-scores and difference measures of IBI reaction for outcome and closeness effects.
A) mean IBI reaction to near misses minus full misses; B) mean IBI reaction to near wins minus full wins; C) mean IBI reaction to full wins minus full misses; D) mean IBI reaction to near wins minus near misses

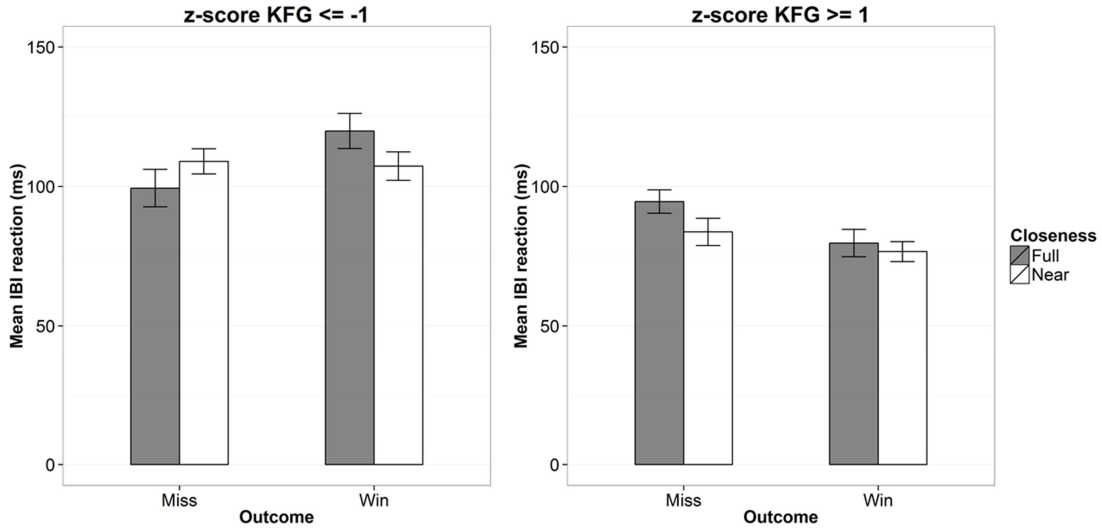

Figure 34. Study 3: Mean IBI reactions for the four outcome types for participants with a z-score KFG <= - 1 (n = 10) and a z-score KFG >= 1 (n = 8).
Error bars denote standard errors of the mean adjusted for within-subject designs according to Cousineau (2005) and Morey (2008).

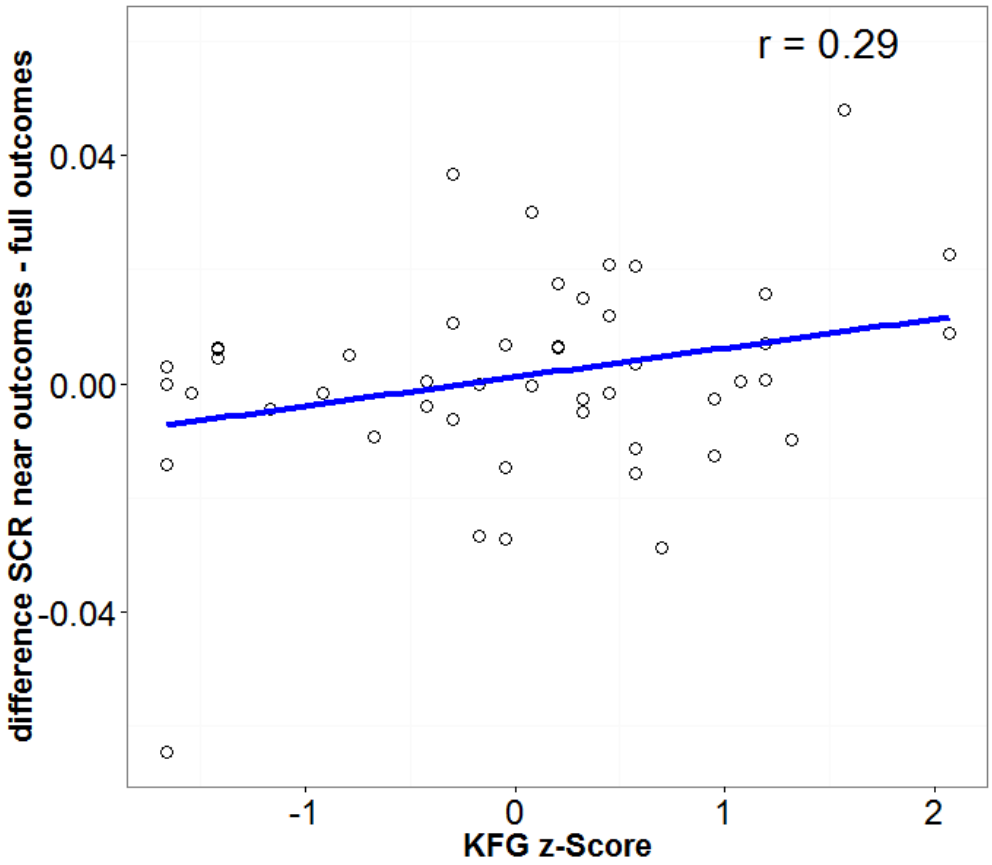

Figure 35. Study 3: Relation between KFG z-scores and the closeness effect in SCR.
The closeness effect was computed as the mean SCR to near outcomes minus the mean SCR to full outcomes; hence, positive values denote a greater mean SCR to near than to full outcomes.

6.3.4 Questionnaires

Correlations between the KFG scores and the questionnaire subscales are depicted in Table 24. The KFG scores correlated significantly with the GRCS sum scores (r = .40, p = .023) and three of its subscales ("Illusion of Control", r = .37, p = .032, "Predictive Control", r = .47, p = .005; "Interpretative Bias", r =.48, p = .005). Furthermore, there were significant correlations with the UPPS "Urgency" subscale (r = .36, p = .032), the DOSPERT Risk-Taking subscale "Gambling" (r = .48, p = .005) and the DOSPERT Risk Perception subscales "Health" (r = .38, p = .023) and "Social" (r = .36, p = .032).

In the follow-up questionnaire, three participants indicated that they had not realized that there were near and full outcomes in the paradigm. Excluding these participants from the analyses did not qualitatively change the results, though, except for two effects. The main effect of the covariate KFG in the IBI reaction analysis was now significant (F(1,45) = 4.27, p = .045, η^2_p = .09). With higher KFG scores, participants showed less overall IBI reaction to the outcomes (r = -.29). Furthermore, the marginally significant interaction of "position" and "IBI number" in the ANOVA of

near outcomes before and after the color boundary was also significant ($F(6,276) = 2.72$, $p = .048$, $\eta^2_p = .06$). However, post-hoc t-tests with corrected *p*-values according to Benjamini and Hochberg (1995) showed no significant differences between near outcomes stopping before and after the color boundary at either IBI number.

Table 24. Study 3: Correlation of KFG with (sub-)scales of GRCS, UPPS, and DOSPERT

Questionnaire	Correlation coefficient (*p*-value)
GRCS	
"Gambling Expectancies"	.23 (.235)
"Illusion of Control"	.37 (.032)*
"Predictive Control"	.47 (.005)**
"Inability to Stop"	.12 (.566)
"Interpretative Bias"	.48 (.005)**
Sum	.40 (.023)*
UPPS	
"Urgency"	.36 (.032)*
"Premediation"	-.08 (.662)
"Perseverance"	-.09 (.625)
"Sensation Seeking"	.14 (.523)
DOSPERT Risk-Taking	
"Ethic"	.00 (.998)
"Investment"	.32 (.056)
"Health"	-.10 (.625)
"Recreational"	-.07 (.662)
"Social"	-.22 (.235)
"Gambling"	.48 (.005)**
DOSPERT Risk Perception	
"Ethic"	.17 (.395)
"Investment"	-.12 (.566)
"Health"	.38 (.023)*
"Recreational"	.27 (.137)
"Social"	.36 (.032)*
"Gambling"	-.07 (.662)

Note. *p*-values were adjusted using an FDR-correction according to Benjamini and Hochberg (1995). *$p < .05$. **$p < .01$. ***$p < .001$

6.4 Discussion

To briefly sum up, the results showed significant differences in event-related IBIs following outcomes in the wheel of fortune. Misses elicited larger IBIs over a longer period of time, while near outcomes elicited longer IBIs shortly after the outcome onset. There were no significant differences in the SCR elicited by the different outcome types. Gambling problems, operationalized as scores in the KFG questionnaire modulated autonomic reactions to outcomes in the wheel of fortune. Specifically, with increasing KFG scores, near compared to full misses elicited smaller IBI reactivity, full wins compared to full misses elicited smaller IBI reactivity and near compared to full outcomes elicited increased SCRs. Finally, the KFG scores correlated positively with cognitive distortions in gambling (especially predictive control and interpretative bias), urgency, risk-taking in the gambling domain and risk perception in the health and social domains.

6.4.1 Heart period

The analysis of the event-related heart period showed a main effect of outcome as well as a significant interaction of IBI number and closeness. Misses compared to wins were followed by longer IBIs. Similar results have been reported in previous research, where negative feedback (Crone et al., 2003; Mueller, Stemmler, & Wacker, 2010; Somsen, van der Molen, Jennings, & van Beek, 2000) or errors (Hajcak, McDonald, & Simons, 2003) also elicited a relative deceleration of the heart rate compared to positive feedback. This deceleration has been interpreted similarly to the ERN and FRN (Somsen et al., 2000), two event-related potentials in the EEG that occur after errors and negative feedback (Gehring, Coles, Meyer, & Donchin, 1990; Gehring & Willoughby, 2002; Miltner et al., 1997). The FRN has been interpreted in terms of indicating a reward prediction error (Holroyd & Coles, 2002), that is, a mismatch between an actual and an expected outcome, and also in terms of indicating more negative valence of the eliciting stimulus (Hajihosseini & Holroyd, 2013; Sambrook & Goslin, 2015). Furthermore, the ERN and FRN are generated in the ACC (Dehaene et al., 1994; Luu et al., 2003; Miltner et al., 1997), a structure that has also been shown to be involved in cardiac regulation (Critchley, Corfield, Chandler, Mathias, & Dolan, 2000; Gianaros, van der Veen, & Jennings, 2004). This is another hint at a common underlying error detection process[14].

In accordance with our hypothesis, near compared to full outcomes led to longer IBIs, but only for the first two IBIs following the outcome. There was no interaction with the factor outcome, suggesting that this pattern is the same for both wins and misses. As such, this result resembles previous findings in the wheel of fortune using EEG and ERPs. Near compared to full outcomes

[14] But see also results showing no correlation between the ERN and cardiac error-related deceleration (Hajcak et al. 2003), suggesting that central nervous and autonomic parameters may reflect different aspects of error processing.

generally elicited a more negative FRN and a smaller P300 (Ulrich & Hewig, 2014; Weiß, 2014). In keeping with the above FRN-analogue interpretation of event-related heart period increase, the larger increase following near outcomes could indicate a more negative evaluation (Hajihosseini & Holroyd, 2013) of those outcomes or a stronger violation of expectations (Alexander & Brown, 2011). Just before receiving a near outcome, the participant might already expect the opposite outcome (e.g. when the wheel is in a field of the chosen color but approaching the boundary to the other color). This expectation is then violated when the near outcome is presented. However, a more processing oriented interpretation of the findings is also conceivable. According to Lacey (1967) an IBI increase following a stimulus suggests increased perceptual processing of that stimulus. Applying this interpretation to the current results suggests that near outcomes could have been processed more deeply. Given the appearance of near outcomes with the pointer stopping close to the boundary of the color fields, it makes intuitive sense that participants need to process the stimulus more deeply to determine on which side of the boundary the pointer actually stopped. It is also possible that the violation of expectations and increased sensory processing go hand in hand. Sensory attenuation, that is, the reduced processing of and reaction to external events caused by one's actions compared external causes, can be explained in terms of forward action models (Hughes, Desantis, & Waszak, 2013). Basically, we expect that our actions have certain consequences, which are then compared to the actual consequences of the action. If the actual consequence matches the expected one, its processing is attenuated, relative to an unexpected consequence. Thus, it is imaginable that, based on their actions in the wheel of fortune, participants expected to win or lose on a given trial. This expectation is then violated by near outcomes, leading to increased sensory processing. Based on the current data we cannot draw final conclusions on which of the explanations is correct. The perceptual processing and FRN-analogue explanations could specifically be tested in a paradigm employing stimuli with less physical difference between near and full outcomes, e.g. a game based purely on numbers. In this case all outcomes should require the same amount of perceptual processing. In such a paradigm a specified range of numbers would lead to a win, whereas numbers outside of that range indicate losses. Numbers that fall just inside or outside of the winning range would be near outcomes. The winning range could also be changed from block to block to avoid confounding effects tied to specific numbers.

6.4.2 Skin conductance

The hypothesis of increased physiological responses for near versus full outcomes was not supported by the results in the current study, as we did not find any significant differences in the elicited SCR among the different outcomes. As such, we did not replicate previous studies which have shown increased SCRs following near misses compared to full misses (Clark et al., 2012) or even

compared to full wins (Dixon et al., 2011). Generally, the SCRs elicited by our paradigm were relatively small. Furthermore, compared to previous studies, we used different outcome probabilities. While Clark et al. (2012) and Dixon et al. (2011) used paradigms in which near misses were less frequent than full misses (and also less frequent than wins in Dixon et al., 2011) our paradigm delivered all four outcome types evenly across the experiment. It has been shown that stimulus probability influences the SCR with less frequently occurring stimuli eliciting larger SCRs (Ben-Shakhar et al., 1982). This might have contributed to increased SCRs following near misses in previous studies.

6.4.3 Correlation with gambling questionnaires

The ANCOVA analyses of IBI reaction and SCR revealed significant influences of the KFG on both physiological parameters. For IBI reaction a three-way interaction was present, indicating that with increased KFG scores both the difference between near and full misses as well as the difference between full wins and full misses changed. Participants with low scores on the KFG showed increased IBI reactions to near compared to full misses and to full wins compared to full misses. This pattern changed as scores on the KFG increased. Participants with high KFG scores showed increased IBI reactions to full misses, both compared to near misses and full wins. The latter difference reflects patterns found in previous research, showing problem gamblers to react less to wins in gambling (Lole, Gonsalvez, Barry, & Blaszczynski, 2014). For SCR there was an interaction between the covariate and the “closeness” factor. Participants with higher scores on the KFG showed increased SCRs to near compared to full outcomes.

In summary, our hypothesis of increased effects of the “closeness” factor with increased gambling problems was only partly supported. SCRs showed the expected pattern of results. For IBI reactivity, the pattern was reversed, though and specific for near compared to full misses, and did not apply to near outcomes in general.

This hints at a possible dissociation of heart period and SCR with respect to gambling problems. Short term heart period reactions are mainly influenced by parasympathetic activity (Berntson et al., 1997), while SCRs are influenced by sympathetic activity (Dawson et al., 2007). Combined with the current findings, this might suggest decreased parasympathetic reactivity and increased sympathetic reactivity with increasing gambling problems. Previous studies on activity of the sympathetic nervous system in problem and pathological gamblers have yielded mixed results, with some studies finding no sympathetic alterations (Labudda, Wolf, Markowitsch, & Brand, 2007) and others finding increased sympathetic activation in problem gamblers (Krueger, Schedlowski, & Meyer, 2005; Meyer et al., 2004). Future studies could specifically address the activity of the parasympathetic and sympathetic system in problem gamblers.

In summary, It might be suggested that problem gamblers show increased arousal (SCR) to all near as compared to full outcomes and a more negatively valenced vagal physiological response (IBI reaction) to full misses only. Based on the assumption that participants have a composite subjective experience of their physiological responses, suggests that full losses elicit a specifically negative (FRN interpretation of IBI) and low arousal experience (no SCR) in problem gamblers, which sets those events apart. In contrast to full misses, problem gamblers show indistinguishable responses to all kinds of near outcomes and finally a possibly reduced arousal (lower SCR) to full wins. This would be in line with the previously shown role of near misses which is in stark contrast to the processing of and response to full misses.

6.4.4 Correlations of KFG and SOGS with other questionnaires

The KFG score correlated significantly with the GRCS subscales “Illusion of Control”, “Predictive Control”, and “Interpretative Bias”. The subscales “Gambling Expectancies” and “Inability to Stop Gambling” did not correlate significantly with the KFG scores. Hence, it can be concluded that the participants did not have unrealistic expectancies about the effects of gambling (e.g. “Gambling will solve all my problems”, subscale “Gambling Expectancies”), neither did they have the feeling that they cannot stop gambling (subscale “Inability to Stop Gambling”). It may be speculated that problem gamblers’ cognitive mindset including “Illusion of Control”, “Predictive Control”, and “Interpretative Bias” may contribute to the differences in physiological responses to near and full miss outcomes described above.

Furthermore, the KFG correlated with the UPPS “Urgency” subscale. Participants with higher scores on KFG also showed increased scores on the “Urgency” subscale. “Urgency” as measured in the UPPS indicates the occurrence of strong impulses which are difficult to control (Whiteside & Lynam, 2001). However, the KFG scores did not correlate with the other subscales of impulsivity measured in the UPPS. Taken together, this result fits with previous research showing increased impulsivity in pathological and problem gamblers (Verdejo-Garcia, Lawrence, & Clark, 2008) and a study showing the subscale “Urgency” to be the only UPPS subscale significantly predicting SOGS scores (Whiteside et al., 2005). It may be hypothesized that the strong impulses indicative of “Urgency” are related to physiological responses tied to such impulses and thus to the specific physiological reactions to near and full outcomes presented above.

Finally, the KFG scores were correlated with the DOSPERT risk-taking and risk perception scales to assess whether signs of problem gambling are related with increased risk-taking across domains or only specifically in gambling. The KFG scores only correlated significantly with the risk-taking “Gambling” subscale, but not with other risk-taking subscales. At the same time, there was no significant correlation with the “Gambling” subscale in the risk perception part of the questionnaire.

Hence, the increased risk-taking in the gambling domain shown by participants with increased KFG scores cannot be explained by a decreased risk perception in this domain. Instead, unexpected additional significant positive correlations between the KFG scores and the risk perception "Social" and "Health" subscales were found. This finding should be addressed further in future studies including larger and more diverse samples. However, the results could be related to generally increased anxiety levels in people with current gambling problems (Barrault & Varescon, 2013; Grüsser, Plöntzke, & Albrecht, 2005; Toneatto et al., 2008). This increased anxiety could manifest itself in increased perception of the risk associated with certain activities.

6.4.5 Differences between near outcomes before and after the color boundary

Exploratory analyses of possible differences between near outcomes stopping before and after the color boundary did not reveal significant differences in heart period and SCR. Rerunning the original analyses separately with near outcomes stopping before and after the color boundary only slightly changed the results in one case. Including only near outcomes stopping before the color boundary yielded a now significant interaction of the factors "outcome" and "closeness" for SCR, with near misses descriptively eliciting larger SCRs than full misses and near wins. This slight change in results compared to the original analysis including all near outcomes possibly stems from descriptively slightly larger SCRs elicited by near misses stopping before the color boundary (M = 0.047) compared to near misses stopping after the color boundary (M = 0.045). Previous studies have shown greater dissociations between near misses stopping before and after the payline in a slot machine task (Clark et al., 2013; Clark et al., 2009)[15]. Motivational effects and activation in the ACC were increased for near misses stopping before the payline. Thus, the current results descriptively fit with this pattern.

6.4.6 Limitations

When interpreting the current study's results, some limitations need to be kept in mind. Firstly, breathing of participants was not measured. Breathing influences the heart period (Berntson, Cacioppo, & Quigley, 1993), with inhalation leading to a decrease in heart period and exhalation leading to an increase in heart period. This breath-related variance in heart period could not be accounted for in the currents study, thus it cannot be ruled out that part of the variance in the heart period measure is related to participants' breathing. Secondly, the running total was not controlled across participants but was random from participant to participant, since the sequence of outcomes was determined randomly for every participant. The running total might serve as a context in which

[15] The article by Clark et al. (2009) actually states, that the effects are larger for near misses stopping after the payline. However, Clark et al. (2013) note that this result was due to a coding error in the analysis. The results actually showed, that near misses stopping before the payline elicited stronger effects.

the outcome of a given trial is evaluated. Since this context was not controlled across participants, this might have created additional error variance in the dependent variables.

6.5 Summary

The current study showed that near and full wins and misses differ in their elicited heart period reactions. Misses are followed by generally longer IBIs, whereas after near compared to full outcomes only the first two IBIs are longer. Furthermore, IBI reactivity and SCR following outcomes were moderated by scores on the KFG. Participants with higher scores on the KFG showed an increased IBI reactivity to full misses compared to the other outcomes, as well as an increased SCR to near compared to full outcomes in general, hinting at increased arousal caused by near outcomes and win-like processing of near wins and near misses in problem gambling. The results also showed that gambling problems are associated with increased risk-taking in the gambling-domain only

7 Study 4: Processing of outcome sequences in gambling (EEG)

7.1 Introduction

The fourth study used EEG, more specifically ERPs and time-frequency analysis, to assess the processing of outcome sequences. A group of problem gamblers was compared to a group of controls to analyze the influence of gambling problems on the processing of outcome sequences. Furthermore, participants' choice behavior in the gamble was analyzed.

7.1.1 Summary of previous results

Previous studies have shown that the FRN and the P300 in feedback processing are not only sensitive to the current outcome, but also to previous outcomes (e.g. Goyer et al., 2008; Mushtaq et al., 2016; Osinsky et al., 2012). FRN and P300 are increased, when the previous outcomes are different to the current outcome (Osinsky et al., 2012). This effect could be explained by changes in expectancy caused by the previous outcome sequences. A run of outcomes might cause participants to expect its continuation. This expectation is violated when the another outcome occurs in the current trial, thus leading to increased FRN and P300 amplitudes, both of which have been shown to be increased for unexpected stimuli (e.g. Hajcak et al., 2007; Squires et al., 1976).

As already described in section 3.1.1.2.1, a measure that is conceptually related to the FRN and also influenced by expectancy, is power in the theta frequency band. Theta power following feedback has been shown to be increased for unexpected feedback (e.g. Cavanagh, Zambrano-Vazquez et al., 2012; Cohen et al., 2007; Hajihosseini & Holroyd, 2013; Tzur & Berger, 2009).

Concerning choice behavior in gambling, previous research has shown evidence for the gambler's fallacy, that is, the belief that following a run of outcomes in an independent binary sequence, the other outcome is more likely to occur, and the hot hand fallacy, that is, the belief that a streak of outcomes in an independent sequence is more likely to continue (Ayton & Fisher, 2004).

7.1.2 Current study

The current study sought to investigate choice behavior and processing of outcome sequences in a simple coin toss task to draw conclusions on the gambler's fallacy and the hot hand fallacy. In this task, participants repeatedly had to predict the outcome (heads, tails) of a coin toss performed by the computer. The sample consisted of a group of problem gamblers and a group of matched controls to assess whether problem gamblers show an increased proneness towards the gambler's and hot hand fallacies and whether they differ from controls in the processing of outcome

sequences. Three-outcome sequences (outcome n-2, outcome n-1, current outcome) were analyzed to investigate the effects of previous outcomes on the processing of the current outcome.

7.1.3 Hypotheses

Osinsky et al. (2012) showed that the FRN and the P300 for a given outcome are increased if this outcome differs from the previous two outcomes. It was expected that this pattern would be replicated in the control group and would also emerge for theta power. Furthermore, in accordance with the hot hand fallacy, participants in the control group were expected to show increased expectations of winning again in the next trial following a streak of wins compared to other outcome sequences. It was also expected that participants show a gambler's fallacy, that is following a run of heads or tails participants should more likely choose the other side of the coin in the next trial. These effects (increased FRN, P300 and theta power following the break of a streak of an outcome, increased expectations of winning following a streak of wins, increased probability of choosing the other symbol following a run of one symbol) were expected to be stronger in the problem gambling group, based on self-report data indicating increased gambling-related cognitions in problem and pathological gamblers (e.g. Cunningham et al., 2014; Joukhador et al., 2004; Myrseth et al., 2010). Furthermore, based on previous results (Lole et al., 2015; Oberg et al., 2011) problem gamblers were expected to show overall reduced P300 levels.

7.2 Methods

7.2.1 Participants

The sample consisted of the same participants who also took part in the wheel of fortune EEG study. Table 5 shows the sample characteristics. To briefly sum up, two groups participated in the experiment: one problem gambling group (n = 20, mean age = 25.7, 18 ♂) and one group of matched controls (n = 20, mean age = 25.10, 18 ♂). Participants were recruited based on online screening questionnaires for gambling behavior (KFG, Petry, 1996; SOGS, Lesieur & Blume, 1987) and an ensuing interview, assessing the *DSM-IV-TR* A-criteria for pathological gambling (SKID-PG, Grant et al., 2004). Participants were recruited for the problem gambling group if they scored at or above the cutoff for pathological gambling in both gambling screening questionnaires (KFG cutoff: 16, SOGS cutoff: 5) and fulfilled at least 2 *DSM-IV-TR* A-criteria for pathological gambling. Participants in the control group had to score below the respective cutoffs in the gambling screening questionnaires and fulfill less than 2 *DSM-IV-TR* A-criteria for pathological gambling.

7.2.2 Paradigm: Coin toss

The participants task in the coin toss paradigm was to predict the outcome of a coin toss (heads or tails) performed by the computer. The paradigm was programmed and presented via Presentation software (Version 17.1; Neurobehavioral Systems).

Figure 36 gives an overview of a sample trial in the coin toss paradigm. Every trial started with the presentation of the front side and the backside of a 1 Euro coin next to each other. The side of presentation was balanced across the experiment, with both coin sides appearing on the left and right side of the screen equally often, in a random order across trials. Via button press, participants chose one of the coin sides. Following a fixation cross interval, the outcome of the coin toss was presented, indicating that participants had won 10 Cents if they were correct or lost 10 Cents if their prediction was incorrect. Every fourth occurrence of a given outcome sequence (see below) was followed by a rating on the probability of winning in the next trial ("How likely do you think you will win in the next trial?"), which was answered on a scale from 0% to 100% in steps of 10%.

The coin toss paradigm consisted of 249 trials altogether. Similar to the wheel of fortune paradigm, the last 7 trials were not analyzed and contained more wins than losses to ensure that participants ended the game with more money than their starting balance of 400 Cents. The remaining 242 trials consisted of 121 wins and 121 losses. The same fixed sequence of outcomes, which controlled for the occurrence of three-outcome sequences, was used for every participant. The main focus of this experiment was analyzing the processing of the current outcome in relation to the previous two outcomes. This yields eight different three-outcome sequences in the form outcome in trial n-2, outcome in trial n-1, current outcome: "winwinwin", "winwinloss", "winlosswin", "winlossloss", "losswinwin", "losswinloss", "losslosswin", "losslossloss". Throughout the 242 trials, each of these outcome sequences occurred 30 times[16]. After 121 trials, a 15 second break was presented. Participants started with 400 Cents in a virtual account and were instructed that the final balance of the account would be added to their fixed payment of 10.00 €. Due to the fixed outcome sequence, every participant ended the coin toss paradigm with 430 Cents in their account. The final account balance was shown at the end of the paradigm.

[16] The outcome sequences were overlapping. For example, the outcomes „win, win, loss, win, loss" contain the outcome sequences „winwinloss", „winlosswin", and „losswinloss".

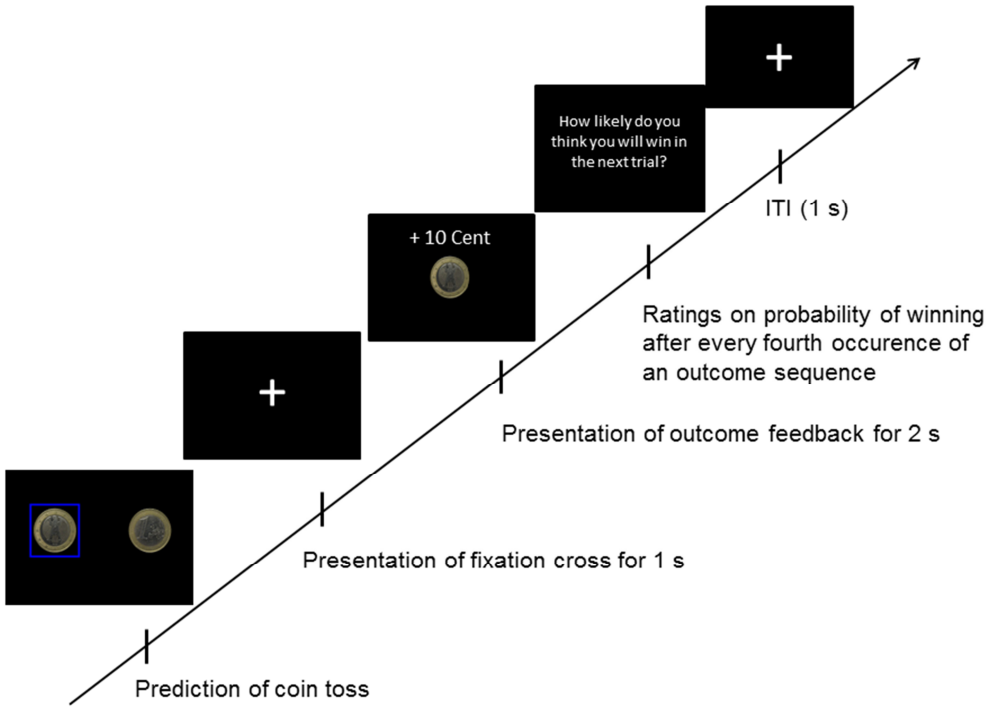

Figure 36. Study 4: Sample trial in the coin toss paradigm.

7.2.3 Procedure

Participants gave written informed consent after having read information on the experiment and the procedure. Next, the EEG was prepared and participants were seated in an electrically shielded chamber, where they played the wheel of fortune and the coin toss paradigm (see below). 30 participants (15 PG and 15 nonPG) started with the wheel of fortune, while 10 (5 PG and 5 nonPG) started with the coin toss. In the break between the two games, EEG electrode impedances were checked and corrected if necessary.

7.2.4 EEG recordings and quantification

The EEG was recorded using 32 Ag/AgCl electrodes placed according to the 10-20 system (Jasper, 1958), including an electrode below the left eye to correct for blinks and eye movements (electrode positions: Fp1, Fp2, F3, F4, C3, C4, P3, P4, O1, O2, F7, F8, T7, T8, P7, P8, Fz, FCz, Pz, FC1, FC2, CP1, CP2, FC5, FC6, F9, F10, TP9, TP10, PO9, PO10, IO). The ground electrode was placed at AFz and Cz was used as online reference. The EEG signal was amplified using a BrainAmp DC amplifier (Brain Products GmbH) and the BrainVision Recorder 1.20 (Brain Products GmbH) was used to record the signal with a sampling rate of 250 Hz and an online high-pass filter of 0.016 Hz (10 s) as well as a low-pass filter of 250 Hz. Electrode impedances were kept below 5 kΩ. For offline data processing the BrainVision Analyzer 2.0 software (Brain Products GmbH) was used. Table 25 gives an overview of the

processing. First, data were rereferenced to linked mastoids (average of TP9 and TP10) and the online reference Cz was reinstated as channel. Next, data were filtered using a 0.1 Hz low-cutoff and a 20 Hz high-cutoff filter (each with 48 dB/octave slope). An automatic raw data inspection was applied using built-in algorithms of the Brain Vision Analyzer. Intervals of 400 ms were marked as artifact when there was either an activity less than 0.5 µV in time windows of 100 ms, voltage steps exceeding 50 µV/ms within the interval or a maximal amplitude difference above 400 µV in an interval of 200 ms. Channel IO (below the left eye) was excluded from the artifact detection to prevent blinks from being discarded as artifacts exceeding the amplitude difference criterion. For two subjects most of the relevant data was excluded in the raw data inspection. However, visual inspection showed that this was mainly due to blinks being detected as artifacts due to the amplitude difference criterion. For these participants raw data inspection was rerun excluding the amplitude difference criterion. Following raw data inspection blinks and eye movements were corrected using an ICA method implemented in the Analyzer. Next, data were segmented with reference to the outcome onset of the last outcome in any three-outcome sequence. For ERP analyses, segments started 200 ms before outcome onset and lasted until 1000 ms following outcome onset. For time-frequency analyses, the segments lasted from -1000 ms until +1000 ms with respect to outcome onset. An artifact rejection algorithm was applied to all channels, excluding segments with maximal voltage differences above 200 µV in 200 ms windows to exclude potentially remaining blink and eye-movement artifacts that were not properly corrected by the ICA. For ERP analyses, average waveforms were calculated separately for every participants and each of the eight three-outcome sequences[17]. The averages were then baseline corrected, with the first 100 ms before outcome onset serving as baseline. The peaks of the P2, N2 and P300 were then detected semiautomatically and corrected manually if necessary. P2 and N2 were detected at electrodes Fz, FCz and Cz. The detection time windows were 150 ms to 250 ms for the P2 peak and 200 ms to 350 ms for the N2 peak. The P300 was detected in the time window from 250 ms to 600 ms at electrode Pz. For the statistical analysis the average voltage value in an interval of 24 ms around the detected peaks (+/- 12 ms corresponding to +/- 3 datapoints) was exported. The FRN was then quantified as the difference P2-N2, thus larger positive values denote a larger FRN. For time-frequency analyses complex morelet wavelets were used. The wavelets spanned the frequency range from 1 to 20 Hz in 19 logarithmic steps. A morelet parameter of 5 was chosen. The resulting power values were baseline corrected, using the time between -400 ms to -200 ms before outcome onset as baseline. Following wavelet

[17] Averages for the ERPs were based on the following mean numbers of segments (minimum number given in parentheses): winwinwin: 29.3 (21), winwinloss: 28.83 (14), winlosswin: 29.45 (24), winlossloss: 28.93 (18), losswinwin: 29.25 (22), losswinloss: 28.98 (14), losslosswin: 29.2 (22), losslossloss: 29.35 (21)

convolution, averages for every subject and every three-outcome sequence were computed[18]. Finally, the mean power from three layers (central frequencies of the exported layers: 4.47 Hz, 5.28 Hz, 6.24 Hz), corresponding to the theta range, was extracted at electrodes Fz, FCz and Cz in the time range 250 ms to 500 ms post-outcome.

Table 25. Study 4: Preprocessing and export for ERP and time-frequency analyses in the coin toss paradigm

ERP	Time-Frequency
Rereferencing linked mastoids	
Filter 0.1 Hz low-cutoff and 20 Hz high-cutoff, 48 dB/octave slope	
Raw data inspection (channel IO excluded) Gradient criterion (≥50 µV/ms) Low Activity criterion (≤ 0.5 µV in 100 ms) Difference criterion (≥ 400 µV in 200 ms)	
Ocular Correction via ICA	
Segmentation -200 ms to +1000 ms	*Segmentation* -1000 ms to +1000 ms
Artifact Rejection (all channels) Difference criterion (≥ 200 µV in 200 ms)	
Average	*Complex Morelet Wavelets* 1 Hz - 20 Hz 19 layers, logarithmic Morelet parameter 5 -400 ms to -200 ms baseline
Baseline Correction -100 ms to 0 ms	*Average*
Peak Detection (semiautomatic) P2: 150-250 ms N2: 200-350 ms P300: 250-600 ms	*Mean Power Export* Layers 10 - 12 (central frequencies: 4.47 Hz, 5.28 Hz, 6.24 Hz) 250 ms to 500 ms Fz, FCz, Cz
Peak Export P2 + N2: Fz, FCz, Cz P300: Pz 24 ms (+/-12 ms) around peak	

[18] Averages for the wavelets were based on the following mean numbers of segments (minimum number given in parentheses): winwinwin: 28.9 (20), winwinloss: 28.6 (14), winlosswin: 29.05 (22), winlossloss: 28.45 (14), losswinwin: 28.93 (22), losswinloss: 28.58 (13), losslosswin: 28.7 (21), losslossloss: 28.88 (20)

7.2.5 Analysis of choice behavior

To see whether participants showed a gambler's fallacy like behavior, the probability of choosing a coin side again following a run of this coin side was analyzed. Since the outcome sequence (win vs. loss) was predetermined, the sequence of coin sides (heads vs. tails) could not be controlled and thus varied across participants. For every participant runs of the same coin side of lengths 1 to 5[19] were extracted and the probability of choosing the same coin side again in the next trial was calculated (Ayton & Fischer, 2004).

7.2.6 Statistical analysis

The FRN amplitudes and theta power were analyzed with a 2*4*3*2 mixed ANOVA with the within-subject factors "current outcome" (win vs. loss), "previous outcomes" (winwin vs. winloss vs. losswin vs. lossloss) and "electrode" (Fz, FCz, Cz), and the between-subject factor "group" (PG vs. nonPG). The P300 amplitudes were analyzed with a 2*4*2 mixed ANOVA with the within-subject factors "current outcome" (win vs. loss), "previous outcomes" (winwin vs. winloss vs. losswin vs. lossloss) and the between-subject factor "group" (PG vs. nonPG). The mean probability rating data for each three-outcome sequence were analyzed using 2*4*2 mixed ANOVAs with the within-subject factors "current outcome" (win vs. loss) and "previous outcomes" (winwin vs. winloss vs. losswin vs. lossloss) and the between-subject factor "group" (PG vs. nonPG). Choice behavior was analyzed with a 5*2 mixed ANOVA with the within-subject factor "run length" (1 vs. 2 vs. 3 vs. 4 vs. 5) and the between-subject factor "group" (PG vs. nonPG). In cases of violation of the assumption of sphericity, the Greenhouse-Geisser correction was applied and corrected degrees of freedom are reported. Partial eta-squared values are reported as measures of effect size. The analyses were run in SPSS 21 (IBM).

7.3 Results

7.3.1 FRN

The analysis of the FRN data showed significant main effects of the factors "electrode" ($F(1.150,43.685) = 15.80$, $p < .001$, $\eta^2_p = .29$) and "group" ($F(1,38) = 17.59$, $p < .001$, $\eta^2_p = .32$). These effects were qualified by a marginally significant interaction of the factors "electrode" and "group" ($F(1.150,43.685) = 2.98$, $p = .087$, $\eta^2_p = .07$). Finally, there was a marginally significant interaction of the factors "current outcome", "electrode", and "group" ($F(1.338,50.826) = 3.60$, $p = .051$, $\eta^2_p = .09$). Post hoc paired t-tests indicated that the PG group showed a smaller FRN than the nonPG group at all included electrodes (Fz: $t(38) = -3.71$, $p = .001$; FCz: $t(38) = -4.17$, $p < .001$; Cz: $t(38) = -4.40$, $p =$

[19] The runs were also overlapping. A run of length 5 (e.g. heads, heads, heads, heads, heads) also contains one run each of lengths 1 to 4.

.001). Within the PG group, the FRN decreased from anterior to posterior electrodes (wins: Fz-FCz: $t(19) = 1.81$, $p = .086$; Fz-Cz: $t(19) = 4.12$, $p = .001$; FCz-Cz: $t(19) = 4.44$, $p < .001$; losses: Fz-FCz: $t(19) = 3.52$, $p = .002$; Fz-Cz: $t(19) = 5.38$, $p < .001$; FCz-Cz: $t(19) = 5.64$, $p < .001$), whereas in the nonPG group, the only significant differences emerged between electrode FCz and Cz (wins: $t(19) = 2.95$, $p = .008$; losses: $t(19) = 2.65$ $p = .016$), with the FRN being larger at FCz. Concerning the effect of the factor "current outcome", the PG group only showed a marginally significantly larger FRN following losses compared to wins at electrode Fz ($t(19) = 1.79$, $p = .090$), whereas the nonPG group showed no significant effects of outcome at either electrode. Table 26 shows the full results of the ANOVA. Figures 37 and 38 show the ERPs and the mean FRN values at electrodes Fz, FCz and Cz.

Table 26. Study 4: Results of the ANOVA for the FRN

Effect	*F* (*df*)	*p*	η^2_p
"Current Outcome"	0.78 (1,38)	.382	.02
"Previous Outcomes"	1.39 (2.345,89.124)	.254	.04
"Electrode"	15.80 (1.150,43.685)	< .001***	.29
"Group"	17.59 (1,38)	< .001***	.32
"Current Outcome" x "Previous Outcomes"	0.17 (2.608,99.109)	.895	< .01
"Current Outcome" x "Electrode"	1.67 (1.338,50.826)	.203	.04
"Current Outcome" x "Group"	1.80 (1,38)	.188	.05
"Previous Outcomes" x "Electrode"	2.02 (2.909,110.531)	.117	.05
"Previous Outcomes" x "Group"	1.87 (2.345,89.124)	.154	.05
"Electrode" x "Group"	2.98 (1.150,43.685)	.087	.07
"Current Outcome" x "Previous Outcomes" x "Electrode"	0.44 (2.806,106.614)	.712	.01
"Current Outcome" x "Previous Outcomes" x "Group"	1.39 (2.608,99.109)	.254	.04
"Current Outcome" x "Electrode" x "Group"	3.60 (1.338,50.826)	.051	.09
"Previous Outcomes" x "Electrode" x "Group"	0.15 (2.909,110.531)	.929	< .01
"Current Outcome" x "Previous Outcomes" x "Group" x "Electrode"	1.19 (2.806,106.614)	.316	.03

Note. *$p < .05$. **$p < .01$. ***$p < .001$

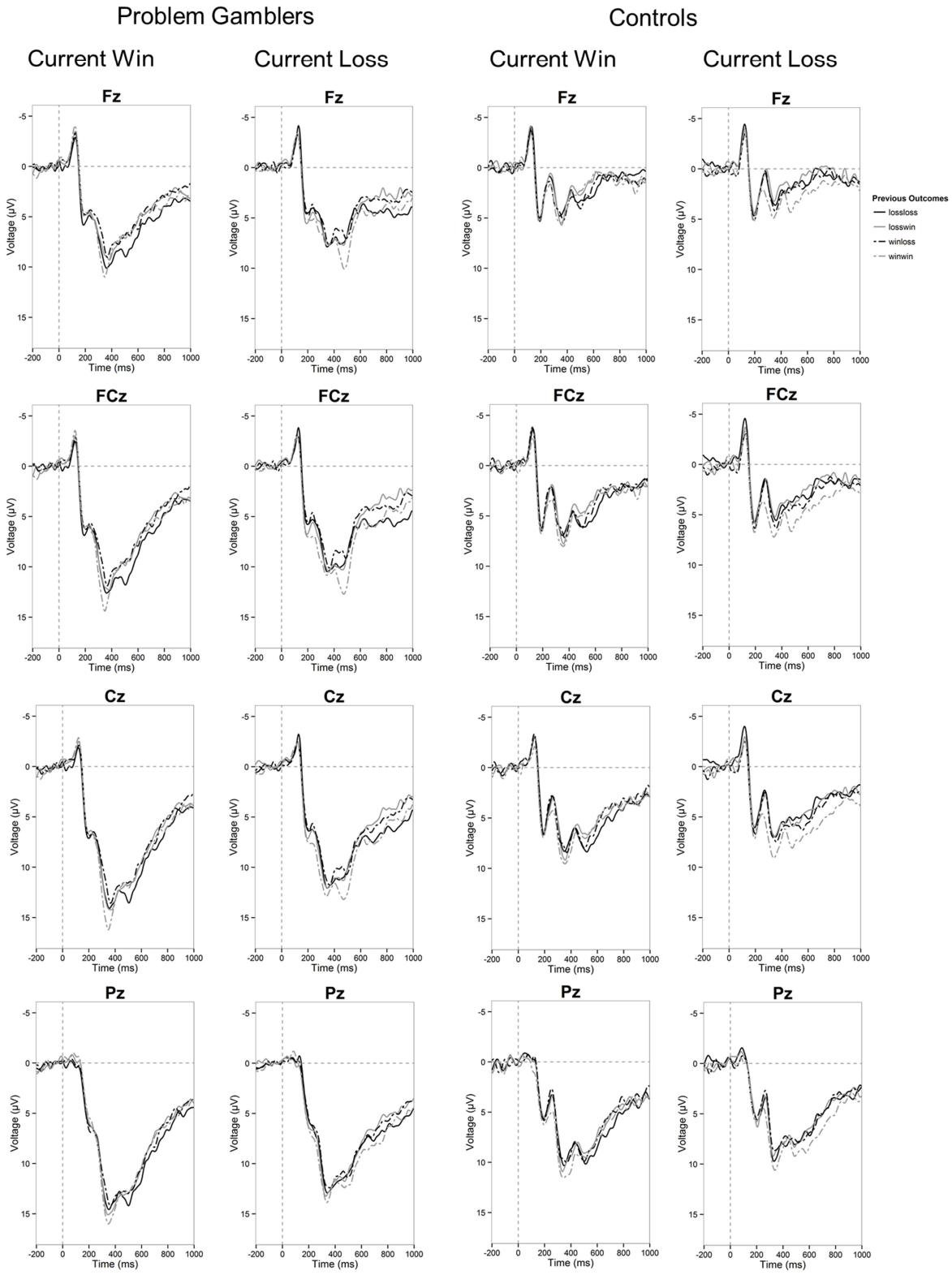

Figure 37. Study 4: ERP waveforms following current wins and current losses as a function of the previous outcomes at electrodes Fz, FCz, Cz and Pz, for problem gamblers and controls.

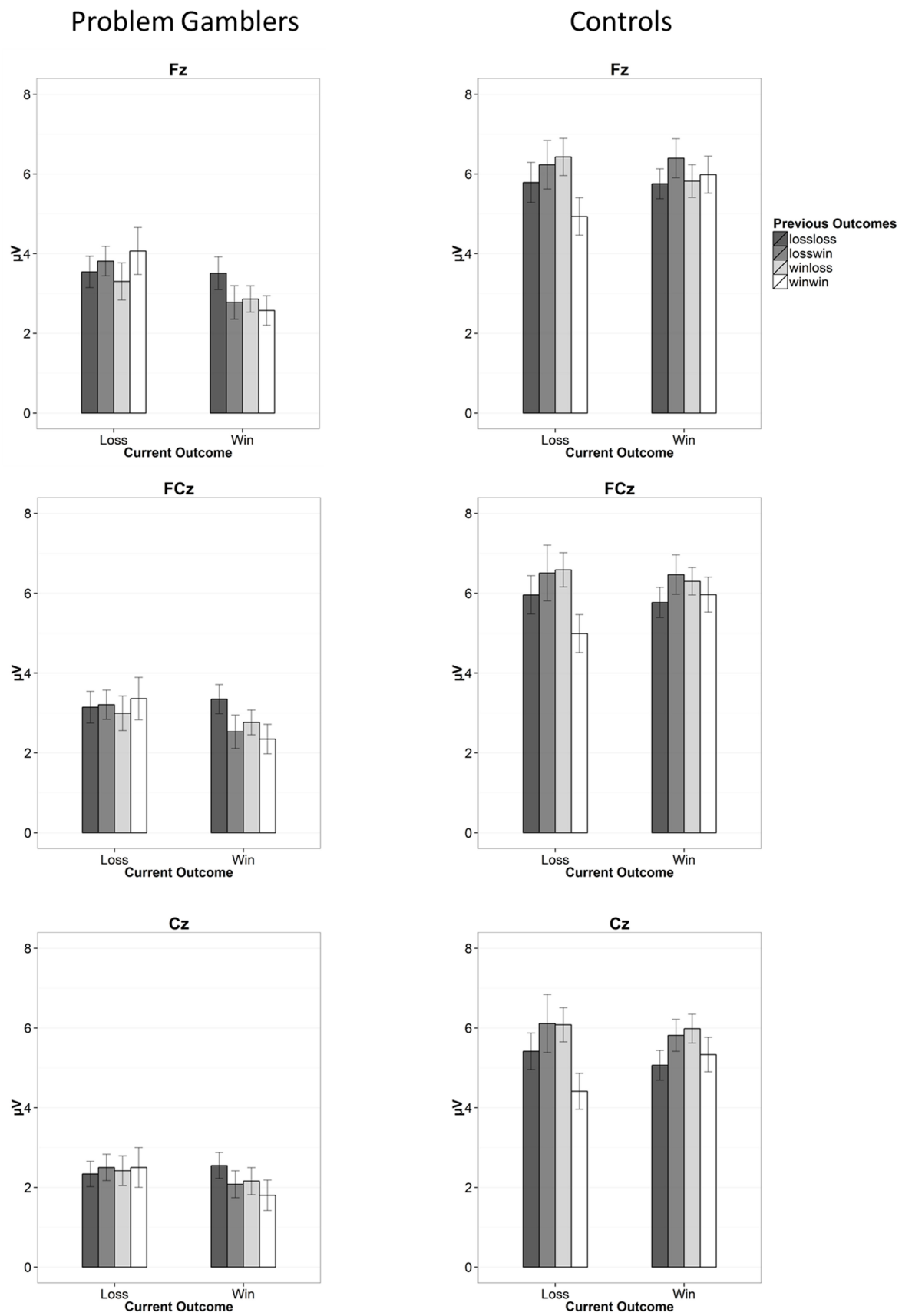

Figure 38. Study 4: Mean FRN values at electrodes Fz, FCz, and Cz for problem gamblers and controls. Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

7.3.2 Theta

The analysis of the theta power showed a significant main effect of the factor "current outcome" ($F(1,38) = 7.54$, $p = .009$, $\eta^2_p = .17$). This main effect was qualified by two interactions, one with the factor "electrode" ($F(1.581,60.063) = 6.79$, $p = .004$, $\eta^2_p = .15$) and the other with the factor "previous outcomes" ($F(2.263,85.997) = 5.23$, $p = .005$, $\eta^2_p = .12$). Furthermore, there was also a significant main effect of the factor "electrode" ($F(1.390,52.828) = 21.155$, $p < .001$, $\eta^2_p = .36$). Post-hoc paired t-tests showed that misses compared to wins elicited (marginally) larger theta power at all tested electrodes (Fz: $t(39) = 2.89$, $p = .006$; FCz: $t(39) = 3.07$, $p = .004$; Cz: $t(39) = 1.80$, $p = .079$). Theta Power was largest at electrode FCz (wins: FCz-Fz: $t(39) = 2.86$, $p = .007$; FCz-Cz: $t(39) = 3.72$, $p = .001$; losses: FCz-Fz: $t(39) = 3.42$, $p = .002$; FCz-Cz: $t(39) = 5.74$ $p < .001$). Theta power at Fz was also higher than at Cz (wins: $t(39) = 2.05$, $p = .048$; losses: $t(39) = 4.39$, $p < .001$). Concerning the interaction of "current outcome" and "previous outcomes", post-hoc paired t-tests indicated, that losses compared to wins only showed increased theta power for the previous sequences "winwin" ($t(39) = 3.60$, $p = .001$) and "losswin" ($t(39) = 2.79$, $p = .008$), but not for the previous sequences "winloss" ($t(39) = 1.08$, $p = .288$) and "lossloss" ($t(39) = -0.54$, $p = .593$). For current wins, theta power was (marginally) significantly higher following the previous sequence "lossloss" compared to "winwin" ($t(39) = 2.14$, $p = .039$) and "losswin" ($t(39) = 1.95$, $p = .058$). For current losses, the previous sequence "winwin" resulted in (marginally) significantly larger theta power compared to the other previous-outcome sequences ("winloss": $t(39) = 3.04$, $p = .004$; "losswin": $t(39) = 1.80$, $p = .08$; "lossloss": $t(39) = 2.95$, $p = .005$). Table 27 shows the full ANOVA results. Figures 39 and 40 show the time frequency plots at electrodes Fz, FCz and Cz, and the mean theta values at Fz, FCz, and Cz, separately for both groups. Figure 41 shows the topography of theta power difference between misses and wins, reflecting the effect of the factor "current outcome". Figure 42 shows the theta power difference between sequences breaking a streak of outcomes and the average of the other sequences, reflecting the interaction of the factors "current outcome" and "previous outcomes".

Table 27. Study 4: Results of the ANOVA for theta power

Effect	*F* (*df*)	*p*	η^2_p
"Current Outcome"	7.54 (1,38)	.009**	.17
"Previous Outcomes"	0.92 (3,114)	.434	.02
"Electrode"	21.16 (1.390,52.828)	< .001***	.36
"Group"	0.59 (1,38)	.447	.02
"Current Outcome" x "Previous Outcomes"	5.23 (2.263,85.997)	.005**	.12
"Current Outcome" x "Electrode"	6.79 (1.581,60.063)	.004**	.15
"Current Outcome" x "Group"	0.65 (1,38)	.425	.02
"Previous Outcomes" x "Electrode"	1.92 (3.646,138.538)	.116	.05
"Previous Outcomes" x "Group"	0.94 (3,114)	.425	.02
"Electrode" x "Group"	0.17 (1.390,52.828)	.768	< .01
"Current Outcome" x "Previous Outcomes" x "Electrode"	2.07 (3.118,118.493)	.106	.05
"Current Outcome" x "Previous Outcomes" x "Group"	0.48 (2.263,85.997)	.641	.01
"Current Outcome" x "Electrode" x "Group"	0.99 (1.581,60.063)	.362	.03
"Previous Outcomes" x "Electrode" x "Group"	0.41 (3.646,138.538)	.781	.01
"Current Outcome" x "Previous Outcomes" x "Group" x "Electrode"	0.21 (3.118,118.493)	.895	.01

Note. *$p < .05$. **$p < .01$. ***$p < .001$

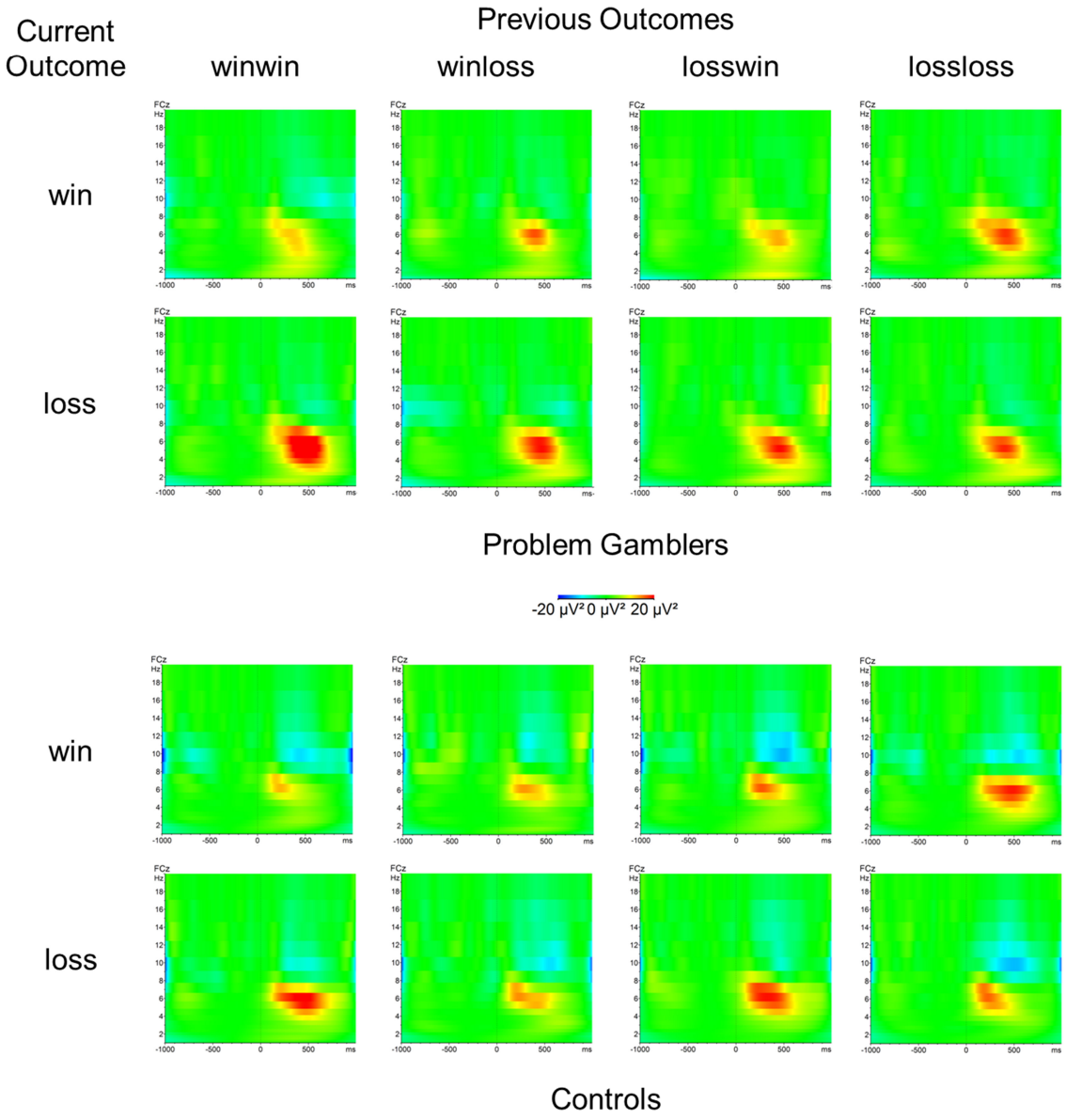

Figure 39. Study 4: Time-frequency plots for the eight outcome sequences at electrode FCz separately for problem gamblers and controls.

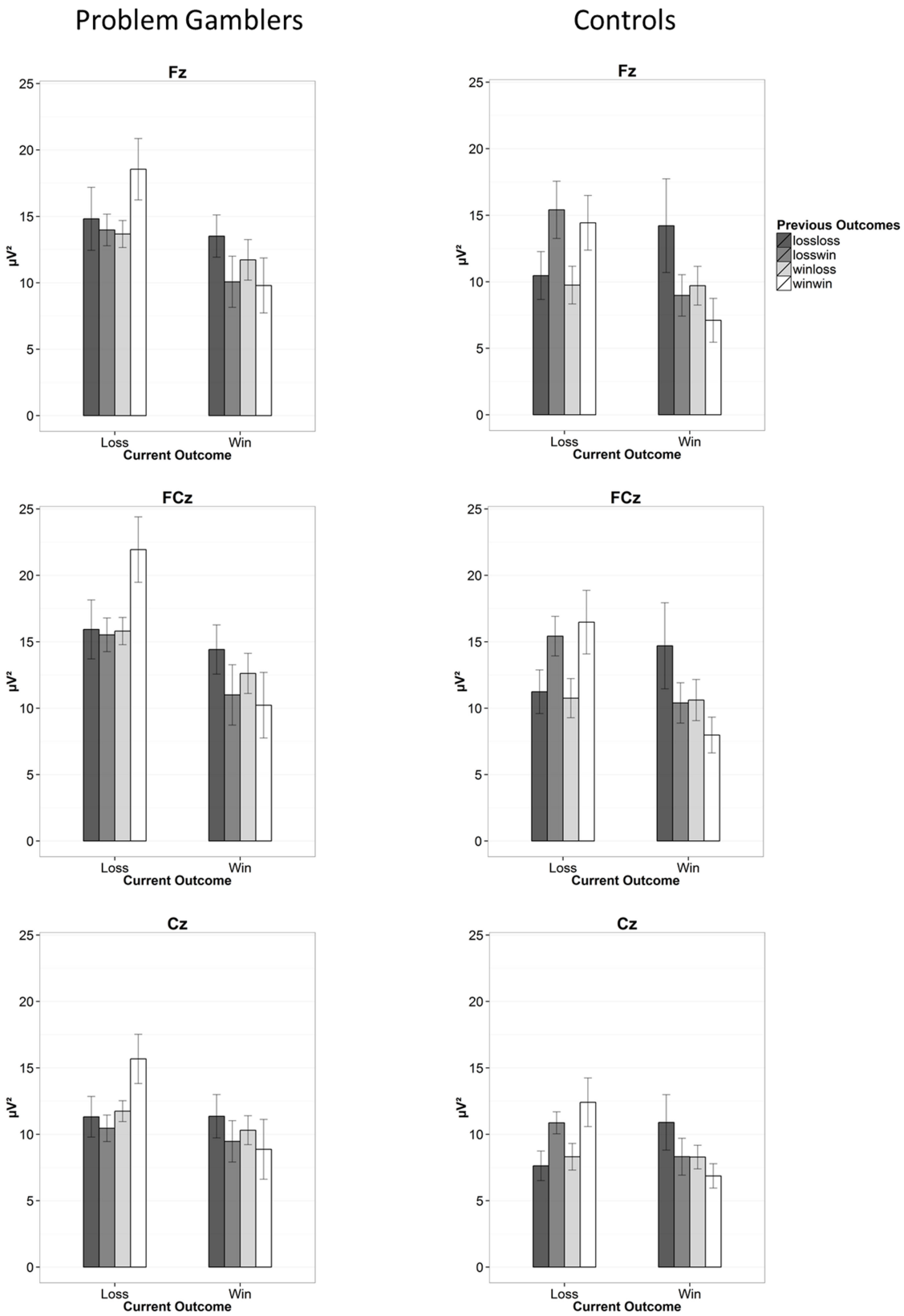

Figure 40. Study 4: Mean theta values at electrodes Fz, FCz, and Cz for problem gamblers and controls.
Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

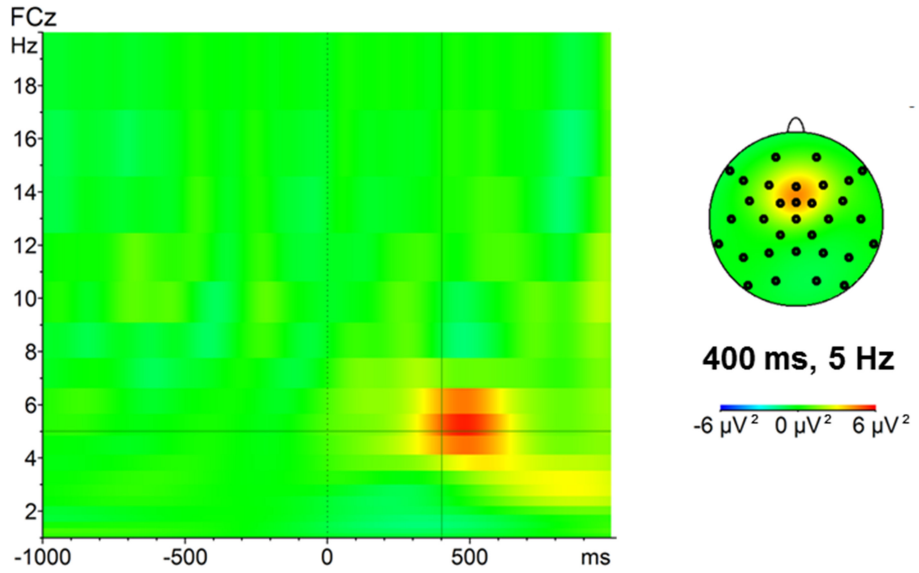

Figure 41. Study 4: Power difference between misses and wins (misses - wins) at electrode FCz and topography of the difference at 400 ms and 5 Hz, reflecting the outcome effect in theta power.

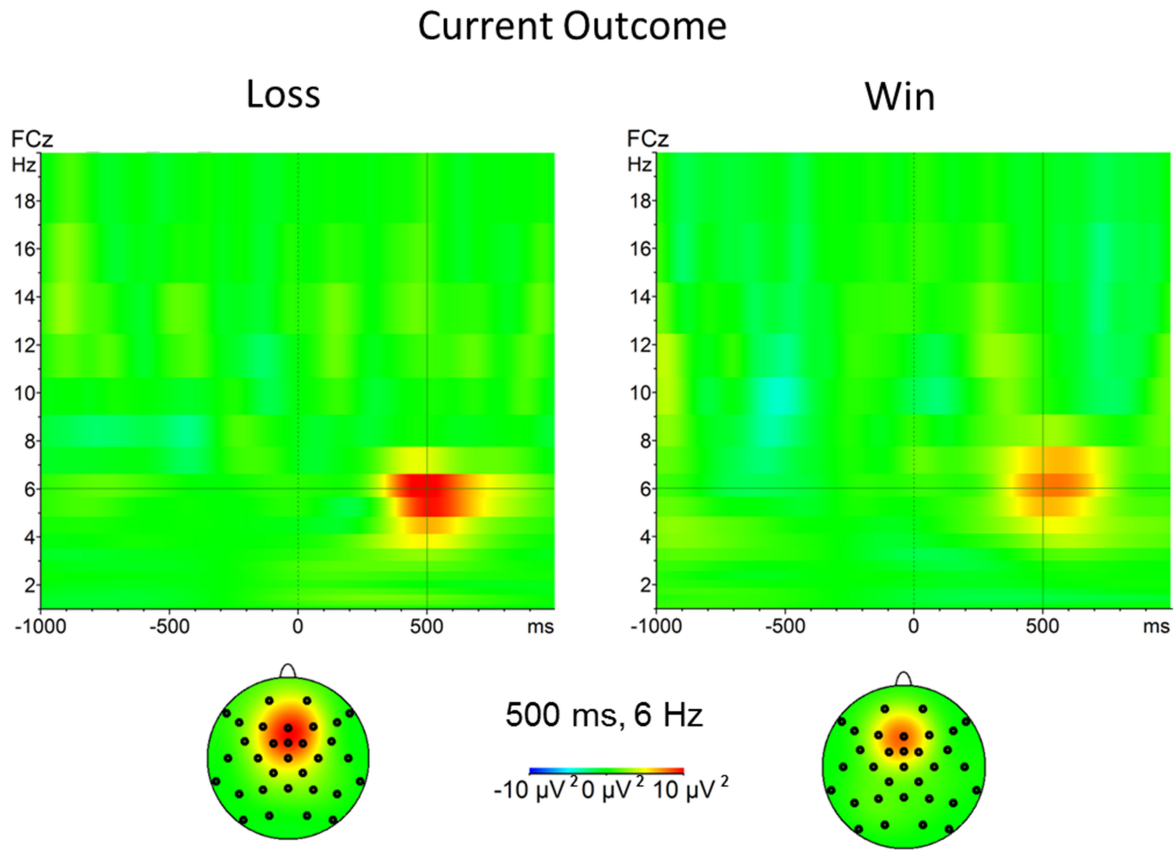

Figure 42. Study 4: Power differences and topographies at FCz reflecting the interaction of current outcome and previous outcomes.
The difference on the left hand side was calculated as power following outcome sequence "winwinloss" minus average power of the sequences "winlossloss", "losswinloss", and "losslossloss". The difference on the right hand side was calculated as the power following outcome sequence "losslosswin" minus average power of the sequences "winwinwin", "winlosswin", and "losswinwin".

7.3.3 P300

The ANOVA of the P300 yielded significant main effects for the factors "current outcome" ($F(1,38) = 17.97$, $p < .001$, $\eta^2_p = .32$), "previous outcomes" ($F(3,114) = 7.95$, $p < .001$, $\eta^2_p = .17$), and "group" ($F(1,38) = 5.02$, $p = .031$, $\eta^2_p = .12$). The P300 was larger following wins compared to misses. Post-hoc paired t-tests indicated that the P300 following wins at positions n-2 and n-1 was significantly larger compared to the other n-2 and n-1 outcome sequences (winloss: $t(39) = 4.30$, $p < .001$; losswin: $t(39) = 4.12$, $p < .001$; lossloss: $t(39) = 3.35$ $p = .002$), whereas the other previous outcome sequences did not significantly differ from one another in the P300 amplitude (all $p > .16$).Finally, the PG group had more positive P300 amplitudes than the nonPG group. Table 28 lists the full results of the ANOVA. Figures 37 and 43 show the ERP waveform at Pz and the mean P300 amplitudes. Figure 44 shows the topography of the difference wave between current losses and wins, reflecting the effect of the factor "current outcome", while Figure 45 shows the topography of the difference wave between the wwx sequence and the other sequences, reflecting the effect of the factor "previous outcomes".

Table 28. Study 4: Results of the ANOVA for the P300

Effect	*F* (*df*)	*p*	η^2_p
"Current Outcome"	17.97 (1,38)	< .001***	.32
"Previous Outcomes"	7.95 (3,114)	< .001***	.17
"Group"	5.02 (1,38)	.031*	.12
"Current Outcome" x "Previous Outcomes"	0.30 (2.631,99.990)	.799	.01
"Current Outcome" x "Group"	0.54 (1,38)	.469	.01
"Previous Outcomes" x "Group"	0.43 (3,114)	.729	.01
"Current Outcome" x "Previous Outcomes" x "Group"	0.42 (2.631,99.990)	.717	.01

Note. *$p < .05$. **$p < .01$. ***$p < .001$

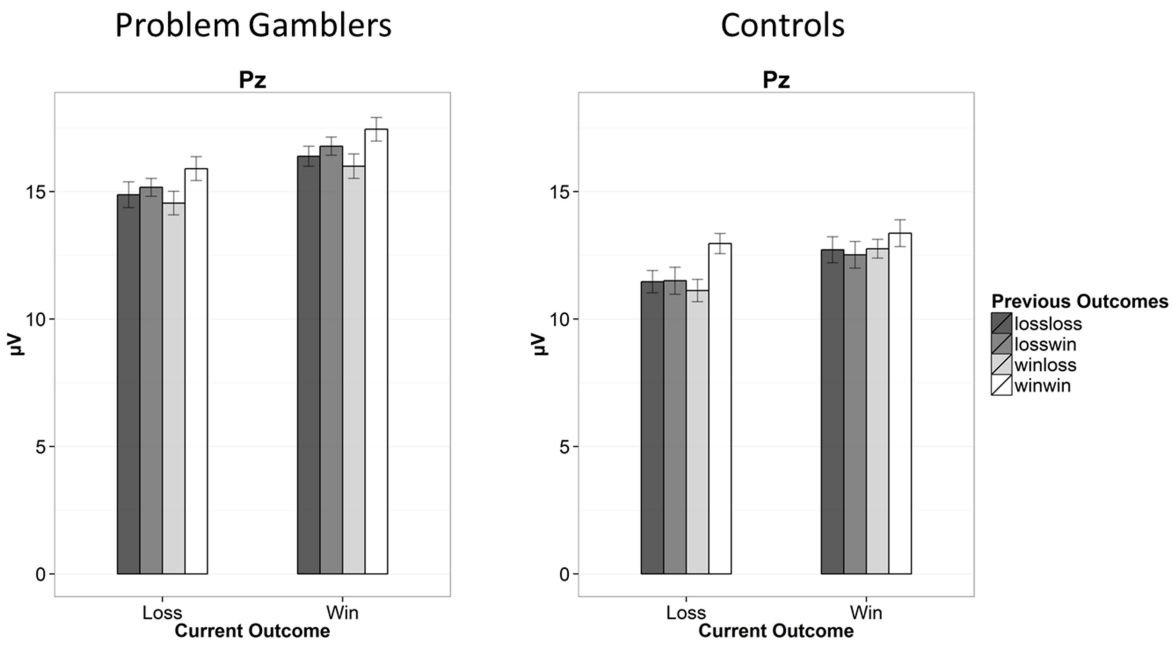

Figure 43. Study 4: Mean P300 values at electrode Pz for problem gamblers and controls. Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

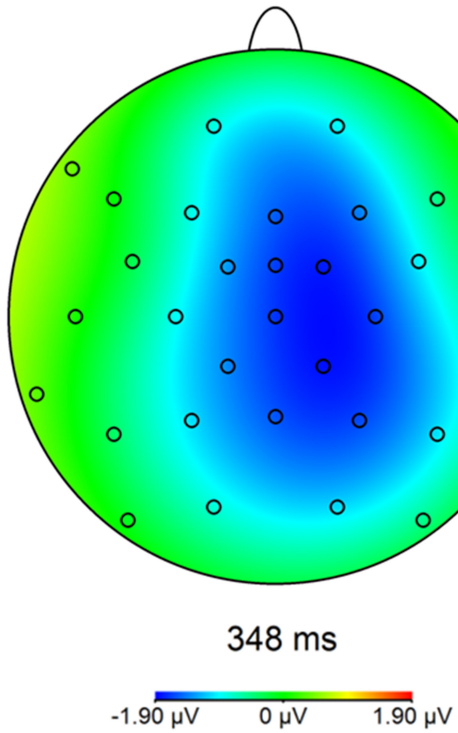

Figure 44. Study 4: Topography of the difference current loss - current win across all participants, reflecting the main effect of current outcome for the P300.

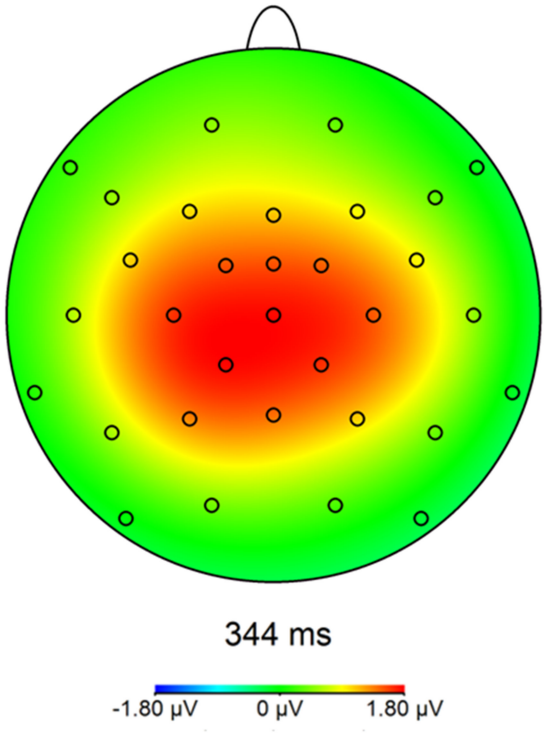

Figure 45. Study 4: Topography of the difference between wwx and the aggregate of the other (wlx, lwx, llx) sequences, reflecting the main effect of previous outcome for the P300.

7.3.4 Probability ratings

The analysis of the probability ratings yielded marginally significant main effects of the factors "previous outcomes" ($F(1.766,67.102) = 2.86$, $p = .071$, $\eta^2_p = .07$) and "group" ($F(1,38) = 3.41$, $p = .073$, $\eta^2_p = .08$). The PG group generally rated their probability of winning in the next trial slightly higher than the nonPG group. Post-hoc paired t-tests indicated that the probability of winning ratings were (marginally) significantly larger following the previous outcome sequence "winwin" compared to the other previous outcome sequences (winloss: $t(39) = 2.29$, $p = .027$; losswin: $t(39) = 3.01$, $p = .005$; lossloss: $t(39) = 1.75$ $p = .088$), whereas the other previous outcome sequences did not differ from one another (all $p > .68$). Table 29 shows the full ANOVA results, while Figure 46 shows the mean probability ratings for problem gamblers and controls.

Table 29. Study 4: Results of the ANOVA for the ratings on probability of winning in the next trial

Effect	*F* (*df*)	*p*	η^2_p
"Current Outcome"	2.04 (1,38)	.161	.05
"Previous Outcomes"	2.86 (1.766,67.102)	.071	.07
"Group"	3.41 (1,38)	.073	.08
"Current Outcome" x "Previous Outcomes"	2.14 (2.276,86.486)	.117	.05
"Current Outcome" x "Group"	0.80 (1,38)	.376	.02
"Previous Outcomes" x "Group"	1.88 (1.766,67.102)	.165	.05
"Current Outcome" x "Previous Outcomes" x "Group"	0.47 (2.276,86.486)	.650	.01

Note. **p* < .05. ***p* < .01. ****p* < .001

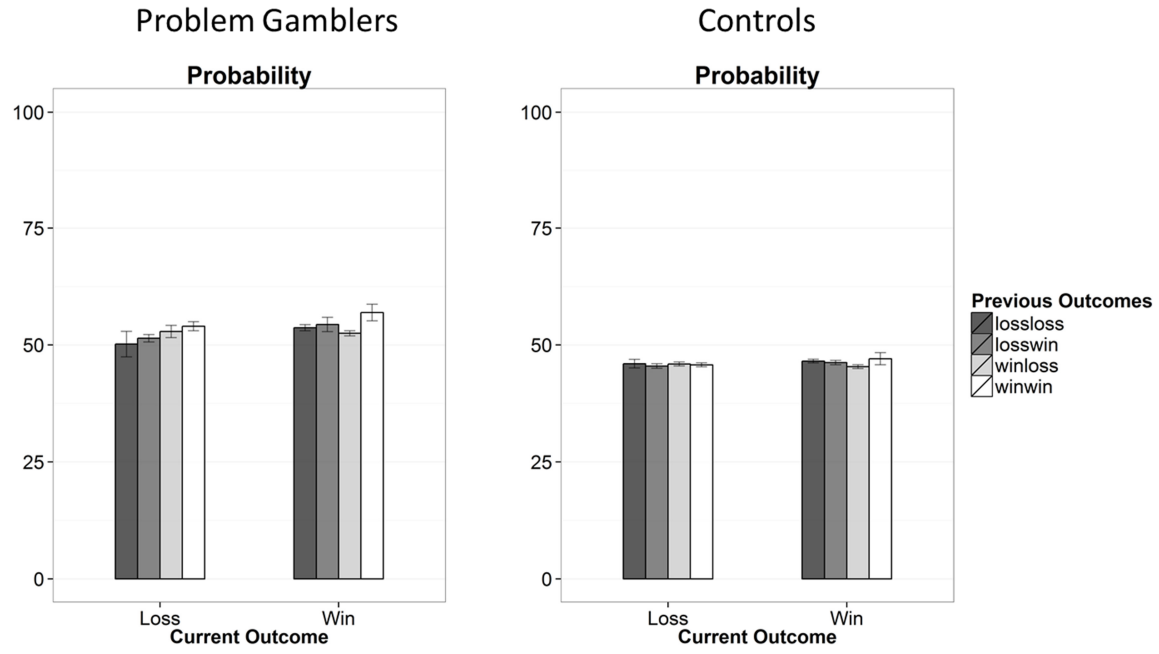

Figure 46. Study 4: Subjective ratings of probability of winning in the next trial for problem gamblers and controls.
Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

7.3.5 Choice behavior

Table 30 shows the mean number of runs of a given length separately for the PG and nonPG group. The ANOVA yielded a marginally significant main effect of the factor "group" ($F(1,38) = 3.93$ $p = .055$ $\eta^2_p = .09$). The PG group showed a trend of switching the chosen side more often compared to the nonPG group. Table 31 shows the full ANOVA results. Figure 47 shows the mean probability of choosing the last outcome symbol as a function of previous run length.

Table 30. Study 4: Mean number of runs of a given length per group

	Run Length				
Group	1	2	3	4	5
PG	122.05 (6.82)	62.50 (3.41)	29.50 (2.78)	14.45 (2.86)	6.70 (1.84)
nonPG	121.15 (7.09)	60.30 (3.08)	30.25 (2.69)	14.35 (2.96)	7.75 (3.16)

Note: *SD* values are given in parentheses

Table 31. Study 4: Results of the ANOVA for choice behavior

Effect	*F* (*df*)	*p*	η^2_p
"Run Length"	1.13 (2.635,100.121)	.338	.03
"Group"	3.93 (1,38)	.055	.09
"Run Length" x "Group"	0.44 (2.635,100.121)	.700	.01

Note. *$p < .05$. **$p < .01$. ***$p < .001$

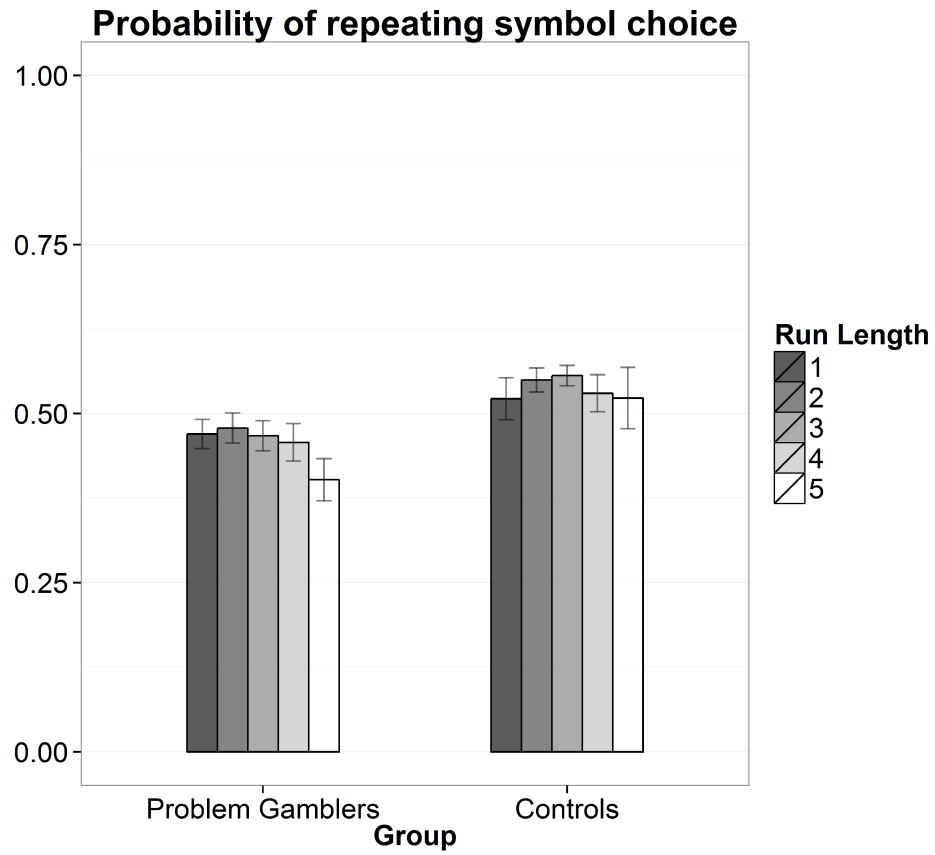

Figure 47. Study 4: Probability of choosing the last outcome symbol as a function of run length of this outcome symbol for problem gamblers and controls.
Error bars denote SEM for within-subject designs according to Cousineau (2005) and Morey (2008).

7.4 Discussion

To sum up, the sequence of previous outcomes showed an effect on theta power and P300, but not on the FRN. Theta power was increased when the current outcome was preceded by two opposite outcomes. The P300 was largest when the two previous outcomes were wins. The data on the probability of winning in the next trial showed a trend in the same direction, with the ratings being slightly higher when the previous two outcomes were wins. Analysis of the choice behavior showed no effect of the length of previous runs of coin sides.

7.4.1 Processing of outcomes

7.4.1.1 FRN

It was expected that the FRN should be increased if the current outcome was preceded by two opposite outcomes, with the effect being stronger in the PG group. However, no support for this hypothesis was found. The sequence of the two previous outcomes did not have an effect on the FRN and there was no interaction with the group factor. Similar to previous studies (e.g. Hajcak et al.,

2006; Osinsky et al., 2012; San Martin, Manes, Hurtado, Isla, & Ibañez, 2010; Yeung & Sanfey, 2004), the FRN showed a frontocentral distribution, with higher values at electrodes FCz and Fz compared to Cz. Remarkably, losses did not produce a larger FRN in this paradigm, expect for a trend at the Fz electrode in the PG group. Since this effect has previously been shown in numerous studies (e.g. Gehring & Willoughby, 2002; Kreussel et al., 2012; Osinsky et al., 2012; Ulrich & Hewig, 2014; Yeung & Sanfey, 2004) and was also present in the wheel of fortune paradigm using the same sample, the lack in the current paradigm might be explained by a methodological issue. The majority of the sample (30 out of 40 participants) completed the wheel of fortune first and the coin toss second. Previous studies have indicated that ERPs (e.g. the ERN) might habituate over time (Rushby, Barry, & Doherty, 2005; Segalowitz et al., 2010). Both the wheel of fortune and the coin toss are gambling paradigms, involving the same amounts (10 Cents) and probabilities (50%) of wins and losses. Thus, the reaction to the outcomes might have habituated over time, making it more difficult to detect differences between wins and losses. In a similar manner, possible effects of previous outcome sequences might have diminished over time. However, additional exploratory analyses run separately for those who completed the coin toss paradigm first and who completed it second, showed no outcome effect on the FRN in the group who completed the coin toss first ($F(1,8) = 0.01$, $p = .936$, $\eta^2_p < .01$), arguing against the habituation hypothesis. Furthermore, as will be discussed below, the analysis of the theta power yielded effects of the current outcome and an interaction of current outcome and previous outcome sequences. This points towards another possible methodological issue that might have contributed to the lack of effects in the FRN. Based on previous studies (e.g. Hajihosseini & Holroyd, 2013; Osinsky et al., 2012; Yeung & Sanfey, 2004), the FRN was quantified in a peak-to-peak manner, involving the detection of the P2 and N2 peaks separately per subject and outcome sequence. The current paradigm contained a rather small number of trials per outcome sequence (30 at most). Thus, peak detection in the averaged ERP might not have been reliable. The analysis of theta power, on the other hand, is not affected by this problem, since it does not involve the detection of peaks.

Finally, as in the wheel of fortune paradigm, the PG group showed a smaller peak-to-peak FRN in the coin toss paradigm. This suggests that the results in the wheel of fortune paradigm are not due to the paradigm per se, but rather reflect a genuine difference between the groups. As mentioned above, the reduced peak-to-peak FRN in the PG group might reflect a generally more favorable evaluation of all outcome types.

7.4.1.2 Theta

As shown in previous studies, theta power was largest at frontocentral electrode sites (e.g. Bernat, Nelson, & Baskin-Sommers, 2015; Cavanagh, Zambrano-Vazquez et al., 2012; Cohen et al., 2007). The effect of current outcome (increased theta power for losses compared to wins) was also larger at frontocentral sites.

It was expected that theta power should be increased when the current outcome breaks an outcome streak, that is, it is different from the previous outcomes. This pattern showed up in the results. For current wins, theta power was highest if the previous two outcomes had been losses. For current losses, theta power was highest when the previous two outcomes had been wins. Thus, these results mirror the FRN findings reported by Osinsky et al. (2012). Theta power has been shown to be increased for unexpected outcomes (e.g. Cavanagh, Zambrano-Vazquez et al., 2012; Cohen et al., 2007; Hajihosseini & Holroyd, 2013; Tzur & Berger, 2009). The repetition of an outcome might lead people to believe that this outcome is more likely to be repeated again (see, for example, a model of expectancy proposed by Squires et al., 1976). The subsequent occurrence of the other outcome is a violation of this expectation, thus leading to increased theta power in the processing of this outcome.

7.4.1.3 P300

In accord with previous studies, the P300 was larger following wins compared to losses (e.g. Kreussel et al., 2012; Ulrich & Hewig, 2014; Zhou et al., 2010). Furthermore, the P300 was also influenced by the previous outcome sequence. However, contrary to the expectation, this effect did not interact with the current outcome. Irrespective of the current outcome, the P300 amplitudes were more positive when the two preceding outcomes were wins. This pattern is similar to the results reported by Osinsky et al. (2012) for the P200, which was larger following current wins and previous sequences of two wins. Based on the effect of previous outcome sequences on the P200, Osinsky et al. (2012) concluded that the local reward history is represented in the P200. The results of the current study indicate that the P300 can take over a similar role. Alternatively, as has been suggested by Mushtaq et al. (2016), the increased P300 following two previous wins might reflect ongoing increased attention triggered by the positive outcomes, facilitating the processing of the next outcome. However, the current results are not in line with previous studies showing increased P300 amplitudes when the current outcome or stimulus disrupts a previous streak of outcomes or run of stimuli (e.g. Jentzsch & Sommer, 2001; Osinsky et al., 2012; Sommer et al., 1990; Squires et al., 1976). Thus, future studies should disentangle the conditions under which different effects of previous sequences on the P300 occur.

Contrary to previous studies (Lole et al., 2015; Oberg et al., 2011), a larger P300 amplitude was observed in the PG group. This could indicate a generally increased motivational significance of the outcomes in the PG group compared to the controls. However, another explanation related to the sequence of the wheel of fortune and coin toss paradigms is also conceivable. In the wheel of fortune paradigm, no difference in the P300 amplitudes between groups was observed. As mentioned above, the majority of the participants first played the wheel of fortune and then the coin toss paradigm. Hence, the participants who started with the wheel of fortune might have already habituated to monetary outcomes by the time they played the coin toss paradigm, leading to a smaller P300. This habituation effect might be smaller in problem gamblers, because of the overall increased motivational salience of monetary outcomes in this group. Exploratory analyses consisting of running the ANOVAs described for the P300 analysis separately for participants who played the coin toss paradigm first and who played it second, indicated no significant P300 difference between the PG and nonPG groups among those who played the coin toss first ($F(1,8) = 0.35$ $p = .570$ $\eta^2_p = .04$) and a significantly larger P300 amplitude in the PG group for those who played the coin toss second ($F(1,28) = 5.12$ $p = .032$ $\eta^2_p = .16$). This supports the idea that the control group might have habituated to the outcomes more quickly.

7.4.1.4 No differences between problem gamblers and controls

Contrary to the corresponding hypothesis, the PG group did not differ from the nonPG group in the effect of previous outcome sequences on the processing of the current outcome, irrespective of the analyzed variable (FRN, theta power, P300). Since the rating data (see below) also did not show differential effects of the previous outcome sequences between groups, it might be concluded that problem gamblers and controls are influenced similarly by previous outcome sequences. Alternatively, differences between groups might occur at later processing stages which were not captured by the current analyses.

7.4.2 Rating data

For the ratings of probability of winning in the next trial, it was expected that streaks of wins should lead to increased ratings, with the effect being stronger in problem gamblers. The results do not fully support these hypotheses. At a trend level, previous outcomes had an effect on probability ratings, with probability ratings being highest following two previous wins. However, this was the case irrespective of the actual current outcome[20]. Thus, it seems that previous streaks of wins can lead to longer lasting increased expectations of winning, even if the streak is disrupted by a loss in

[20] As Figure 46 shows, the probability ratings were higher for the outcome sequence “winwinwin” compared to “winwinloss” at a descriptive level. However, for both current wins and current losses, the previous sequence “winwin” descriptively led to the highest probability ratings.

the meantime. Since participants could not learn an optimal behavior in this task, as the outcome sequence was predetermined, the increased expectation of winning following two previous wins can be seen as indication of a hot hand fallacy, which affected both problem gamblers and controls equally. However, at a trend level, problem gamblers were generally more optimistic regarding their chances of winning than the control group. Overestimating one's chances of winning in a gambling game is part of gambling-related cognitive distortions. Thus, this result fits with previous studies indicating increased cognitive distortions in pathological and problem gamblers (e.g. Joukhador et al., 2004; MacLaren et al., 2015; Myrseth et al., 2010).

7.4.3 Choice behavior

Concerning choice behavior, it was expected to see a gambler's fallacy like behavior, that is, decreased probabilities of choosing a symbol again following a run of this symbol, which was predicted to be stronger in problem gamblers. However, the analysis showed no effect of run length on choice behavior, that is, participants' choices were not influenced by the previous number of repetitions of a coin side. Previous studies have shown evidence for the gambler's fallacy, with participants choosing a symbol less likely, the longer the previous run of this symbol was (e.g. Ayton & Fischer, 2004; Barron & Leider, 2010; Studer et al., 2015). Some studies kept the previous outcome sequence visible on screen for the participants to minimize working memory demands (Barron & Leider, 2010; Studer et al., 2015), possibly also making the previous outcomes more salient. In the current paradigm, previous outcomes were not shown on screen, which might have reduced effects of previous runs on choice behavior. There were also no differences between problem gamblers and controls regarding the effect of previous runs of a symbol. However, the results showed a trend for a general difference between problem gamblers and controls in their choice behavior, with problem gamblers choosing the previous outcome symbol less often than the control group. In a way, this could be interpreted as a general gambler's fallacy like tendency in problem gamblers compared to controls. Problem gamblers might generally expect the outcome symbol to change more often than the control group does, which is reflected in their choice behavior. This does not fully correspond to the classical gambler's fallacy, which usually refers to longer runs of an outcome, but nevertheless points towards a possible misconception of randomness in problem gamblers, contributing to the choice behavior observed here and the increased self-reported cognitive distortions shown in studies 1 and 2, as well as in previous research (e.g. Joukhador et al., 2004; MacLaren et al., 2015; Myrseth et al., 2010).

7.4.4 Limitations

Some limitations should be kept in mind when interpreting the current results. As already mentioned in section 4.4.5, one can argue about the choice of the control group in problem gambling research. Further limitations concern methodological issues. First, it would have been preferable to fully balance the order of the wheel of fortune and the coin toss paradigms. Second, concerning the choice behavior, the current paradigm did not control for the occurrence of runs of symbols. Instead, the streaks of outcomes were controlled. The control of both runs of symbols and streaks of outcomes while still allowing participants to choose a symbol on each trial is not possible. Since the emphasis was put on outcome processing, the outcome sequences were controlled. However, the occurrence of runs of symbols did not seem to differ too much across participants, even though it was not controlled.

7.5 Summary

The current results showed that the break of an outcome streak resulted in increased theta power during outcome processing. Furthermore, streaks of previous wins led to increased P300 amplitudes and a trend for higher probability of winning ratings. Similar to study 1, problem gamblers showed reduced FRN amplitudes to all outcomes, indicating a more favorable evaluation of the outcomes compared to the control group. At a trend level, problem gamblers rated their chances of winning higher, suggesting an optimism bias. Furthermore, they tended to switch their chosen outcome symbol relative to the previous outcome more often, indicative of a gambler's fallacy tendency, although this notion did not increase significantly with run length of the previous outcome.

8 Discussion

8.1 Summary of the studies

To assess the processing of near outcomes and outcome sequences, as well as their modulation by gambling problems, four studies were conducted. Studies 1, 2, and 3 used the wheel of fortune paradigm to investigate the processing and evaluation of near outcomes, using EEG (study 1), fMRI (study 2) and peripheral physiology (study 3). Study 4 used a coin toss paradigm to analyze the processing of outcome sequences using EEG. The influence of gambling problems was assessed by comparing a group of problem gamblers to a group of controls (studies 1 and 4) and by integrating gambling problems into the analysis as a continuous variable (studies 2 and 3). Furthermore, studies 1 to 3 evaluated the relationship between gambling problems and various personality traits. The main findings will be summarized in the following paragraphs.

Studies 1 to 3 showed differences in the processing of near and full outcomes, although these two outcome types have identical valence (win vs. loss) and amounts of money involved. Near compared to full outcomes elicited a smaller P300 (study 1), increased activity in the bilateral parietal cortex (study 2) as well as longer IBIs (study 3). There were no consistent differences in the self-reported valence and motivation following near compared to full outcomes.

Evidence for the modulation of near outcome processing by gambling problems was found in study 3. Descriptively, participants with gambling problems showed a stronger IBI reaction to full misses relative to the other outcomes, whereas participants with no gambling problems showed the strongest reaction to full wins. Furthermore, with increasing gambling problems near compared to full outcomes were followed by increased SCRs.

Study 4 showed that theta power was increased following the break of a streak of outcomes. Furthermore, P300 amplitude was increased following two previous wins. These effects were not modulated by gambling problems. Concerning choice behavior, there was no evidence of the classical gambler's fallacy.

Studies 1 and 4 showed that problem gamblers generally showed decreased FRN amplitudes and at a trend level rated their chances of winning in the gambling games higher than the control group. They also chose the previous outcome symbol less often than controls in study 4. Furthermore, self-rated arousal in study 1 was higher in problem gamblers.

Concerning personality and problem gambling, the studies showed increased self-reported cognitive distortions and belief in good luck for problem gamblers. Furthermore, "Urgency", a facet

of self-reported impulsivity, was positively related to gambling problems. Finally, study 3 showed that risk-taking with increased gambling problems is specific for the domain of gambling.

8.2 Processing and evaluation of near outcomes

Studies 1, 2 and 3 showed that near outcomes in the wheel of fortune elicit smaller amplitudes in the P300, increased activation in the bilateral superior parietal cortex, and longer IBIs compared to full outcomes. There was no evidence that these effects were valence specific, thus, irrespective of the outcome being a win or a miss, outcome closeness had the same effects.

Taken together, the reduced P300, the increased activity in the superior parietal cortex and the longer IBIs could indicate that near outcomes required more attention to be processed properly. According to Johnson (1986), the P300 amplitude is, among others, related to stimulus discriminability and is lower when the stimuli are harder to discriminate. This could be the case for near outcomes, since the wheel stops close to the boundary between two color fields, which might not render the outcome color discernible at the first glance. As a consequence, visual attention might be directed to the stimulus, as indexed by increased activity in the superior parietal cortex, which has been shown to be involved in spatial attention (Kanwisher & Wojciulik, 2000). Furthermore, longer IBIs have also been linked with increased attention and perceptual processing (Lacey, 1967; van der Molen, Somsen, & Jennings, 1996)

Previous research has shown that negative feedback (Crone et al., 2003; Mueller et al., 2010; Somsen et al., 2000) and errors (Hajcak et al., 2003) are followed by longer IBIs/a deceleration in heart rate relative to wins and correct responses. Analogue components in the ERP are the FRN and ERN, occurring after negative feedback (Gehring & Willoughby, 2002; Miltner et al., 1997) and errors (Gehring et al., 1990), respectively. The cardiac deceleration following errors and negative feedback has been interpreted similarly to the FRN (Somsen et al., 2000), which has been taken to indicate a negative evaluation of the eliciting stimulus (Hajihosseini & Holroyd, 2013). Applying this interpretation to the cardiac deceleration indicates a more negative initial evaluation of near compared to full outcomes. Interestingly, previous studies using the wheel of fortune paradigm could show similar effects on the FRN, which was increased (more negative) for near versus full outcomes (Ulrich & Hewig, 2014; Weiß, 2014), although this effect could not be replicated in study 1.

The more negative initial evaluation of near outcomes could be related to the difficulty in discerning them, making these outcomes somewhat ambiguous. It has been shown, that similarly to negative feedback, ambiguous feedback also elicits an FRN (Gu, Huang, & Luo, 2010; Hirsh & Inzlicht, 2008). Furthermore, ambiguous bets, for which the outcome probabilities are not known, have been shown to be rated as more negative than risky bets, for which the outcome probabilities are explicitly

stated (Rubaltelli, Rumiati, & Slovic, 2010). In addition, previous research has shown that near misses are also conceptually ambiguous. Dixon and Schreiber (2004) showed that participants perceive near misses as being between a win and a full miss. It is conceivable that the same holds for near wins. Thus, the more negative evaluation of near outcomes as indexed by the longer IBIs might be related to the perceptual and conceptual ambiguity of those outcomes. However, this initial negative evaluation is not present in the rating data, which consistently showed no valence differences between near and full outcomes. This suggests that for their subjective ratings, participants relied more on the feedback whether they had won or lost money, which was the same for near and full outcomes.

Finally, the studies presented here could not replicate previous findings of the involvement of reward-related circuitry, like the ventral striatum, in near miss processing (Chase & Clark, 2010; Clark et al., 2009; Dymond et al., 2014), or valence and motivation effects of near misses (Clark et al., 2009; Qi et al., 2011). The analyses were mainly focused on finding common effects elicited by near outcomes, so one could argue that the previous findings were not replicated because near outcomes were not analyzed separately for wins and misses. However, potential differential effects of near outcomes depending on outcome valence would have shown up in the corresponding interaction terms, which did not indicate any valence specificity of closeness effects. The different results compared to previous studies might be due to different paradigms used, more specifically different probabilities of the outcomes. For example, the slot machine used by Clark and colleagues (e.g. Clark et al., 2009) delivers wins on 1/6 of the trials and near misses on 2/6 of the trials. It is conceivable that near miss effects also depend on the probability of wins, with the effects being stronger the less frequently wins occur. For example, the frustration reflected in the valence ratings following near misses (Clark et al., 2009; Qi et al., 2011) might be increased if wins are rather rare events.

In summary, the results indicate that near outcomes are initially evaluated as being more negative than their full counterparts. This initial negative evaluation might stem from the ambiguity inherent in these stimuli. Future studies could address to what extent conceptual and visual ambiguity affect this evaluation.

8.3 Processing of outcome sequences

Study 4 showed that the break of an outcome streak led to a phasic increase in theta power. Furthermore, P300 amplitudes were larger when the two preceding outcomes had been wins (irrespective of the current outcome). Subjective ratings showed a trend for a higher expectancy of winning again following a sequence of two previous wins.

Phasic theta increases have been shown to be related to the expectancy of an outcome, with larger phasic theta increases following unexpected outcomes (Cavanagh, Figueroa et al., 2012; Cohen et al., 2007; Hajihosseini & Holroyd, 2013). It is conceivable that participants expected outcome streaks to continue and thus showed a phasic theta increase when, contrary to their expectations, the outcome streaks did not continue. In terms of the fallacies discussed, this might indicate that participants show a hot hand fallacy and expect winning streaks to continue. However, based on the EEG results, this expectation does not seem to be specific for wins, but rather occurs for outcome streaks in general, such that a win following a streak of losses also leads to increased phasic theta power.

Contrary to previous studies (Jentzsch & Sommer, 2001; Matt et al., 1992; Osinsky et al., 2012; Sommer et al., 1990; Squires et al., 1976), we found no effect of breaks in outcome streaks on the P300. Instead, the P300 was largest when the previous two trials had resulted in wins, a pattern that is similar to the one reported for the P200 by Osinsky et al. (2012). The authors interpret the P200 findings in terms of a local reward history, which is triggered by the current outcome presentation and is indexed in the P200 amplitude. It is conceivable that a similar process is indexed in the P300 in the coin toss paradigm. Future studies should further investigate under what conditions the P300 shows effects of breaks in outcome streaks or stimulus sequences, and under what conditions it reflects differences in prior outcomes only. Differences between the current P300 results and previous studies showing an effect of the break of streaks or runs on the P300 might be related to the complexity of the outcomes used in the current study. While previous studies used simple stimuli (e.g. arrows pointing downwards or upwards for loss or win respectively, Osinsky et al., 2012; high- or low-pitched tones, Squires et al., 1976; Sommer et al., 1990), the outcome stimuli used in the coin toss paradigm were more complex. They consisted of a feedback message (“+10 Cent”/”-10 Cent”) indicating the amount won or lost and a picture of the actual outcome of the coin toss performed by the computer (the heads or tails on a 1 euro coin). The outcome of the coin toss was dependent on the prediction of the participant, since wins and losses were predetermined. That is, if the participant predicted the coin would land on heads and the next outcome in the sequence was supposed to be a win, the outcome of the coin toss would be heads. Thus, win feedback could be indicated by both the heads and tails symbol combined with the “+10 Cent” message, depending on the choice behavior of the participant. It is possible that the sequence effects previously reported for the P300 depend on the use of simple relative to more complex stimuli.

Subjective ratings on the probability of winning in the next trial showed a similar pattern compared to the P300 results. On a trend level, participants rated their chances of winning again in the next trial higher when they had won on the previous two trials. Taken together with the P300

results, this points to a special role of previous win sequences. It is possible that win sequences are more salient in memory and thus have a greater influence on the current behavior. A similar proposal has been made by Rachlin, Safin, Arfer, and Yen (2015). They argue that in gambling the sequence of outcomes, not the single outcomes, are the important unit that influences behavior. Specifically, they propose that the outcome sequences are perceived as a series of losses terminated by a win. As such, wins are more salient compared to losses.

To sum up, the results indicate that participants had the expectation that a streak of outcomes would continue. Furthermore, a sequence of previous wins was especially salient, as indexed by increased P300 amplitudes and, at a trend level, led to increased expectancies of winning again in the next trial. These results indicate that the P300 could be a neural process underlying the hot hand fallacy. A larger P300 might denote an increased hot hand fallacy, in the sense of an increased belief in winning again. Future studies should test this hypothesis, for example by correlating the P300 amplitudes with the probability ratings, both within and across participants.

8.4 Gambler's fallacy behavior

The choice behavior in studies 1 and 4 did not show signs of the classic gambler's fallacy in the sense of an increased tendency to choose the other outcome symbol following runs of one outcome symbol of increasing length.

Interestingly, in the wheel of fortune paradigm, there was a marginal effect of run length. With increasing run length, the tendency to choose the same color as in the run actually increased, instead of decreased. As such, this pattern rather reflects the belief in a hot outcome, instead of the gambler's fallacy (Sundali & Croson, 2006). A potential explanation for this finding is perceived skill involvement. Compared to the coin toss paradigm, participants had more possibilities to interact with the wheel of fortune, by first selecting the color to bet on and then pressing a button to stop the wheel. Especially the button press to stop the wheel might be perceived as an element of skill, suggesting the possibility to learn how to stop the wheel on the chosen color. Following a run of outcomes of one color, participants might have had the feeling of having learnt how to stop the wheel on that color and thus were more likely to choose this color again in the next trial. Compared to the wheel of fortune, the coin toss paradigm likely seemed more random, lacking the alleged skill element. Thus, participants might have felt less control over the outcomes in the coin toss paradigm. Shao, Sun, and Lee (2016) showed that participants showed a gambler's fallacy pattern in their bets (smaller bets following wins compared to losses) following a trial in which the computer choose the card in a card guessing game, thus reducing participants supposed ability to influence the game. However, contrary to the expectations, participants also did not show the classic gambler's fallacy in

the coin toss paradigm. Working memory demands might have contributed to the absence of this effect. Participants had to remember the previous outcome sequence without any external aid in the current studies, whereas some previous studies have displayed the previous outcome sequence to decrease working memory demands (Barron & Leider, 2010; Studer et al., 2015).

In summary, participants did not show a classic gambler's fallacy behavior in the wheel of fortune and coin toss paradigms. However, the color choice behavior in the wheel of fortune indicated a hot outcome fallacy among the participants. Future studies should explicitly test to what extent these results can be attributed to different amounts of perceived control in the two paradigms.

8.5 Modulation of the processing of near outcomes and outcome sequences and choice behavior by problem gambling

The results of studies 1, 2 and 4 did not show evidence for a strong modulation of outcome processing by gambling problems. The FRN and P300 amplitudes as well as theta power following near outcomes and outcome sequences were not influenced by gambling problems, with all participants showing decreased P300 amplitudes following near compared to full outcomes, increased P300 amplitudes following sequences of two previous wins and increased theta power following a break in outcome sequences. The fMRI results hint at a possible modulation of superior frontal cortex activity following near outcomes. However, this result is based on an exploratory uncorrected analysis of the data and thus should be interpreted with caution and rather be used to generate hypotheses for future studies.

Study 3 showed that peripheral reactions to near outcomes are modulated by gambling problems. With increasing gambling problems participants showed increased IBI reactivity to full misses compared to the other outcomes, as well as increased SCR following near compared to full outcomes. The latter result indicates that near outcomes are more arousing than full outcomes for people with gambling problems. The results of the IBI reactivity indicate that with increasing gambling problems, full wins, near wins and near misses are being processed similarly, with full misses eliciting a distinct response. Participants with gambling problems might process full wins, near wins and near misses as wins or win-like outcomes, with full misses being perceived as the only real loss.

In sum, near outcomes elicit more physiological arousal with increasing gambling problems and are processed as more win-like relative to full misses.

Concerning the processing of outcome sequences, there were no differences between participants with respect to gambling problems. Similar to healthy controls, participants with

gambling problems showed increased theta activity following breaks of streaks in the outcome sequence, as well as increased P300 amplitudes and a trend for increased probability of winning ratings following sequences of two previous wins. Thus, in the current study win sequences seemed to be equally salient for participants with and without gambling problems. However, in the coin toss paradigm problem gamblers showed a trend of generally switching the predicted symbol more often than the control group, which might be indicative of an increased gambler's fallacy in this group.

In sum, the results regarding outcome sequences show no differences in the processing of outcome sequences between problem gamblers and controls, but point towards differences in decision behavior, with problem gamblers showing a tendency towards a gambler's fallacy like behavior.

8.6 Further differences between problem gamblers and controls

The studies presented in the previous sections showed some general differences between problem gamblers and controls. First, studies 1 and 4 showed that the problem gamblers generally had smaller peak-to-peak FRN amplitudes compared to controls. This could indicate an increased reward positivity in problem gamblers, reflecting a generally more favorable evaluation of all gambling outcomes relative to the control group. Combined with the increased arousal ratings in problem gamblers (see below), this might indicate that problem gamblers compared to controls in general found the gambling games used in the current studies more interesting. Some previous studies have shown evidence for a reward- and punishment-hyposensitivity in problem gamblers (Balodis et al., 2012; de Ruiter et al., 2009; Lole et al., 2015), while others have shown evidence of a reward-hypersensitivity in problem gamblers (Hewig et al., 2010; Oberg et al., 2011). Future studies should specifically test under which condition each of the results is observable. For example, it is conceivable that results indicating a reward- and punishment-hyposensitivity occur in paradigms which are perceived as rather boring by the problem gamblers compared to the gambling games they are used to, while results indicating reward-hypersensitivity have involved paradigms which allowed the gambler to make risky decisions.

Furthermore, problem gamblers also showed increased subjective arousal levels across all outcome types in the wheel of fortune, suggesting that this game was generally more arousing for problem gamblers compared to controls. Similar results have been reported for physiological arousal (Blanchard et al., 2000; Carroll & Huxley, 1994; Sharpe et al., 1995), which during gambling is higher in pathological and problem gamblers compared to controls. The increased arousal ratings for problem gamblers could also indicate that the wheel of fortune paradigm was more attractive or less boring for the problem gambling compared to the control group.

Problem gamblers also showed a trend for rating their chances of winning in the next trial higher than the control group, both in the wheel of fortune and in the coin toss paradigm. This points towards an overly optimistic view regarding the capability of winning games of chance in problem gamblers. This is in line with previous research suggesting an optimism bias in pathological gamblers (Atkins & Sharpe, 2003), as well as overconfidence in a knowledge game (Goodie, 2005; Hudgens-Haney et al., 2013).

Finally, problem gambling was related to several other self-report questionnaires. With increasing gambling problems, participants scored higher on gambling-related cognitive distortions, the impulsivity facet urgency and self-reported risk-taking in the gambling domain. These results replicate previous research showing increased cognitive distortions (Cunningham et al., 2014; Joukhador et al., 2004; Joukhador et al., 2003; MacLaren et al., 2015; Myrseth et al., 2010; Xian et al., 2008) and urgency (Whiteside et al., 2005) in pathological and problem gamblers and also extend the research by showing that risk-taking in problem gamblers seems to be domain specific, as the gambling subscale was the only subscale in the risk-taking questionnaire correlating with the problem gambling screening questionnaire.

In sum, based on the current results, problem gamblers can be characterized as people who, relative to people without gambling problems, are aroused by gambling situations and react more favorably to all outcomes. Furthermore, they show increased gambling-related cognitive distortions, reflected in questionnaires, ratings and behavior, as well as increased impulsivity. The latter does not extend to a generally increased propensity for risk-taking, though, as the current results suggest specificity for the gambling domain.

8.7 Future studies

The results obtained in the current studies point towards interesting further questions and serve as a starting point for future studies. First, future studies should test whether the decreased P300, increased parietal activation and increased IBIs following near outcomes are related to the way near outcomes are operationalized in the wheel of fortune paradigm, possibly requiring more visual attention to correctly perceive them. To do so, closeness should be operationalized independently from spatial configuration. In the wheel of fortune, closeness was defined based on the spatial configuration of the wheel at the end of the spin. Another possibility to operationalize closeness is based on proximity in a range of numbers. For example, a paradigm involving a die throw could be used. Participants repeatedly have to throw a die (e.g. a 20-sided die) and win a small amount of money if the result falls within a certain range of numbers (e.g. 7-13). Otherwise, they lose a small amount of money. In this case, outcomes that are relatively far away from the boundaries of the

winning range constitute full outcomes (e.g.: full miss: 1, 19; full win: 10, 11), whereas numbers close to the boundaries of the winning range constitute near outcomes (e.g. near miss: 6, 14; near win: 7, 13). Of course, the operationalization of closeness in such a paradigm still depends on how close the outcome is to a predefined boundary. However, the closeness no longer has to be integrated in the visual display of the outcome, since one can simply display the corresponding number on the die and all numbers should be discernable equally well.

Further studies could also combine the biopsychological measures used in the current studies in a single study. This enables the analysis of potential correlations between the various measures. For example, using the method of cardio-electroencephalographic covariance tracing (CECT) as described by Mueller et al. (2010) could help to elucidate the exact time course of covariations between EEG and heart activity in the wheel of fortune paradigm and in the processing of near outcomes in general.

Furthermore, future studies might aim at including pathological, problem and non-problem gamblers in their samples. This makes it possible to examine whether there is a quantitative or a qualitative difference between the deficits shown by problem and pathological compared to non-pathological gamblers, and thus whether gambling problems can be seen as a continuum or whether pathological and problem gamblers need to be seen as separate classes. Previous results on this issue have been mixed, with some researchers arguing that pathological gamblers constitute a separate class (James, O'Malley, & Tunney, 2014; Kincaid et al., 2013) while other studies showed that gambling problems represent a continuum (Carragher & McWilliams, 2011; Strong & Kahler, 2007). However, so far studies have mainly focused on the symptoms of problematic gambling behavior as included in the *DSM* or in screening questionnaires for pathological gambling. Future studies should extend this line of research to other deficits, for example the reduced peak-to-peak FRN observed in the current studies, to address the question whether these deficits increase along with gambling problems and thus fit the idea of a continuum, or whether they are only present in pathological gamblers, supporting the idea of a separate class.

In addition, future research might use longitudinal designs to assess whether the differences between problem gamblers and controls shown in the current studies develop over time as gambling problems occur, or whether they represent a predisposition increasing the risk for developing gambling problems in the first place.

Another direction for future studies is the more thorough investigation of domain specificity of risk-taking in gamblers. Future studies in this direction should aim to include a broader range of gamblers, including casual, regular, problem and pathological gamblers. The preferred gambling game should also be assessed, as studies have shown personality and behavioral differences among

gamblers with different favorite gambling games (Goudriaan et al., 2005; Lorains et al., 2014; Savage, Slutske, & Martin, 2014). Such personality differences might also extend to risk-taking behavior. Furthermore, the specificity of the risk-taking behavior should not only be evaluated by means of self-report questionnaires, but also by using behavioral tasks, for example. Such tasks should include tasks related to monetary risk and gambling (e.g. BART task, Lejuez et al., 2002; Blackjack Task, Hewig et al., 2007), but also tasks that assess risk behavior unrelated to money (e.g. Crossing the Street Test, Rubio, Hernández, Zaldívar, Márquez, & Santacreu, 2010).

Finally, another line of research could investigate the possibility to change the processing of near outcomes in problem gamblers. Previous research has shown that the processing of outcomes in a gambling game, as indexed by the feedback ERPs, can be influenced by giving instructions to reappraise the outcomes (Yang, Gu, Tang, & Luo, 2013). Furthermore, the subjective evaluation of near misses has been shown to be influenceable by different interventions in recreational and problem gamblers (Dixon, Nastally, Jackson, Habib, & Ninness, 2009; Nastally & Dixon, 2012). Thus, future studies could test short interventions targeting the processing and evaluation of near misses in problem gamblers to assess potential changes in physiological reactions and ensuing gambling behavior.

8.8 Conclusion

Gambling-related cognitive distortions are increased in problem and pathological gamblers (Cunningham et al., 2014; Joukhador et al., 2004; Joukhador et al., 2003; MacLaren et al., 2015; Myrseth et al., 2010; Xian et al., 2008). The current studies aimed at analyzing the neural basis of two kinds of distortions: the illusion of control and the hot hand and gambler's fallacies. Using a multimethod approach, the processing of near outcomes and outcome sequences were analyzed in samples of varying degrees of gambling problems to draw conclusions about the neuronal underpinnings of these distortions.

Concerning near outcomes and the illusion of control, the results point towards the involvement of brain areas and processes involved in attention and salience attribution. However, as noted previously, these results might be due to the specific operationalization of near outcomes in the current studies and thus need to be replicated in future studies. Concerning outcome sequences and the hot hand and gambler's fallacies, results showed an involvement of processes indicating salience and violation of expectations.

Based on the results, some implications for gambling problems can be derived. Participants with gambling problems showed increased physiological arousal and a more win-like processing of near outcomes. This indicates that both near wins as well as near misses are perceived and

processed similar to full wins by people with gambling problems, which in turn might prolong their gambling behavior, as they subjectively experience more win-like outcomes than non-problem gamblers. This might also explain why problem gamblers tended to be more optimistic than controls concerning their chances of winning. If they subjectively experience a higher proportion of the outcomes as win-like, they will naturally derive increased expectations of winning again, compared to non-problems gamblers who subjectively experience less win-like outcomes. Future research should work on developing and testing interventions targeted at changing this misperception in problem gamblers to potentially change their gambling behavior. After all, a miss is as good as a mile.

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10 Annex

A Information for participants

1. Studies 1 and 4

2. Study 2

3. Study 3

B Written informed consent

1. Studies 1 and 4

2. Study 2

3. Study 3

C Instructions for generating personalized key

D Follow-up questionnaire study 3

A Information for Participants

1. Studies 1 and 4

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Teilnehmerinformation

Julius-Maximilians-Universität Würzburg
Lehrstuhl für Psychologie I

Titel der Studie:

Biopsychologische Grundlagen der Glücksspielsucht

Herzlich willkommen bei unserer Studie zum "*Biopsychologische Grundlagen der Glücksspielsucht*"! Wir danken Ihnen für Ihr Interesse an dieser Studie.

Wir untersuchen mit dieser Studie, ob und wie sich die Verarbeitung und Bewertung von Ergebnissen im Glücksspiel zwischen Personen mit und ohne problematischem Glücksspielverhalten unterscheidet.

Ablauf der Studie

Das folgende Experiment besteht aus drei Teilen. Insgesamt dauert das Experiment ca. 2 bis 2,5 Stunden.

Zunächst wird der Versuchsleiter Ihnen ein Elektroenzephalogramm (EEG) anlegen, mit dem ihre Gehirnaktivität während des anschließenden Glücksspiels aufgezeichnet wird. Weitere Informationen zum EEG entnehmen Sie bitte dem beiliegenden Informationsblatt.

Im Rahmen des Glücksspiels werden Sie dann an einem Glücksrad spielen und ein Münzwurf-Spiel spielen. Dazu wird Ihnen von uns ein Geldbetrag zum Spielen auf Ihrem Spielkonto zur Verfügung gestellt. Sie bekommen nach Abschluss beider Spiele das Guthaben Ihrer Spielkontos ausbezahlt. Weitere Informationen zum Ablauf der Spiele erhalten Sie nach dem Anlegen des EEG.

Sollten Sie noch Fragen haben, wenden Sie sich damit bitte an den Versuchsleiter.

Freiwilligkeit und Anonymität

Die Teilnahme an der Studie ist freiwillig. Sie können jederzeit und ohne Angabe von Gründen Ihre Einwilligung zur Teilnahme an dieser Studie widerrufen, ohne dass Ihnen daraus Nachteile entstehen. Auch wenn Sie die Studie vorzeitig abbrechen, haben Sie Anspruch auf eine entsprechende Vergütung für den bis dahin erbrachten Zeitaufwand.

Die im Rahmen dieser Studie erhobenen Daten und persönlichen Mitteilungen werden vertraulich behandelt. So unterliegen diejenigen Mitarbeiter, die durch direkten Kontakt mit Ihnen über personenbezogene Daten verfügen, der Schweigepflicht. Des Weiteren wird die Veröffentlichung der

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Ergebnisse der Studie in pseudonymisierter Form erfolgen, d. h. ohne dass Ihre Daten Ihrer Person zugeordnet werden können.

Datenschutz

Die Erhebung der Daten erfolgt vollständig pseudonymisiert, d. h. an keiner Stelle wird Ihr Name erfragt. Ihre Antworten und Ergebnisse werden unter einem persönlichen Codewort gespeichert, das Sie selbst anhand einer Regel erstellt haben und das außer Ihnen niemand kennt. Die pseudonymisierten Daten werden mindestens 10 Jahre gespeichert. Sie können allerdings, wenn immer Sie dies möchten, die Löschung der von Ihnen erhobenen Daten verlangen. Dazu müssen Sie uns nicht Ihren Namen verraten, sondern nur Ihr Codewort. Für die Erstellung Ihres Codeworts erhalten Sie die auf einem Blatt die Anleitung „Wie erstellen Sie Ihr persönliches Codewort?“ Dieses Blatt verbleibt bei Ihnen.

Vergütung

Für die Teilnahme an der Untersuchung erhalten Sie eine pauschale Vergütung in Höhe von 10 € plus das Guthaben Ihrer Spielekontos. Die Vergütung wird Ihnen in bar ausgezahlt. Bei Empfang der Vergütung in bar müssen Sie eine Quittung mit Angabe Ihres Namens unterschreiben.

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Teilnehmerinformation für EEG-Studien

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Titel der Studie:

Biopsychologische Grundlagen der Glücksspielsucht

Liebe Teilnehmerin, lieber Teilnehmer,

im Rahmen einer wissenschaftlichen Studie möchten wir bei Ihnen ein Elektroencephalogramm (EEG) aufzeichnen. In den folgenden Abschnitten erfahren Sie Näheres über diese Untersuchung. Fragen Sie uns gerne, wenn Sie etwas nicht verstanden haben oder mehr über die Untersuchungsmethode erfahren möchten.

Zweck und Ablauf der Untersuchung

Die Untersuchung dient der Erforschung der Funktionsweise des menschlichen Gehirns.

Während der bevorstehenden Untersuchung wird mit Hilfe von Elektroden, die mit Hilfe einer elastischen Kappe auf der Kopfoberfläche befestigt werden, das EEG aufgezeichnet. Hierbei handelt es sich um die elektrische Aktivität des Gehirns, die an der Kopfoberfläche gemessen werden kann.

Die Aufzeichnung des EEGs ist beim Menschen mit keinen Risiken verknüpft. Der Kontakt zwischen Elektrode und Kopfoberfläche wird über ein Elektrodengel hergestellt. Die verwendeten Chemikalien sind klinisch getestet und lassen sich nach Abschluss des Experiments leicht auswaschen. In seltenen Fällen können trotzdem Hautirritationen auftreten. Manchmal bleiben noch für eine Weile Druckstellen an den Orten zurück, an denen die Elektroden bzw. die Elektrodenkappe befestigt wurde; in ganz seltenen Fällen sind die Stellen, an denen die Elektroden saßen, noch für ein paar Tage sichtbar (z. B. Rötungen). Bitte teilen Sie uns mit, falls Sie an bestimmten Hautallergien oder Überempfindlichkeiten der Haut leiden.

Auffällige Befunde

Die Untersuchung dient ausschließlich Forschungszwecken. Eine medizinische oder psychologische Beurteilung Ihrer Daten erfolgt nicht. Es könnte uns jedoch ein ungewöhnliches Untersuchungsergebnis auffallen. In diesem Fall werden wir Sie darüber informieren und Ihnen empfehlen, dieses Ergebnis bei Ihrem Hausarzt diagnostisch weiter abklären zu lassen. Nur wenn Sie damit einverstanden sind, dass wir Sie ggf. über einen auffälligen Befund informieren, können Sie an dieser Studie teilnehmen. Sofern bei dieser diagnostischen Abklärung eine Erkrankung festgestellt werden sollte, könnten Ihnen daraus unter Umständen Nachteile entstehen, z. B. der Abschluss einer privaten Krankenversicherung oder einer Lebensversicherung erschwert werden.

2. **Study 2**

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Wir untersuchen mit dieser Studie, ob und wie sich die Verarbeitung und Bewertung von Ergebnissen im Glücksspiel zwischen Personen mit unterschiedlichem Glücksspielverhalten unterscheidet.

Ablauf der Studie

Das folgende Experiment dauert ca. 1,5 bis 2 Stunden.

Zunächst werden Sie für die MRT-Messung vorbereitet und anschließend zwei Glücksspiele während der MRT-Messung spielen. Abschließend füllen Sie Fragebögen aus. Weitere Informationen zur MRT-Messung entnehmen Sie bitte dem beiliegenden Informationsblatt.

Im Rahmen des Glücksspiels werden Sie dann an einem Glücksrad spielen und ein Münzwurf-Spiel spielen. Dazu wird Ihnen von uns ein Geldbetrag zum Spielen auf Ihrem Spielkonto zur Verfügung gestellt. Sie bekommen nach Abschluss beider Spiele das Guthaben Ihrer Spielkontos ausbezahlt. Weitere Informationen zum Ablauf des Spiels erhalten Sie nach der Vorbereitung für das MRT.

Sollten Sie noch Fragen haben, wenden Sie sich damit bitte an den Versuchsleiter.

Freiwilligkeit und Anonymität

Die Teilnahme an der Studie ist freiwillig. Sie können jederzeit und ohne Angabe von Gründen Ihre Einwilligung zur Teilnahme an dieser Studie widerrufen, ohne dass Ihnen daraus Nachteile entstehen. Auch wenn Sie die Studie vorzeitig abbrechen, haben Sie Anspruch auf eine entsprechende Vergütung für den bis dahin erbrachten Zeitaufwand.

Die im Rahmen dieser Studie erhobenen Daten und persönlichen Mitteilungen werden vertraulich behandelt. So unterliegen diejenigen Mitarbeiter, die durch direkten Kontakt mit Ihnen über personenbezogene Daten verfügen, der Schweigepflicht. Des Weiteren wird die Veröffentlichung der Ergebnisse der Studie in pseudonymisierter Form erfolgen, d. h. ohne dass Ihre Daten Ihrer Person zugeordnet werden können.

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Datenschutz

Die Erhebung der Daten erfolgt vollständig pseudonymisiert, d. h. an keiner Stelle wird Ihr Name erfragt. Ihre Antworten und Ergebnisse werden unter einem persönlichen Codewort gespeichert, das Sie selbst anhand einer Regel erstellt haben und das außer Ihnen niemand kennt. Die pseudonymisierten Daten werden mindestens 10 Jahre gespeichert. Sie können allerdings, wenn immer Sie dies möchten, die Löschung der von Ihnen erhobenen Daten verlangen. Dazu müssen Sie uns nicht Ihren Namen verraten, sondern nur Ihr Codewort. Für die Erstellung Ihres Codeworts erhalten Sie die auf einem Blatt die Anleitung „Wie erstellen Sie Ihr persönliches Codewort?“ Dieses Blatt verbleibt bei Ihnen.

Vergütung

Für die Teilnahme an der Untersuchung erhalten Sie eine pauschale Vergütung in Höhe von 10 € plus das Guthaben Ihres Spielekontos. Die Vergütung wird Ihnen in bar ausgezahlt. Bei Empfang der Vergütung in bar müssen Sie eine Quittung mit Angabe Ihres Namens und Ihrer Adresse unterschreiben.

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97070 Würzburg

Prof. Dr. Johannes Hewig

Ansprechpartner für eventuelle Rückfragen:

Dipl.-Psych. Natalie Ulrich

Telefon: 0931-31 83438

Teilnehmerinformation für MRT-Studien

Julius-Maximilians-Universität Würzburg
Lehrstuhl für Psychologie I

Titel der Studie:

Biopsychologische Grundlagen der Glücksspielsucht

Liebe Teilnehmerin, lieber Teilnehmer,

im Rahmen einer wissenschaftlichen Studie möchten wir bei Ihnen eine Magnetresonanztomographie (MRT; Kernspintomographie) durchführen. In den folgenden Abschnitten erfahren Sie Näheres über diese Untersuchung. Fragen Sie uns gerne, wenn Sie etwas nicht verstanden haben oder mehr über die Untersuchungsmethode erfahren möchten.

Zweck und Ablauf der Untersuchung

Die Untersuchung dient der Erforschung der Funktionsweise des menschlichen Gehirns.

Die MRT-Technologie ist ein sogenanntes nicht-invasives Verfahren, d. h. es ist für den Körper nach heutigem Erkenntnisstand unschädlich. Im Unterschied zu anderen diagnostischen Verfahren wird bei der MRT-Technologie keine ionisierende Strahlung (Radioaktivität) eingesetzt. Nach heutigem Wissensstand, basierend auf mehr als 20-jähriger Erfahrung mit der MRT-Technologie, die täglich in allen größeren Kliniken eingesetzt wird, sind keine Nebeneffekte bekannt. Darüber hinaus gibt es keine Hinweise auf negative Langzeiteffekte der MRT-Technologie auf den menschlichen Körper.

Sie werden auf einem Tisch liegen, welcher Sie in die zylinderförmige Öffnung des MR-Tomographen hineinfährt, wo sich die starken Magnetfelder befinden. Zusätzlich wird ein Rahmen (die Magnetspule) um Ihren Kopf gelegt. Während der Messung werden Sie ein Klopfen hören. Um Schäden am Gehör zu vermeiden, werden Sie vor der Messung einen Gehörschutz erhalten. Die Untersuchungszeit liegt bei ca. 55 Minuten. Sie werden gebeten, zwei einfache Aufgabe im MR-Tomographen durchzuführen (Dauer ca. 45 min). Danach erfolgt eine genauere Aufnahme von der Struktur Ihres Gehirns (Dauer ca. 10 min). Vor der Untersuchung ist deshalb ein Gang zur Toilette ratsam. Sie haben während der Untersuchung jederzeit die Möglichkeit, mit den Untersuchern über eine Wechselsprechanlage in Kontakt zu treten. Zusätzlich bekommen Sie einen Alarmknopf (Druckball) mit in den MR-Tomographen. Auf Ihren Wunsch hin können Sie jederzeit aus dem MR-Tomographen hinausgefahren werden. Abgesehen von möglichen Unbequemlichkeiten, die vom langen, stillen Liegen resultieren, sollten Sie keine Beschwerden während der Untersuchung haben.

Die Anwendung von Magnetfeldern bei der MRT-Untersuchung schließt die Teilnahme von Personen aus, die elektrische Geräte (z. B. Herzschrittmacher, Medikamentenpumpen usw.) oder Metallteile (z.

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B. Schrauben nach Knochenbruch) im oder am Körper haben. Die räumlichen Verhältnisse im MR-Tomographen lassen es nicht zu, Personen mit starken Rückenbeschwerden oder übermäßigem Übergewicht zu untersuchen. Auch sollten große, schnelle Bewegungen im MR-Tomographen unterbleiben, um keinen Magnetstrom zu induzieren.

Auffällige Befunde

Die Untersuchung dient ausschließlich Forschungszwecken. Eine medizinische oder psychologische Beurteilung Ihrer Daten erfolgt nicht. Es könnte uns jedoch ein ungewöhnliches Untersuchungsergebnis auffallen. In diesem Fall werden wir Sie darüber informieren und Ihnen empfehlen, dieses Ergebnis bei Ihrem Hausarzt diagnostisch weiter abklären zu lassen. Nur wenn Sie damit einverstanden sind, dass wir Sie ggf. über einen auffälligen Befund informieren, können Sie an dieser Studie teilnehmen. Sofern bei dieser diagnostischen Abklärung eine Erkrankung festgestellt werden sollte, könnten Ihnen daraus unter Umständen Nachteile entstehen, z. B. der Abschluss einer privaten Krankenversicherung oder einer Lebensversicherung erschwert werden.

3. Study 3

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Prof. Dr. Johannes Hewig

Ansprechpartner für eventuelle Rückfragen:

Dipl.-Psych. Natalie Ulrich

Telefon: 0931-31 83438

Teilnehmerinformation

Julius-Maximilians-Universität Würzburg
Lehrstuhl für Psychologie I

Titel der Studie:

Glücksrad Peripherphysiologie

Herzlich willkommen bei unserer Studie „Glücksrad Peripherphysiologie"! Wir danken Ihnen für Ihr Interesse an dieser Studie.

Wir untersuchen mit dieser Studie die körperlichen Reaktionen auf Ergebnisse im Glücksspiel.

Ablauf der Studie

Das folgende Experiment besteht aus drei Teilen. Insgesamt dauert das Experiment ca. 1 bis 1,5 Stunden.

Zunächst wird der Versuchsleiter Ihnen Elektroden anlegen, mit denen die Hautleitfähigkeit und die Herzaktivität gemessen werden. Zur Messung der Hautleitfähigkeit wird Ihnen je eine Elektrode an den Zeige- und Mittelfinger der linken Hand geklebt. Zur Messung der Herzaktivität wird Ihnen jeweils eine Elektrode im Bereich des linken und rechten Schlüsselbeins sowie an der linken Seite unterhalb des Brustkorbs geklebt. Die dabei verwendeten Pasten sind hinterher einfach mit einem Papiertuch entfernbar.

Im Rahmen des Glücksspiels werden Sie dann an einem Glücksrad am Computer spielen. Dazu wird Ihnen von uns ein Geldbetrag zum Spielen auf Ihrem Spielkonto zur Verfügung gestellt. Liegt Ihr Gewinn über einem bestimmten Betrag erhalten Sie einen Bonus zusätzlich zu Ihrer Vergütung ausbezahlt. Weitere Informationen zum Ablauf des Spiels erhalten Sie nach dem Anlegen der Elektroden am Computer.

Nach Abschluss des Glücksspiels werden Ihnen die Elektroden abgenommen und Sie füllen Fragebögen und eine Nachbefragung aus.

Sollten Sie noch Fragen haben, wenden Sie sich damit bitte an den Versuchsleiter.

Freiwilligkeit und Anonymität

Die Teilnahme an der Studie ist freiwillig. Sie können jederzeit und ohne Angabe von Gründen Ihre Einwilligung zur Teilnahme an dieser Studie widerrufen, ohne dass Ihnen daraus Nachteile entstehen.

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Auch wenn Sie die Studie vorzeitig abbrechen, haben Sie Anspruch auf eine entsprechende Vergütung für den bis dahin erbrachten Zeitaufwand.

Die im Rahmen dieser Studie erhobenen Daten und persönlichen Mitteilungen werden vertraulich behandelt. So unterliegen diejenigen Mitarbeiter, die durch direkten Kontakt mit Ihnen über personenbezogene Daten verfügen, der Schweigepflicht. Des Weiteren wird die Veröffentlichung der Ergebnisse der Studie in pseudonymisierter Form erfolgen, d. h. ohne dass Ihre Daten Ihrer Person zugeordnet werden können.

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Vergütung

Für die Teilnahme an der Untersuchung erhalten Sie eine pauschale Vergütung in Höhe von 10,00 € und abhängig vom Abschneiden im Glücksspiel einen zusätzlichen Bonus von 2,00 €. Die Vergütung wird Ihnen in bar ausgezahlt. Bei Empfang der Vergütung in bar müssen Sie eine Quittung mit Angabe Ihres Namens unterschreiben.

B Written informed consent

1. Studies 1 and 4

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Ansprechpartner für eventuelle Rückfragen:

Dipl.-Psych. Natalie Ulrich

Telefon: *0931-31 83438*

Einwilligungserklärung

Julius-Maximilians-Universität Würzburg
Lehrstuhl für Psychologie I

Titel der Studie:

Biopsychologische Grundlagen der Glücksspielsucht

Ich (Name des Teilnehmers /der Teilnehmerin in Blockschrift)

_____

bin schriftlich über die Studie und den Versuchsablauf aufgeklärt worden. Ich habe alle Informationen vollständig gelesen und verstanden. Sofern ich Fragen zu dieser vorgesehenen Studie hatte, wurden sie von Herrn/Frau _____ vollständig und zu meiner Zufriedenheit beantwortet.

Mit der beschriebenen Handhabung der erhobenen Daten bin ich einverstanden. Die Aufzeichnung und Auswertung der Daten erfolgt pseudonymisiert, d. h. unter Verwendung eines persönlichen Codewortes, das ich selbst erstellt habe und das nur ich kenne. Das Blatt, auf dem ich dieses Codewort erstellt habe, befindet sich in meinem Besitz. Mir ist bekannt, dass ich mein Einverständnis zur Aufbewahrung bzw. Speicherung meiner Daten widerrufen kann, ohne dass mir daraus Nachteile entstehen. Ich bin darüber informiert worden, dass ich jederzeit eine Löschung all meiner Daten verlangen kann. Ich bin einverstanden, dass meine pseudonymisierten Daten zu Forschungszwecken weiter verwendet werden können und mindestens 10 Jahre gespeichert bleiben.

Ich bin darüber informiert, dass mein Name, meine Anschrift und meine Telefonnummer nur auf dieser Einwilligungserklärung stehen und nicht gespeichert werden und dass die Einwilligungserklärung getrennt aufbewahrt und nach Studienende (Ende 2015) vernichtet wird. Mir ist bekannt, dass eine Zuordnung der erhobenen Daten zu einer bestimmten Person auch vorher nicht möglich ist.

Sollten sich aus meiner Untersuchung im EEG und in der Testdiagnostik Hinweise auf behandlungsbedürftige Auffälligkeiten ergeben, bin ich damit einverstanden, dass mir diese mitgeteilt werden, so dass ich diese ggf. weiter abklären lassen kann. Ich wurde darüber informiert, dass die Information über auffällige Befunde u. U. mit versicherungsrechtlichen Konsequenzen verbunden sein kann.

Da alle Daten vollständig pseudonymisiert vorliegen, bin ich über das folgende Vorgehen informiert worden: Im Falle von behandlungsbedürftigen Auffälligkeiten werden sämtliche in Frage kommenden Versuchsteilnehmer angeschrieben und um Mitteilung gebeten, ob das betreffende persönliche Codewort auf sie zutrifft. Ich bin darüber informiert, dass ich mich – sofern es sich um mein eigenes Codewort handelt – bei der angegebenen Adresse melden und nähere Informationen einholen sollte.

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Wenn das angegebene Codewort nicht mein eigenes ist, kann ich dieses Schreiben ignorieren.

Ich hatte genügend Zeit für eine Entscheidung und bin bereit, an der o.g. Studie teilzunehmen. Ich weiß, dass die Teilnahme an der Studie freiwillig ist und ich die Teilnahme jederzeit ohne Angaben von Gründen beenden kann. Ich weiß, dass ich in diesem Fall Anspruch auf eine Vergütung für die bis dahin erbrachten Stunden habe.

Eine Ausfertigung der Teilnehmerinformation über die Untersuchung, über EEG-Studien und eine Ausfertigung der Einwilligungserklärung habe ich erhalten. Die Teilnehmerinformationen sind Teil dieser Einwilligungserklärung.

Ort, Datum & Unterschrift des Teilnehmers: _____

Name des Teilnehmers in Druckschrift: _____

E-Mailadresse des Teilnehmers: _____

Telefonnummer des Teilnehmers: _____

Ort, Datum & Unterschrift des Versuchsleiters: _____

Name des Versuchsleiters in Druckschrift: _____

Bei Fragen oder anderen Anliegen kann ich mich an folgende Personen wenden:

Versuchsleiter:	Projektleiter:
Dipl.-Psych. Natalie Ulrich *Lehrstuhl für Psychologie I* *Arbeitsgruppe Prof. Hewig* *Marcusstraße 9-11* *97070 Würzburg* *Tel.: 0931-31 83438* *natalie.ulrich@psychologie.uni-wuerzburg.de*	*Prof. Dr. Johannes Hewig* *Lehrstuhl für Psychologie I* *Arbeitsgruppe Prof. Hewig* *Marcusstraße 9-11* *97070 Würzburg* *Tel.: 0931-82463* *hewig@psychologie.uni-wuerzburg.de*

2. Study 2

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Prof. Dr. Johannes Hewig

Ansprechpartner für eventuelle Rückfragen:

Dipl.-Psych. Natalie Ulrich

Telefon: *0931-31 83438*

Einwilligungserklärung

Julius-Maximilians-Universität Würzburg
Lehrstuhl für Psychologie I

Titel der Studie:

Biopsychologische Grundlagen der Glücksspielsucht

Ich (Name des Teilnehmers /der Teilnehmerin in Blockschrift)

_____

bin schriftlich über die Studie und den Versuchsablauf aufgeklärt worden. Ich habe alle Informationen vollständig gelesen und verstanden. Sofern ich Fragen zu dieser vorgesehenen Studie hatte, wurden sie von Herrn/Frau _____ vollständig und zu meiner Zufriedenheit beantwortet.

Mit der beschriebenen Handhabung der erhobenen Daten bin ich einverstanden. Die Aufzeichnung und Auswertung der Daten erfolgt pseudonymisiert, d. h. unter Verwendung eines persönlichen Codewortes, das ich selbst erstellt habe und das nur ich kenne. Das Blatt, auf dem ich dieses Codewort erstellt habe, befindet sich in meinem Besitz. Mir ist bekannt, dass ich mein Einverständnis zur Aufbewahrung bzw. Speicherung meiner Daten widerrufen kann, ohne dass mir daraus Nachteile entstehen. Ich bin darüber informiert worden, dass ich jederzeit eine Löschung all meiner Daten verlangen kann. Ich bin einverstanden, dass meine pseudonymisierten Daten zu Forschungszwecken weiter verwendet werden können und mindestens 10 Jahre gespeichert bleiben.

Ich bin darüber informiert, dass mein Name, meine Anschrift und meine Telefonnummer nur auf dieser Einwilligungserklärung stehen und nicht gespeichert werden und dass die Einwilligungserklärung getrennt aufbewahrt und nach Studienende (Ende 2015) vernichtet wird. Mir ist bekannt, dass eine Zuordnung der erhobenen Daten zu einer bestimmten Person auch vorher nicht möglich ist.

Sollten sich aus meiner Untersuchung in den Hirnbildern aus der MRT-Untersuchung und in der Testdiagnostik Hinweise auf behandlungsbedürftige Auffälligkeiten ergeben, bin ich damit einverstanden, dass mir diese mitgeteilt werden, so dass ich diese ggf. weiter abklären lassen kann. Ich wurde darüber informiert, dass die Information über auffällige Befunde u. U. mit versicherungsrechtlichen Konsequenzen verbunden sein kann.

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Wenn das angegebene Codewort nicht mein eigenes ist, kann ich dieses Schreiben ignorieren.

Ich bin über Wesen, Bedeutung und Tragweite der geplanten MRT-Untersuchung aufgeklärt worden. Ich habe die Information zum Zweck der Untersuchung und die Teilnehmerinformation für MRT-Studien gelesen und verstanden. Zusätzlich bin ich ausführlich mündlich aufgeklärt und informiert worden. Ich habe weder Metallteile noch elektrische Geräte im Körper. Meine derzeitige körperliche Verfassung schließt eine Teilnahme an der MRT-Untersuchung nicht aus. Ich habe den Fragebogen für die Teilnahme an MRT-Studien wahrheitsgemäß ausgefüllt. Ich bin darauf aufmerksam gemacht worden, dass keine medizinische Diagnostik durchgeführt wird und die Hirnbilder auch nicht zur medizinischen Diagnostik verwendet werden. Für Frauen: Ich bin nicht schwanger.

Ich hatte genügend Zeit für eine Entscheidung und bin bereit, an der o.g. Studie teilzunehmen. Ich weiß, dass die Teilnahme an der Studie freiwillig ist und ich die Teilnahme jederzeit ohne Angaben von Gründen beenden kann. Ich weiß, dass ich in diesem Fall Anspruch auf eine Vergütung für die bis dahin erbrachten Stunden habe.

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3. Study 3

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97070 Würzburg

Prof. Dr. Johannes Hewig

Ansprechpartner für eventuelle Rückfragen:

Dipl.-Psych. Natalie Ulrich

Telefon: *0931-31 83438*

Einwilligungserklärung

Julius-Maximilians-Universität Würzburg
Lehrstuhl für Psychologie I

Titel der Studie:

Glücksrad Peripherphysiologie

Ich (Name des Teilnehmers /der Teilnehmerin in Blockschrift)

_____

bin schriftlich über die Studie und den Versuchsablauf aufgeklärt worden. Ich habe alle Informationen vollständig gelesen und verstanden. Sofern ich Fragen zu dieser vorgesehenen Studie hatte, wurden sie von Herrn/Frau _____ vollständig und zu meiner Zufriedenheit beantwortet.

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Sollten sich aus meiner Untersuchung im EKG und in der Testdiagnostik Hinweise auf behandlungsbedürftige Auffälligkeiten ergeben, bin ich damit einverstanden, dass mir diese mitgeteilt werden, so dass ich diese ggf. weiter abklären lassen kann. Ich wurde darüber informiert, dass die Information über auffällige Befunde u. U. mit versicherungsrechtlichen Konsequenzen verbunden sein kann.

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Codewort handelt – bei der angegebenen Adresse melden und nähere Informationen einholen sollte. Wenn das angegebene Codewort nicht mein eigenes ist, kann ich dieses Schreiben ignorieren.

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Name des Versuchsleiters in Druckschrift: _____

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C Instructions for generating personalized key

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97070 Würzburg

Prof. Dr. Johannes Hewig

Ansprechpartner für eventuelle Rückfragen:

Dipl.-Psych. Natalie Ulrich

Telefon: *0931-31 83438*

Wie erstellen Sie Ihr persönliches Codewort?

Um Ihre Daten richtig zuordnen zu können, ohne die Geheimhaltung zu verletzen, benötigen wir ein Kenn- oder Codewort. Das Codewort ist so aufgebaut, dass niemand von Ihrem Codewort auf Ihre Person rückschließen kann, auch wir nicht. Sie selbst können Ihr Codewort aber jederzeit rekonstruieren, wenn Sie danach gefragt werden und es vergessen haben sollten. Wir brauchen Ihnen nur die Regel zu verraten, nach der Sie es herstellen müssen.

Aus diesen Teilen besteht Ihr Codewort:

1. der Anzahl der Buchstaben des *Vornamens* Ihrer Mutter
2. den beiden letzten Buchstaben des Mädchennamens (*Geburtsnamens*) Ihrer Mutter
3. den beiden letzten Buchstaben des *Vornamens* Ihres Vaters
4. Ihrem eigenen Geburtstag (nur dem Tag, nicht Monat und/oder Jahr)

* Bitte schreiben Sie alle Zahlen zweistellig, d.h. wenn nötig mit führender Null.
* Bei mehreren oder zusammengesetzten Vornamen berücksichtigen Sie bitte nur den ersten.
* Wenn Sie den jeweiligen Namen nicht kennen, schreiben Sie statt der Buchstaben XX bzw. für die Zahl 00.

Beispiel (fiktiv)

Name der Mutter:	**Elke**-Hannelore Müller geb. Mayerhof**er**
Name des Vaters:	Wo**lf**-Rüdiger Müller
Ihr Geburtstag:	**09**.11.1987
Daraus ergibt sich als Codewort:	**04ERLF09**

Bitte tragen Sie jetzt in die Kästchen Ihr Codewort ein:

Die Anzahl der Buchstaben des (ersten) Vornamens Ihres Mutter:	… …
Die beiden letzten Buchstaben des Geburtsnamens Ihrer Mutter:	… …
Die beiden letzten Buchstaben des (ersten) Vornamens Ihres Vaters:	… …
Ihr eigener Geburtstag (nur der *Tag*):	… …

Wichtig: Diese Liste verbleibt bei Ihnen. Sie sollten sie niemandem zeigen!

Vorlage der Ethikkommission der Deutschen Gesellschaft für Psychologie für die Erstellung des Codewortes
16. Juli 2012

D Follow-up questionnaire study 3

Vp_Nr/Codewort: _____ Datum: _____

Nachbefragung
(Stichpunktartiges beantworten der Fragen genügt)

Haben Sie eine bestimmte Strategie im Glücksspiel benutzt, um möglichst erfolgreich zu sein? Bitte beschreiben Sie diese.

_____
_____
_____
_____
_____
_____

Haben Sie eine der beiden Farben bevorzugt gewählt? Wenn ja, welche?

_____
_____

Haben Sie schon einmal an einem ähnlichen Experiment teilgenommen?

_____
_____

Was denken Sie, sollte im aktuellen Experiment untersucht werden?

_____
_____
_____
_____
_____

Ist Ihnen aufgefallen, dass die Gewinne und Verluste unterschiedlich knapp waren, also das Rad mal in der Mitte eines Farbfeldes und mal am Rande stehen geblieben ist?

_____
_____

Publications List

Articles in peer-reviewed journals

Ulrich, N., & Hewig, J. (2014). A miss is as good as a mile? Processing of near and full outcomes in a gambling paradigm. *Psychophysiology, 51*(9), 819–823. doi:10.1111/psyp.12232

Rodrigues, J., Ulrich, N., & Hewig, J. (2015). A neural signature of fairness in altruism: A game of theta? *Social Neuroscience, 10*(2), 192–205. doi:10.1080/17470919.2014.977401

Conference Contributions & Talks

Ulrich, N., & Hewig, J. (2016, May). *Reduced FRN in problem gamblers but no differences in near outcome processing compared to controls.* Poster presented at the 42nd conference “Psychologie und Gehirn”, Berlin, Germany.

Ulrich, N. (2015, December). *Ausmaß der Glücksspielproblematik moduliert peripherphysiologische Reaktionen auf knappe Ergebnisse im Glücksspiel.* Talk given at the 16th annual meeting of the Interdisciplinary Center for Addiction Research of the University of Würzburg (IZSW), Würzburg, Germany.

Ulrich, N., Ambach, W., & Hewig, J. (2015, September). *Heart rate responses to near outcomes in gambling in relation to gambling severity.* Poster presented at the 54th annual meeting of the Society for Psychophysiological Research (SPR), Seattle, USA.

Ulrich, N., Ambach, W., & Hewig, J. (2015, June). *Heart rate responses to near outcomes in gambling.* Poster presented at the 41st conference “Psychologie und Gehirn”, Frankfurt, Germany.

Ulrich, N. (2014, December). *Verarbeitung knapper Ergebnisse bei pathologischen und problematischen Glücksspielern. Paradigma und Design.* Talk given at the 15th annual meeting of the Interdisciplinary Center for Addiction Research of the University of Würzburg (IZSW), Würzburg, Germany.

Ulrich, N., & Hewig, J. (2014, September). *Knapp daneben ist auch vorbei? FRN und P300 nach knappen Ergebnisse im Glücksspiel.* Poster presented at the 49th conference of the German Society for Psychology (DGPs), Bochum, Germany.

Ulrich, N., Weiss, M., & Hewig, J. (2014, September). *Changing the processing of near misses through an informatory intervention.* Poster presented at the 54th annual meeting of the Society for Psychophysiological Research (SPR), Atlanta, USA.

Ulrich, N., & Hewig, J. (2014, June). *Verarbeitung knapper und voller Ergebnisse im Glücksspiel: Feedback-Negativierung und Theta-Power.* Poster presented at the 40th conference "Psychologie und Gehirn", Lübeck, Germany.

Ulrich, N., & Hewig, J. (2013, October). *A miss is as good as a mile? Processing of near and full outcomes in a gambling paradigm.* Poster presented at the 53rd annual meeting of the Society for Psychophysiological Research (SPR), Florence, Italy.

Ulrich, N., & Hewig, J. (2013, May). *Knapp daneben ist auch vorbei? Verarbeitung und Bewertung knapper Ergebnisse im Glücksspiel.* Poster presented at the 39th conference“ Psychologie und Gehirn”, Würzburg, Germany.

Curriculum Vitae

Personal Details

Name	**Natalie Ulrich**
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Telephone	+49 931/31 83438 (work) +49 931/45461708 (private)
Mobile	+49 177/3301572
E-Mail	natalie.ulrich@psychologie.uni-wuerzburg.de Natalie_Ulrich@gmx.de
Date and place of birth	29.09.1988, Wertheim (Germany)

Education

since 04/2013	**Julius-Maximilians University** (Würzburg, Germany) **Graduate School of Life Sciences** Expected degree: **Dr.rer.nat** Topic: "Biopsychological Basis of Gambling Addiction" Primary Supervisor: Prof. Dr. Johannes Hewig
10/2008-12/2012	**Julius-Maximilians University** (Würzburg, Germany) **Studies in Psychology** **Diploma** (Final Grade: 1.06; 1 down to 6) Diploma thesis: “A miss is as good as a mile? Differential influences on processing of near and full outcomes in a gambling paradigm -an EEG-study-“ Primary Supervisor: Prof. Dr. Johannes Hewig
09/1999-06/2008	**Johannes Butzbach Gymnasium** (Miltenberg, Germany) **Abitur** (A-level equivalent) (Final Grade: 1.0; 1 down to 6)

Research Experience

since 09/2013	**Research fellow** at the Department of Psychology at the University of Würzburg (Lehrstuhl für Psychologie I, Prof. Hewig) **Tasks**: Design, program & conduct experiments, analyze EEG-, fMRI- and heart rate-data, prepare publications
10/2015 - 03/2016 11/2013 - 04/2014 03/2013 - 08/2013	**Research assistant** at the Department of Psychology at the University of Würzburg (Lehrstuhl für Psychologie I, Prof. Hewig) **Tasks**: Design, program & conduct experiments, analyze EEG- and heart rate-data, prepare publications
05/2011 - 02/2013	**Student research assistant** at the Department of Psychology at the University of Würzburg (Lehrstuhl für Psychologie I, Prof. Hewig) **Tasks**: Conducted experiments in the EEG laboratory, analyzed EEG-data, introduced students to conducting EEG research

08/2011 - 10/2011	**Internship** at the **Institute for Frontier Areas of Psychology and Mental Health** (Freiburg, Germany) **Tasks**: Conducted experiments including several psychophysiological measures, analyzed the data, prepared and gave talks about the concealed information test
10/2009 - 08/2010	**Student research assistant** at the Department of Psychology at the University of Würzburg (Lehrstuhl für Psychologie III, Prof. Hofmann) **Tasks:** Conducted experiments, literature search

Teaching Experience

Seminars and Practical Courses	**„Differential Psychology in Unusual Contexts“** (undergraduate level psychology, summer term 2014, winter term 2014/2015) **„Biopsychology of Personality“** (undergraduate level psychology, winter term 2013/2014) **Practical course** on conducting experiments and analyzing data (undergraduate level psychology, winter term 2013/2014, summer term 2015, winter term 2015/2016)
Thesis Supervisions	**„Manipulation des Near-Miss Effects in einem Glücksradparadigma - eine EEG-Studie“** (Bachelor's Thesis psychology, summer term 2014) **„Interindividuelle Differenzen der Feedbackverarbeitung. Zusammenhänge zwischen der dunklen Triade und früher Fehlerverarbeitung“** (Bachelor's Thesis psychology, summerterm 2015) **„Finanzielle Risikoentscheidungen in einem Ausschreibungsspiel: Eine fMRT Studie“** (Bachelor's Thesis psychology, winterterm 2015/2016) **„Alles unter Kontrolle oder schicksalsergeben? Hot-Hand und erlernte Hilflosigkeit: Wie nehmen Menschen Zufälle wahr? - Eine EEG Studie -“** (Bachelor's Thesis psychology, winterterm 2015/2016)
Other	**Student teaching assistant** at the Department of Psychology at the University of Würzburg (Lehrstuhl für Psychologie III, Prof. Krüger)

Fellowships & Awards

09/2014	Neuroscience Research Award (3000 €) of the Graduate School of Life Science at the University of Würzburg
05/2014	Travel Fellowship (1000 €) to attend the 54th annual meeting of the Society for Psychophysiological Research awarded from the Equal Opportunities Commission of the Philosophical Faculty II at the University of Würzburg
09/2013 - 08/2016	Research Scholarship, Bavarian Elite Aid Act
10/2008 - 03/2013	Fellowship from the Max-Weber-Program Bavaria

Membership in Professional Associations

since 09/2014	German Society for Psychophysiology and its Application (DGPA)
since 11/2013	German Psychological Society (DGPs) Section: Biological Psychology and Neuropsychology
since 03/2013	Society for Psychophysiological Research (SPR)
12/2014 - 12/2015	Association for Psychological Science (APS)

Social Activities & Service

since 10/2013	Member of the **"Committee to Promote Student Interests"** in the **Society for Psychophysiological Research** (SPR)
since 09/2009	**Voluntary work** with mentally ill people with the **Social Psychiatric Service Würzburg**
12/2008-02/2013	Member of the **Psychology Students' Council** at the University of Würzburg

Affidavit

I hereby confirm that my thesis entitled “Processing of Near Outcomes and Outcome Sequences in Gambling: Implications for the Biopsychological Basis of Problem Gambling” is the result of my own work. I did not receive any help or support from commercial consultants. All sources and / or materials applied are listed and specified in the thesis.

Furthermore, I confirm that this thesis has not yet been submitted as part of another examination process neither in identical nor in similar form.

_____ _____

Place, Date Signature