Conditionability and Reinforcement Sensitivity in Gambling Behaviour

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Abstract

Models of the antecedents of pathological gambling (PG) include the processes of classical and instrumental conditioning. After experiences with gambling, appetitive classical conditioning can lead to a learned relation between the gambling environment and excitement, and this excitement can subsequently become a conditioned positive reinforcer for continued gambling behaviour. The gambling environment can also become associated with negative feelings through the process of aversive conditioning, and these negative feelings can serve as conditioned punishment, ultimately leading to the avoidance of gambling. Gambling involves both positive reinforcement and punishment, and operates on a variable interval reinforcement schedule that makes gambling behaviour difficult to extinguish. Differences in the degree to which individuals acquire classical conditioning (conditionability), and differences in the degree to which individuals approach and respond to instrumental conditioning (reinforcement sensitivity) are understudied in relation to gambling behaviour. The studies reported in this thesis further investigated conditionability and reinforcement sensitivity in relation to gambling behaviour. The first aim was to investigate whether individual differences in differential aversive classical conditioning and reinforcement sensitivity were associated with risk-avoidance on a gambling task. The second aim was to also to include appetitive conditioning in order to investigate if aversive conditioning, appetitive conditioning and reinforcement sensitivity could explain differences in risk-taking during gambling. The third aim was to investigate whether PGs would show diminished conditionability by comparing them with a control group. Three studies were conducted in order to achieve these aims. The first study employed a differential aversive conditioning paradigm with skin conductance as the outcome measure. The results showed that a group of student participants did not show aversive conditioning, and furthermore that this group showed less risk-avoidance when gambling on the Iowa gambling task. No association was found between reinforcement sensitivity and risk-avoidance, therefore it seemed that aversive conditioning alone could contribute to explaining variation in risk-avoidance. The second study employed an evaluative conditioning paradigm where both appetitive and aversive evaluative

conditioning was measured. The results showed that the student participants who did not show appetitive nor aversive conditioning showed less risk-taking on a purpose built simulated slot machine designed to be more similar to commercially available gambling products than the Iowa gambling task. Furthermore, the student participants who had low scores on both self-reported reward responsiveness and punishment sensitivity (i.e. fight-flight-freeze system) also showed less risk-taking when gambling. In the third study, a group of PGs were compared to a control group on a differential aversive classical conditioning paradigm where heart rate responses comprised the outcome variable. The results showed that the PG group showed diminished aversive conditioning compared to the control group. These results combined suggest that the effects of the processes of classical and instrumental conditioning for gambling behaviour are contingent on individuals' conditionability and reinforcement sensitivity.

Abbreviations

ARAS	Ascending reticular activating system
BAS	Behavioural activation system
BIS	Behavioural inhibition system
BOLD	Blood oxygen level dependent
CPGI	Canadian Problem Gambling Index
CS	Conditioned stimulus
DSM	Diagnostic and statistical manual of mental disorders
EC	Evaluative conditioning
FFFS	Fight-flight-freeze system
fMRI	Functional magnetic resonance imaging
HRR	Heart rate response
HRV	Heart rate variability
IAPS	International Affective Picture System
IBI	Inter-beat interval
PG	Pathological gambling
PGs	Pathological gamblers
RST	Reinforcement Sensitivity Theory
SCR	Skin conductance response
SOGS-R	South Oaks Gambling Screen - Revised
US	Unconditioned stimulus
VAS	Visual analogue scale

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1. Introduction

1.1 Definitions, Prevalence and Co-morbidity

1.1.1 Definitions

The dictionary definition of the verb *to gamble* is "to do something risky that might result in loss of money or failure, hoping to get money or achieve success" (Gamble, n.d.). To take risks in the hope that it might benefit oneself or one's kin is a fundamental characteristic of human beings. As with most human characteristics, there are individual differences in risk taking that may have evolutionary roots. In times of strife, risk-taking may be beneficial, hence individual differences in risk-taking may be inherited through generations so that it is found to varying degrees in modern life (Buss, 2009). However, such risk-taking may not be adaptive in present day peaceful societies, as preference for potentially dangerous risk-taking may result in behaviours such as extreme sports, driving too fast, and excessive gambling.

As with other forms of appetitive behaviour, the distribution of gambling frequency in a population falls on a continuum which is positively skewed, implying that as gambling frequency increases, the number of people decreases (Lund & Nordlund, 2003; Orford, 2011). Individuals in the high end of the distribution may develop gambling problems, and be in need of treatment. Although several terms have been proposed in the literature to describe problems that arise from gambling, two are most prominent. The first is the term "pathological gambling" (PG). The second term is "problem gambling". Although these two terms are sometimes used interchangeably, they are theoretically distinct. The present Diagnostic and Statistical Manual for Mental Disorders, uses the term PG, and defines it as "persistent and recurrent maladaptive gambling behavior that disrupts personal, family, or vocational pursuits"(American Psychiatric Association, 2000, p. 671) . The diagnostic criteria for PG are as follows (p. 674):

A. Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following:

1. 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)

2. Needs to gamble with increasing amounts of money in order to achieve the desired excitement

3. Has repeated unsuccessful efforts to control, cut back, or stop gambling

4. Is restless when attempting to cut down or stop gambling

5. Gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g. feelings of helplessness, guilt, anxiety, depression)

6. After losing money on gambling, often returns another day to get even ("chasing" one's losses)

7. Lies to family members, therapist, or others to conceal the extent of involvement with gambling

8. Has committed illegal acts such as forgery, fraud, theft or embezzlement to finance gambling

9. Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling

10. Relies on others to provide money to relieve a desperate financial situation caused by gambling

B. The gambling behavior is not better accounted for by a Manic Episode.

In the DSM-IV-TR (American Psychiatric Association, 2000), PG is classified as an "impulse control disorder not otherwise specified", along with intermittent explosive disorder, kleptomania, pyromania, and trichotillomania. PG first appeared in the third edition of the DSM (American Psychiatric Association, 1980). In the revision of the third edition (American Psychiatric Association, 1987), the diagnostic

criteria for PG were based on the diagnostic criteria for substance dependence, and general addictive tendencies were assumed to be the underlying explanatory model for PG (Walker, 1992). It has been argued that PG is only classified as an impulse control disorder because the DSM does not have a separate section on addictions (Shaffer, 2003). Indeed, a large and growing amount of evidence indicates that PG shows commonalities with substance abuse, suggesting that PG should be classified as a behavioural addiction rather than an impulse control disorder (J. E. Grant, Brewer, & Potenza, 2006). The PG diagnosis includes concepts that are at the core of addiction; preoccupation, tolerance, withdrawal, and loss of control. It is, therefore, assumed that gamblers can become addicted and be diagnosed by the use of criteria that are similar to other addictions. Thus, the theoretical concept that PG is based on has been referred to as "the addiction-based concept of problem gambling" (Svetieva & Walker, 2008).

In contrast, the term "problem gambling" has been referred to as lying within "the problem centred concept of problem gambling" (Svetieva & Walker, 2008). "Problem gambling" has been defined as "the situation where a person's gambling activity gives rise to harm to the individual player, and/or his or her family, and may extend into the community" (Dickerson, McMillen, Hallenbone, Volberg, & Wooley, 1997, p. 106). In using the term, gambling is viewed on a continuum ranging from non-problematic gambling to over-involvement that leads to gambling problems. Gambling is viewed as an activity that is distributed on a continuum also seen in other appetitive behaviour, such as alcohol consumption. The curve shows that the majority of people conform to moderate use, whilst the number of people declines as use becomes more excessive (Orford, 2001a). As such, it places little emphasis on why some individuals gamble excessively. "Problem gambling" is rather a result of excessive gambling, and therefore it is theoretically neutral. Orford (2001b) uses the term "excessive gambling" to describe involvement in and appetite for gambling that conflicts with attempts to restrain gambling. He argues that:

> Addiction should be defined in terms of the strength of a person's attachment to the activity as indicated by such criteria as: frequency, regularity and quantity; preoccupation with and priority given to the activity; the subjective feeling of being dependent or addicted; financial, social and legal harm caused by the

> activity; and difficulty in reducing or giving up despite activity-related harm. (Orford, 2001b, p. 46).

In contrast to the term PG, the term "problem gambling" does not necessarily imply that a person is addicted to gambling. The focus is on the severity of harm suffered by the individual, and not on the mechanisms underlying why some individuals move from gambling moderately to gambling excessively (Svetieva & Walker, 2008). Therefore, the term "problem gambling" has gained popularity as it fits well within a public health model. "Problem gambling" is not only viewed as an addiction that affects individuals, but as taking place in a social context where the negative effects also concern communities (Korn, Gibbins, & Azmier, 2003).

Perhaps confusingly, the term "problem gambling" is sometimes referred to as a less severe form of PG. This is reflected in the use of measurement instruments such as the South Oaks Gambling Screen (Lesieur & Blume, 1993) and the Canadian Problem Gambling Index (Ferris & Wynne, 2001). In several studies, the cut-off point for "problem gambling" is to fulfil 3 or 4 of the DSM criteria, and is synonymous with "at risk" gamblers. It is important to bear in mind that such use of the term may ignore the differing theoretical underpinnings of "problem gambling" and PG.

In this thesis, the term PG is used because its aim is to add to the research literature that attempts to describe the mechanisms that predisposes some individuals to develop gambling problems. Its focus is therefore on individual mental health rather than on public health. By using the term PG, it is not my intention to argue that PGs are qualitatively distinct types of people, but it is rather an acknowledgment that they are individuals who are unfortunate enough to gamble so excessively that they may be in need of intervention, treatment or otherwise, to increase the quality of their lives.

1.1.2 Prevalence of PG

Prevalence estimates of gambling problems in communities are strongly influenced by definitions and measurement instruments. As already discussed, there are several different definitions. There are also over a dozen instruments that have

been proposed for measuring problem gambling and PG (Stinchfield, Govoni, & Frisch, 2007). Three of these are most widely used in prevalence studies. The first and most commonly used is the South Oaks Gambling Screen (Lesieur & Blume, 1987) and the South Oaks Gambling Screen – Revised (Lesieur & Blume, 1993). This instrument was originally designed to measure PG as defined in the DSM-III-R (American Psychiatric Association, 1987) in treatment-seeking populations. It comprises 20 statements where respondents indicate "yes" or "no" to each statement. A score of 3 or 4 is often defined as "problem gambling", and a score of 5 or above is the cut-off point for "probable pathological gambling". A recent overview of prevalence rates of problem gambling and PG for adults (> 15 years of age) across all countries where prevalence rates were available showed that the weighted mean using the SOGS was 1.2% for problem gambling (Stucki & Rihs-Middel, 2007), ranging from 0.4 % in Norway (Lund & Nordlund, 2003) to 3.6% in the USA (Welte, Barnes, Wieczorek, Tidwell, & Parker, 2001). The weighted mean for PG was 1.8%, ranging from 0.2% in Norway (Lund & Nordlund, 2003) to 3.5% in Nevada, USA (Volberg, 2002).

A second widely used instrument for measuring problem gambling and PG is the Canadian Problem Gambling Index (CPGI) (Ferris & Wynne, 2001). Within this framework, problem gambling is defined as "…gambling behaviour that creates negative consequences for the gambler, others in his or her social network, or for the community" (Ferris & Wynne, 2001, Introduction at 1.2). The CPGI contains nine items that are scored to comprise an index with several categories into which respondents may be placed; non-gamblers, non-problem gamblers, low-risk gamblers, moderate-risk gamblers, and problem gamblers. However, a scoring that is commonly used is one where 3 to 7 confirmed items comprises a problem gambling category (or moderate gambling), and 8 or 9 confirmed items comprises a PG category (or severe problem gambling). Stucki and Rihs-Middel's (2007) overview showed that the weighted mean prevalence rate using the CPGI was 2.4% for problem gambling, ranging from 1.0% in Québec, Canada (Ladouceur et al., 2004) to 4.7% in Saskatchewan, Canada (Wynne, 2002). The weighted mean prevalence rate for PG

was 0.8%, ranging from 0.5% in Canada as a whole (Marshall & Wynne, 2003) to 1.4% in New Brunswick, Canada (Focal Research Consultants, 2001).

In addition to the SOGS and the CPGI, some prevalence studies use the DSM definition and the DSM-IV criteria. As with the diagnosis of PG, individuals who indicate agreement with 5 or more criteria are categorised as PG. In addition, individuals who indicate agreement with 3 or 4 criteria are sometimes referred to as problem gamblers. In Stucki and Rihs-Middel's (2007) overview, the weighted mean prevalence rate for problem gambling using the DSM-IV criteria was 1.9%. Prevalence rates ranged from 0.45% in Norway (Götestam & Johansson, 2003) to 4.0% in Hong Kong, China (Wong & So, 2003). For PG, the weighted mean prevalence was 1.2%, ranging from 0.15% in Norway (Götestam & Johansson, 2003) to 2.1% in Singapore (Ministry of Community Development YaS, 2005).

1.1.3 Comorbid Disorders

PG is associated with several comorbid disorders, and more often than not PG is only one among several disorders that an individual may suffer from. In the largest study of comorbidity of PG to date, more than 43,000 people in the United States were interviewed (Petry, Stinson, & Grant, 2005) using NIAAA Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV (B. F. Grant, Dawson, & Hasin, 2001). After adjusting for sociodemographic and socioeconomic characteristics, the results showed that alcohol use disorder, drug use disorder and nicotine dependence were more prevalent among PGs compared to the rest of the sample (6.0, 4.4 and 6.7 times more likely to occur respectively). In addition, PGs were 4.4 times more likely to have a comorbid mood disorder, 3.9 times more likely to have a comorbid anxiety disorder and 8.3 times more likely to have a comorbid personality disorder.

These findings were replicated in a later large US survey (Kessler et al., 2008). This survey also reported that PGs showed strong "multimorbidity". PGs were 10.1 times more likely to report one disorder in addition to PG, 9.1 times more likely to report two additional disorders, and 30 times more likely to report three or more disorders. In addition, the results from the survey attempted to answer the question of which came first of PG and the comorbid disorders using age-of-onset analysis. The

findings suggested that the comorbid disorders predated the onset of PG, however as these findings were based on self-reported age of onset, using a cross-sectional design, care should be taken in interpreting these results.

Based on these findings, it is important to view PG as a disorder typically involving other disorders. The antecedents of PG may not be unique for PG, but may also be antecedents for developing other addictions as well as other psychological disorders.

1.2 Antecedents of PG

The antecedents of PG involve a complex interplay between individual and contextual factors. Here, the focus is in individual factors, therefore the contextual factors that contribute to the development and maintenance of PG are beyond the scope of this thesis. However, it can be mentioned briefly that among the contextual factors that are suggested to be important for the development of PG are increased availability and accessibility of gambling (Blaszczynski & Nower, 2002; Productivity Commision, 1999), exposure to gambling and repeated interaction with gambling games (Shaffer et al., 2004), and contact with gambling sub-cultures (Sharpe, 2002). In addition, structural properties of gambling games that make them more addictive may also be important (Griffiths, 1993; Parke & Griffiths, 2006). In this section, three individual factors are discussed that have been suggested as important for the development and maintenance of PG; learning mechanisms, cognitive distortions, and personality (e.g. Blaszczynski & Nower, 2002; Shaffer, et al., 2004; Sharpe, 2002).

1.2.1 Learning Mechanisms: Classical and Instrumental Conditioning

Classical Conditioning

Classical conditioning is a form of learning that was discovered by Ivan Pavlov through his work on the digestive system in dogs (Pavlov, 1927). It involves two different kinds of stimuli. The first is a “weak” stimulus that by itself does not cause a physiological response apart from an orienting response, and it is commonly called a

conditioned stimulus (CS). Examples of CS are the sound of a metronome, a photograph of a neutral face, or a small dot presented on a computer screen. The second form is a “strong” stimulus that does cause a physiological response, such as the presentation of food, which causes a salvatory response, or a puff of air to the eye, which causes an eye-blink response (Hugdahl, 1995). Such a stimulus is commonly referred to as an unconditioned stimulus (US). The US can be appetitive or aversive. In classical conditioning, the CS is paired with the US, and the result is that the CS comes to elicit a conditioned response (CR) that can be similar to the physiological response elicited by the US (Pavlov, 1927). An example of classical conditioning is differential conditioning. Here, two different CSs (e.g. two tones with different pitch) are used that are presented in random order, one is the CS+ and the other is the CS-. Only presentation of the CS+ is followed by the US (e.g. a loud burst of noise). After repeated presentation, the CS+ comes to yield a CR (e.g. increased electrodermal activity or increased heart rate), but the CS- does not (Hugdahl, 1995). Through this differential conditioning paradigm, the CS+ has come to provide information about the US, and predict the occurrence of the US (Rescorla & Wagner, 1972). A related form of conditioning is evaluative conditioning (EC). EC typically involves the presentation of a picture CS that is evaluated using self-report before and after it has been repeatedly presented together with a positive or negative US (e.g. a shocking picture). The change in evaluation that often occurs in such a paradigm is evidence of EC (Levey & Martin, 1987).

Neurobiological research has implicated the amygdala in the acquisition of classical conditioning (Fendt & Faselow, 1999). For example one study showed that disabling amygdala functioning blocked the acquisition of classical conditioning (Campeau, Miserendino, & Davis, 1992). The hippocampus, the anterior cingulated cortex, the insula and the medial temporal lobe have also been implicated in classical conditioning (LeDoux, 1996; Sehlmeyer et al., 2009). Appetitive and aversive conditioning seem to have different neurological basis. Appetitive conditioning has been shown to evoke neural responses in the medial orbitofrontal cortex, whilst aversive conditioning has been shown to evoke neural responses in the lateral orbitofrontal cortex (Gottfried, O'Doherty, & Doland, 2002).

Classical conditioning is viewed as an important process in the development of gambling behaviour (Blaszczynski & Nower, 2002; Orford, 2001a; Sharpe & Tarrier, 1993). It has been observed that gambling leads to increased arousal, and this has been interpreted as reflecting the subjective feeling of excitement during gambling (Anderson & Brown, 1984; Dickerson, 1972). This excitement can act as an appetitive US. The initially emotionally irrelevant gambling environment can be viewed as a CS. As in Pavlov's experiments, pairing the gambling environment with the experience of excitement caused by winning can lead to classical conditioning, and the gambling environment can come to predict the occurrence of excitement. Alternatively, the negative emotions caused by having lost money can become an aversive US. Pairing the gambling environment with such negative emotions can lead to a learned relation between the gambling environment and negative emotions, and the gambling environment comes to predict the occurrence of negative emotion. Thus, if gambling becomes associated with excitement, an increase in gambling can be predicted. Conversely, if gambling becomes associated with negative emotion, a decrease in or avoidance of gambling can be predicted.

Instrumental Conditioning

Instrumental conditioning is another basic form of learning that was studied by pioneers such as Thorndike (1911) and Skinner (1938). It is different from classical conditioning in that the animal or person makes a behavioural response and thereby produces an outcome. In a classic example, a hungry rat is placed in a small box that is bare apart from a lever on one of the walls and tray where food pellets can be delivered. Such a box is often referred to as a "Skinner box". Pressing the lever results in the delivery of a food pellet into the tray. In instrumental conditioning, the food pellet is called a positive reinforcer. The lever pressing is called an operant response. Initially, the rat will accidentally press the lever, but because this results in the delivery of a food pellet, lever pressing behaviour increases. Therefore, through this instrumental conditioning, the rat has learned to press the lever.

There are several forms of reinforcement (Domjan, 2003). As in the above example, behaviour is positively reinforced because the response produces an

appetitive stimulus. Therefore, the behavioural response is likely to increase in frequency. Punishment is another form of reinforcement. If the behavioural response produces punishment (such as an electric shock), the behavioural response is likely to decrease in frequency. Extinction of instrumental conditioning is expected if the reinforcement stops. However, how long it takes to extinguish instrumental conditioning depends on the schedule of reinforcement. There are several reinforcement schedules that determine the frequency of reinforcement (Domjan, 2003). In the above example, the positive reinforcement is presented every time after the behavioural response, and is referred to as continuous reinforcement. Another schedule of reinforcement is referred to as a variable ratio schedule. Here, reinforcement only takes place after some of the behavioural responses, and the ratio of behavioural responses to reinforcement is variable. It has been suggested that variable ratio schedules of reinforcement produce instrumental conditioning that takes longer to extinguish (Skinner, 1953).

Stimuli such as food pellets and electric shocks are called primary reinforcers because they satisfy basic drives. However, not all reinforcers are primary reinforcers. Almost any stimulus can become a reinforcer through classical conditioning (Wolfe, 1936). For example, if a Skinner box includes a light that flashes when food is presented (the primary reinforcer), the flashing light can become a conditioned reinforcer because it becomes associated with the primary reinforcer. Such conditioned reinforcers may also be more powerful compared to primary reinforcers (Wolfe, 1936). An obvious example of a conditioned reinforcer is money.

In relation to gambling, placing a wager can be viewed as the behavioural response, and winning money can be viewed as positive reinforcement. Therefore, winning during gambling can lead to increasing the frequency of gambling. Losing money can also be viewed as punishment, which can lead to a decrease in the frequency of gambling. Positive reinforcement during gambling occurs on a variable ratio schedule, as it is difficult to determine when the next win is going to take place. This variable ratio reinforcement may lead to longer periods of gambling once at the gambling venue (Dickerson, 1972). Variable ratio schedules of reinforcement may also help explain why gamblers continue to gamble despite losing, as such schedules

produce instrumental conditioning that may take a long time to extinguish (Skinner, 1953). In addition, after a classically conditioned association between gambling and excitement has been learned, excitement can be a conditioned positive reinforcer for continued gambling behaviour (Anderson & Brown, 1984). It could be the case that the excitement a gambler experiences (e.g. whilst the roulette wheel is spinning or the horses are racing) are as powerful a reinforcer for continued gambling as the excitement associated with winning money (Dickerson, 1972). However, this is contingent on the association between gambling behaviour and winning, which is learned through classical conditioning. Gambling behaviour might instead be associated with negative emotions if gambling results in losing. In this case, the negative emotions experienced in anticipation of a gambling outcome may serve as conditioned punishment, which in turn may lead to a decrease in gambling behaviour.

1.2.2 Cognitive Distortions

Cognitive distortions are regarded as important in the development and maintenance of PG (Blaszczynski & Nower, 2002; Sharpe, 2002; Sharpe & Tarrier, 1993; Toneatto, 1999). The thoughts and beliefs gamblers hold about gambling are regarded as important in explaining why some gamblers have difficulties controlling their gambling behaviour. Several cognitive distortions have been described in the literature. A useful framework for categorisation has been proposed that divides cognitive distortions into two general types. These are “Illusion of control” and “Luck/perseverance” (Steenbergh, Meyers, May, & Wehlan, 2002).

Illusion of Control

Illusion of control concerns the belief that one can control the outcome of chance events (Steenbergh, et al., 2002). PGs compared to non-problem gamblers are more likely to be confident that they have skills that can increase their chances of winning (Carroll & Huxley, 1994; Myrseth, Brunborg, & Eidem, 2010). Such magnified belief in skill is probably the result of the effort gamblers make to understand the game and to develop gambling systems (Toneatto, 1999). The fact that they sometimes win becomes evidence that they are right about their belief in skill, as

gamblers tend to focus more on winning rather than on losing. The magnified belief in skills can be upheld by interpretive biases. One such bias is the attributional bias, which refers to the tendency people have to attribute wins in terms of own disposition and place less emphasis on the role of situational factors (Heider, 1958). It may be the case that winning is attributed to own skills, whereas losses are attributed to situational factors. Another interpretive bias is called "the Gambler's fallacy". The gambler's fallacy is the belief that the likelihood of winning increases with the number of losing trials. For instance, if a coin is tossed five times and the result is heads on each toss, the Gambler's fallacy suggests that the likelihood of getting tails on the subsequent toss is greater than for getting heads (Croson & Sundali, 2005).

Luck/perseverance

Luck/perseverance can involve the overestimation of the likelihood of winning and the belief that persistence will ultimately lead to winning. Belief in luck often involves superstitious beliefs, such as talismanic superstitions, which involve the belief that carrying a certain item (such as a ring or a hat) will bring good luck (Toneatto, 1999). Superstitious beliefs can also be behavioural, such as standing on one foot whilst playing a slot machine, kissing the dice before throwing at craps, or yelling "come on, come on" whilst watching football on television. The belief in perseverance can take the form of "chasing". Chasing involves trying to win back money that is lost and is based on the belief that luck will finally turn. The logic is that there is zero chance of winning the money back if he or she stops gambling, but a non-zero chance of winning the money back if he or she persists at gambling (Toneatto, 1999). The potential problems associated with the belief in perseverance becomes evident in that PGs are more likely to indicate agreement with statements such as: "Where I get money to gamble doesn't matter because I will win and pay it back" (Steenbergh, et al., 2002, p. 145).

1.2.3 Personality

Personality is also among the factors that are regarded as important in the development of PG. Biologically based theories of personality seem to converge on

two bio-behavioural systems that are important for understanding human behaviour (Cattell, 1946; Cloninger, Svrakic, & Przybeck, 1993; Costa & McCrae, 1992; Eysenck, 1967; Gray & McNaughton, 2000; Zuckerman, 2007). One system is manifested in approach of appetitive stimuli. The other system is manifested in inhibition when faced with aversive stimuli. In evolutionary terms, both are vital for survival. Approach behaviour towards appetitive stimuli, such as food or sexual partners in order to survive and reproduce, is fundamental for any species (Darwin, 1859/2006). It is also important for survival that individuals inhibit approach behaviour when facing aversive stimuli, such as the sight of a lion when hunting game. Eysenck (1967) suggested that approach (i.e. Extraversion) is governed by the ascending reticular activation system (ARAS), a structure in the brainstem that determines the level of cortical arousal. Extroverts have an ARAS that leaves the cortex understimulated, and they are likely to approach stimulating environments and activities in order to increase cortical arousal. Conversely, the ARAS of introverts causes them to be chronically overstimulated. Hence, introverts are likely to avoid stimulating environments and activities in order to decrease arousal. Eyseck also suggested that individual differences in inhibition (i.e. neuroticism) are due to the sensitivity of the limbic system. Individuals high in neuroticism have limbic systems that are sensitive to emotional stimuli, while individuals low in neuroticism have limbic systems that are more resilient.

A conceptually similar, but neuro-biologically different, view of approach and inhibition was proposed in Gray's Reinforcement Sensitivity Theory (RST) (Gray, 1982; Gray & McNaughton, 2000). This theory suggest that approach behaviour is governed by an underlying behavioural activation system (BAS), which is mediated by the dopaminergic reward circuits in the brain (Depue & Collins, 1999). Dopaminergic reward circuits start in the ventral tegmental area of the midbrain and project to the ventral striatum (including the neucleus accumbens), amygdala and prefrontal cortex (Wise & Rompre, 1989). It has been suggested that individuals high in BAS have low basal dopaminergic activity, and that they approach novel stimuli or situations in order to increasing dopamine activity to an optimal level (Cloninger, et al., 1993).

Gray and McNaughton (2000) distinguish between fear and anxiety, both in function and neuro-biological basis. Fear is regarded as an individual's response to aversive stimuli, such as the sight of a lion approaching. The response to fear stimuli is undirected escape, which is mediated by the brain's periaqueductal grey, or directed escape, which is mediated by the medial hypothalamus. Collectively these are referred to as the fight-flight-freeze system (FFFS). Importantly, fear of some stimuli can be more easily acquired, such as snakes and spiders (Öhman, Dimberg, & Öst, 1985), but fear of more inane stimuli can also be learned through the process of aversive conditioning, such as fearing electrical outlets, dentists or examination rooms.

Anxiety is different from fear, as stated by Gray and McNaughton (2000, p. 5): "…the forms of behaviour that are appropriate when … a rat must *leave* an area where there is a cat are quite different from those that are appropriate when a rat must *enter* an area where a cat has been or might be". Anxiety is determined by the behavioural inhibition system (BIS), which is neurologically mediated by the septo-hippocampal system (Gray & McNaughton, 2000). This is supported by evidence that lesion of the septo-hippocampal system has similar anxiety alleviating effects on rat behaviour as the ingestion of anxiolytic drugs. The role of BIS is to resolve goal conflicts between signals of rewarding and punishing stimuli. An example is when an individual wishes to approach a fresh carcass lying on the ground in an area where there might be a lion. Such conflicts can be resolved by the septo-hippocampal system by increasing the negative affective valence of memories that are associated with goals. If this happens, it results in the inhibition of approach behaviour. It has been suggested that inhibition is governed by basal serotonergic activity (Cloninger, et al., 1993). Individuals high in BIS may have high basal serotonergic activity, and they avoid harmful stimuli in order not to further increase the serotonergic activity. However, it has also been argued that individuals differences in BIS does not result from differences in basal levels of serotonin, but rather, the serotonergic reactivity, as determined by the sensitivity of receptor cells (Zuckerman, 2007). Importantly, for the septo-hippocampal system to be able to increase the negative valence of memories, the association between a stimulus (such as the sight of a dentist) and negative emotion (resulting from pain),

must have taken place. Such associations are learned through the process of aversive conditioning.

According to RST, sensitivity to signals of punishment (i.e. punishment sensitivity) is a reflection of sensitivity in FFFS and BIS (see Figure 1). Sensitivity to signals of punishment (i.e. punishment sensitivity) is a reflection of BAS (Corr, 2004). Figure 1 shows the hypothesised relationships between FFFS, BIS and BAS, at the systems level, and punishment reactivity and reward reactivity at the behavioural level (Corr, 2001). Also depicted is how the personality traits Neuroticism and Extraversion (Eysenck, 1967) are hypothesised to relate to RST. FFFS and BIS have excitatory inputs on punishment reactivity, and inhibitory input on reward reactivity. Thus, a person with high FFFS and BIS is likely to be more indifferent to signals of reward and to avoid signals of punishment, and also to respond more strongly to punishment. BAS has excitatory input on reward reactivity, and inhibitory input on punishment reactivity. Thus, a person with high BAS is likely to approach signals of reward and ignore signals of punishment, and also to respond more strongly to reward.

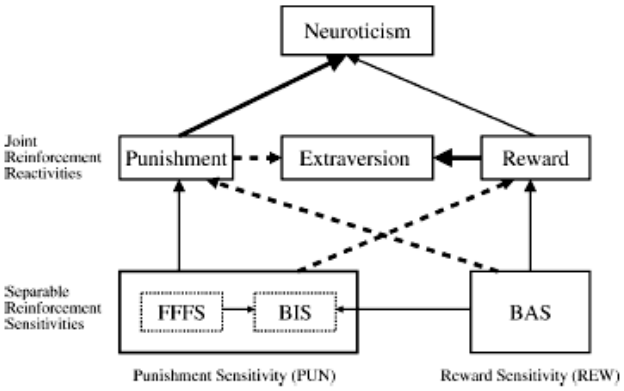

Figure 1. The hypothesised relationship between punishment sensitivity (FFFS, BIS) and reward sensitivity (BAS), and punishment and reward reactivity (Corr, 2001). Unbroken lines denote excitatory effects, and broken lines denote inhibitory effects (Reprinted with permission from Elsevier).

Since instrumental conditioning is often viewed as an important process in gambling behaviour, it may be the case that individuals who develop gambling problems have high BAS and low FFFS and BIS. Research has shown that PGs show impaired decision making on the Iowa gambling task (IGT) (Bechara, Damasio, Damasio, & Anderson, 1994). The IGT is a card playing task that measures preference for large immediate wins accompanied by large losses, resulting in monetary loss over time, versus preference for small immediate wins and small losses, resulting in monetary gain over time (the IGT is presented in detail in section 2.3.1). For instance, one study showed that PGs were more likely to show preference for large rewards and large losses than small rewards and small losses (Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2005). Neuroimaging studies have implicated the functioning of dopaminergic reward pathways and the ventromedial prefrontal cortex in PG (see Yacubian & Büchel, 2009 for a review). For instance, in a study using functional magnetic resonance imaging (fMRI), and a simple gambling task, Reuter et al. (2005) found that a PG group showed lower blood-oxygen-level dependent (BOLD) response in the right ventral striatum during the task compared to healthy controls. This indicates that individuals with PG may have impaired activity in the dopaminergic reward pathways. Thus, PGs may be more likely to approach signals of reward more often because it takes more reinforcement in order to achieve a feeling of reward. Whether deficient dopamine reward pathway functioning is a result or a cause of PG is unclear, however evidence suggests that genetic variations affecting the distribution of dopamine receptors may predispose some individuals to addiction (Yacubian & Büchel, 2009).

PGs may also be less responsive to signals of punishment. For example, one study found lower levels of the serotonin metabolite 5-HIAA, a neurotransmitter involved in impulse control in the frontal lobes, in the cerebral spinal fluid in a PG group compared to a control group (Nordin & Eklundh, 1999). Reuter et al. (2005) also found that individuals with PG showed lower BOLD responses in the ventromedial prefrontal cortex during the gambling task compared to the control group (Reuter, et al., 2005). This has also been found in previous studies using gambling cues, and a Stroop Task (Potenza, Leung, et al., 2003; Potenza, Steinberg, et al., 2003). The

ventromedial prefrontal cortex is an area of the brain which is associated with impulse control in several disorders (Yacubian & Büchel, 2009). Hence, there is evidence that suggests that reward and punishment sensitivity may be different in PGs compared to control groups.

Studies have also measured reinforcement sensitivity in relation to gambling behaviour using self-report measures. A common self-report measure of reinforcement sensitivity is the BIS/BAS scales (Carver & White, 1994), that were based on a previous version of Gray's RST (Gray, 1982). The scales contain a BIS scale and three BAS sub-scales; Reward Responsiveness (BAS RR), Drive (BAS D) and Fun Seeking (BAS FS). Studies of the relationship between BIS/BAS and performance on the IGT using these scales have found that BAS RR was positively associated with IGT performance in one study (Franken & Muris, 2005), whereas BAS RR and BAS FS were *negatively* associated with IGT performance in another study (Suhr & Tsanadis, 2007). Furthermore, one study found that individuals with low BIS and low BAS showed better IGT performance compared to individuals with low BIS and high BAS (Goudriaan, Oosterlaan, De Beurs, & van den Brink, 2006). Furthermore, in a study using a simulated slot machine, BIS and the BIS/BAS ratio were negatively associated with bet-sizes (Demaree, DeDonno, Burns, & Everhart, 2008). Based on these studies, it is difficult to draw clear conclusions about the BIS/BAS and risk-taking relationship.

The inconsistent findings in these studies may reflect that the BIS/BAS scales are based on a theory that has been updated. An attempt has been made to use a different parsing of the items in the BIS/BAS scales in order to comply with the revised RST (Gray & McNaughton, 2000; Heym, Ferguson, & Lawrence, 2008). The task of creating adequate paper and pencil self-report measures of activity in brain structures may, however, seem daunting. Psychophysiological measurement, for example fMRI, may provide more precise measures, however it is not always practical to use, and self-report measures allow for collection of more data, which is crucial in order to test models of the development of PG. More studies using the new parsing of the BIS/BAS scales should be conducted in order to investigate their predictive power.

One aim of this thesis was to further investigate the relationship between reinforcement sensitivity and gambling behaviour using self-report measures.

1.3 Integrated Models of the Development of PG

In this section, three integrated models of the development of PG are described that include the roles of learning mechanisms, cognitive distortions and personality. These models are called "The Biopsychosocial Model of Pathological Gambling" (Sharpe, 2002), "The Syndrome Model of Addiction" (Shaffer, et al., 2004), and "The Pathways Model of Problem and Pathological Gambling" (Blaszczynski & Nower, 2002).

1.3.1 The Biopsychosocial Model of Pathological Gambling

An integrative model for why some gamblers lose control and develop gambling problems is Sharpe's (2002) "Biopsychosocial Model of Pathological Gambling" (see Figure 2). According to this model, some individuals have a genetic vulnerability that affects the functioning of dopaminergic, serotonergic and noradrenergic neurotransmitter pathways in the brain. This may infer a psychological vulnerability for developing gambling problems including traits such as impulsivity. The early environment may also contribute to the forming of attitudes towards gambling, as parents and early social environments can affect an individual's own attitudes towards gambling.

Early experiences with gambling may lead to a psychological vulnerability for developing gambling problems. For instance, individuals who respond strongly to positive reinforcement but respond weakly to punishment may be particularly vulnerable for developing problems with fast paced games that give regular small payouts and small losses, such as electronic gaming machines. Individuals with poor problems-solving skills, such as thinking ahead and creating solutions may also be at greater risk for developing gambling problems. Experience with gambling may affect a perceptual filter for interpreting wins and losses. This is especially relevant for individuals who have early experiences of big wins and who expect that big wins will

occur again. Experiencing a series of many small wins may also lead to irrational beliefs such as an illusion of control over gambling outcomes, and the belief that persistence will lead to payout. Cognitive biases may develop that make individuals pay more attention to winning than to losing, resulting in an overestimation of the chances of winning later in the gambling session, or in a future gambling session.

In Sharpe's (2002) model it is assumed that gambling behaviour is developed and maintained through the processes of classical and operant conditioning. This builds on Anderson and Brown's (1984) suggestion that gambling can become associated with excitement through classical conditioning, and that excitement can serve as a conditioned positive reinforcer for continued gambling. Further, since wins during gambling are analogous to intermittent reinforcement on a variable ratio schedule, it gives a strong breeding ground for cognitive distortions.

According to Sharpe (Sharpe, 2002; Sharpe & Tarrier, 1993), the classically conditioned association between gambling and excitement, and gambling-related cognitions can both act as triggers that lead to an urge for gambling. In addition, gambling urges may be mediated by life circumstances. Here, it is differentiated between different types of gamblers, for instance horse race gamblers and electronic machine gamblers. For horse race gamblers, boredom may be a trigger for gambling because they gamble in order for the excitement of gambling to displace boredom. In electronic machine gamblers, on the other hand, life circumstances that evoke stress or dysphoric mood may trigger gambling because they gamble to escape from stress or dysphoric mood.

Triggers for gambling and the urge to gamble can be suppressed by coping strategies, e.g. controlling arousal, and challenging misconceptions about gambling. In the event that the urge to gamble leads to gambling, winning during gambling leads to further gambling related cognitions through feelings of mastery and belief in luck. Losing during gambling also leads to further gambling related distortions, for instance the belief in reciprocal altruism and the idea that a slot machine is "due" for a payout. Losing money may also lead to a host of financial and social problems, and gamblers

may gamble more in order to alleviate negative emotions associated with such problems.

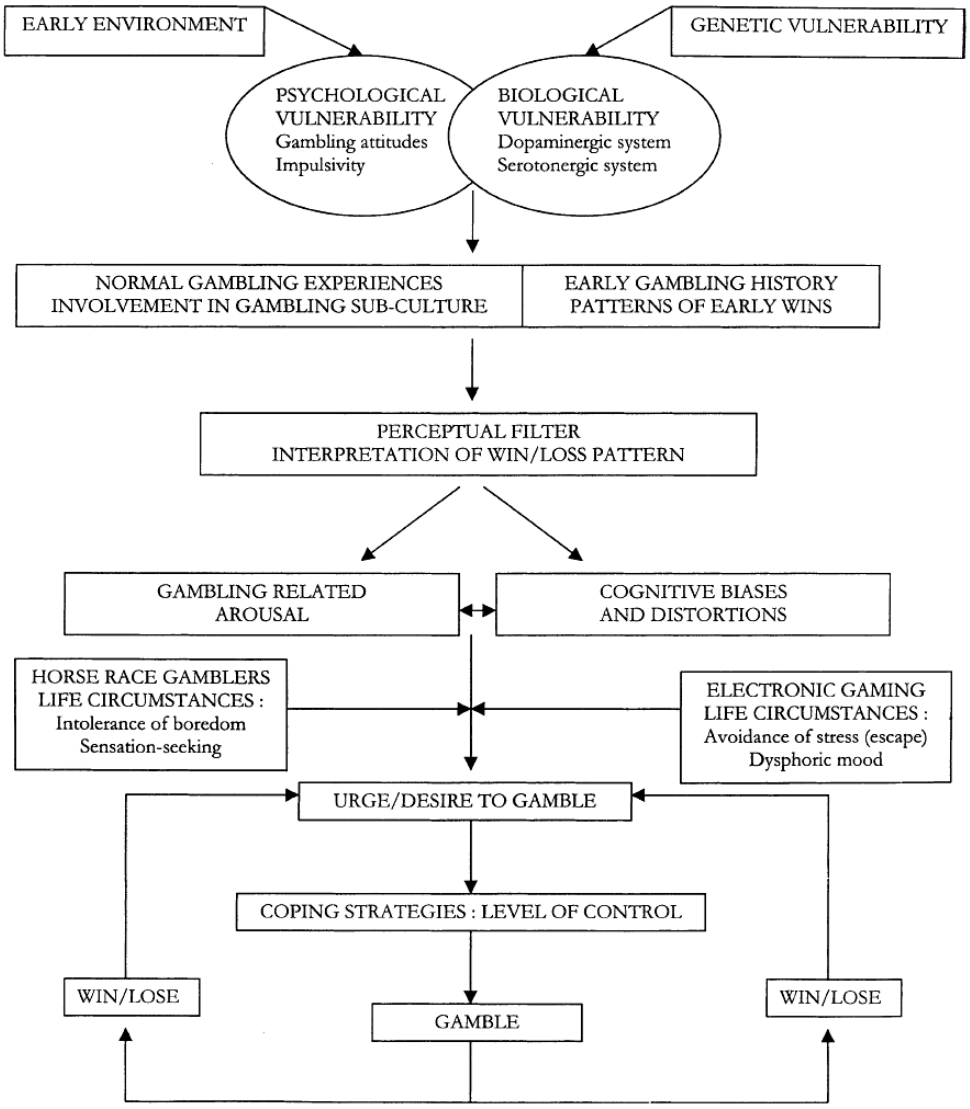

Figure 2. A Biopsychosocial Model of Pathological Gambling (reprinted with permission from Elsevier).

In summary, Sharpe's model (2002) integrates bio-psychological vulnerability, gambling experiences, arousal and perceptions of gambling as predispositional factors for experiencing gambling problems, and a cycle of triggers and urges to gamble as an explanation for why gambling problems are maintained.

1.3.2 The Syndrome Model of Addiction

An integrative model for the development of several forms of addiction is the syndrome model of addiction (Shaffer, et al., 2004). This model builds on evidence that several forms of addictions share commonalities, and may have shared antecedents, see Figure 3. The antecedents for developing addiction according to Shaffer et al. (2004) include interactions between neurobiological elements, such as genetic and neurobiological risk factors, as well as psychosocial elements such as social support and religiosity. This may influence whether the individual is likely to develop addiction. Also among the antecedents is varying degrees of exposure to substances and behaviours that can be addictive. If an at-risk individual is exposed to objects of addiction, neurobiological consequences such as experiencing reward or euphoria can take place. If repeated experiences with the object of addiction takes place, and the experience is positive, it is likely that an addiction develops if the individual is not somehow prevented for further experiences. According to Shaffer et al (2004) this course of development is common in several addictions including gambling, drinking alcohol, smoking and drug use. The expressions of different addictions are also similar. Furthermore, individuals with the syndrome are at risk for continued addictive behaviour, as well as for the development of other addictions.

Shaffer et al.'s model (2004) is similar to Sharpe's (2002) model in that it includes the interaction between underlying vulnerability and experiences with gambling. It is emphasised that the experience of gambling needs to alter mood for addiction to develop. Furthermore, gambling becomes associated with a pleasurable mood state. This association can be learned through classical conditioning. The main difference between the two models is that Sharpe (2002) places more emphasis on the role of cognitive distortions, whilst Shaffer et al. (2004) places more emphasis on the shift towards associating gambling with a pleasurable mood state. Since gambling

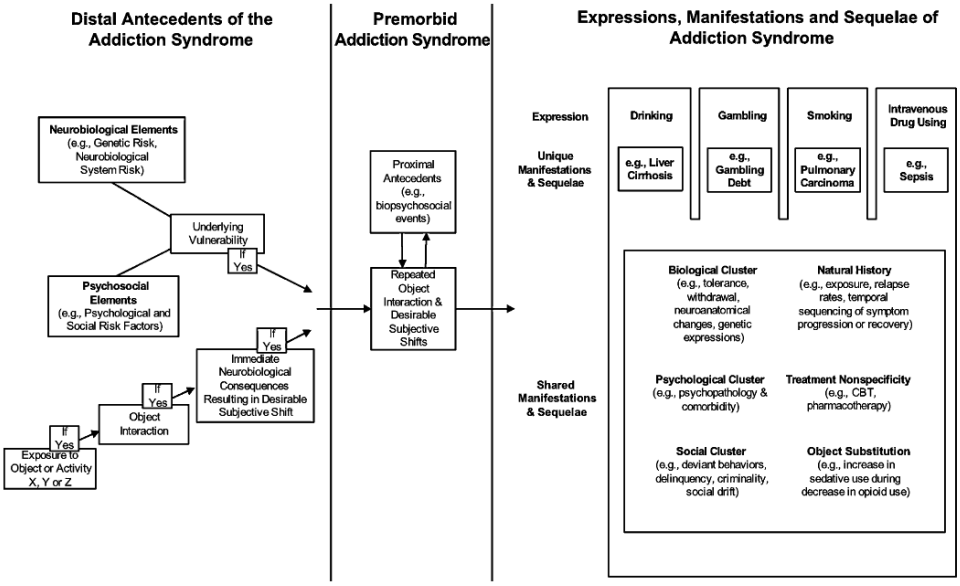

Figure 3. Model of the addiction syndrome (reprinted with permission from Routledge).

can lead to winning or losing, it may be the case that some individuals learn to associate gambling with pleasure, whilst other individuals learn to associate gambling with *dis*pleasure. It could also be the case that predispositional factors determine whether gambling becomes associated with pleasure or displeasure.

1.3.3 A Pathways Model of Problem and Pathological Gambling

An integrative model which proposes three pathways for the development of gambling problems was postulated by Blaszczynski and Nower (2002) (presented in Figure 4). A basic premise for all three pathways is that gambling opportunities must be available. Common to all three pathways are the processes of classical and instrumental conditioning described by Sharpe and Tarrier (1993). Here, gambling

becomes associated with excitement through exposure to winning during gambling, and this excitement subsequently becomes a conditioned positive reinforcer for

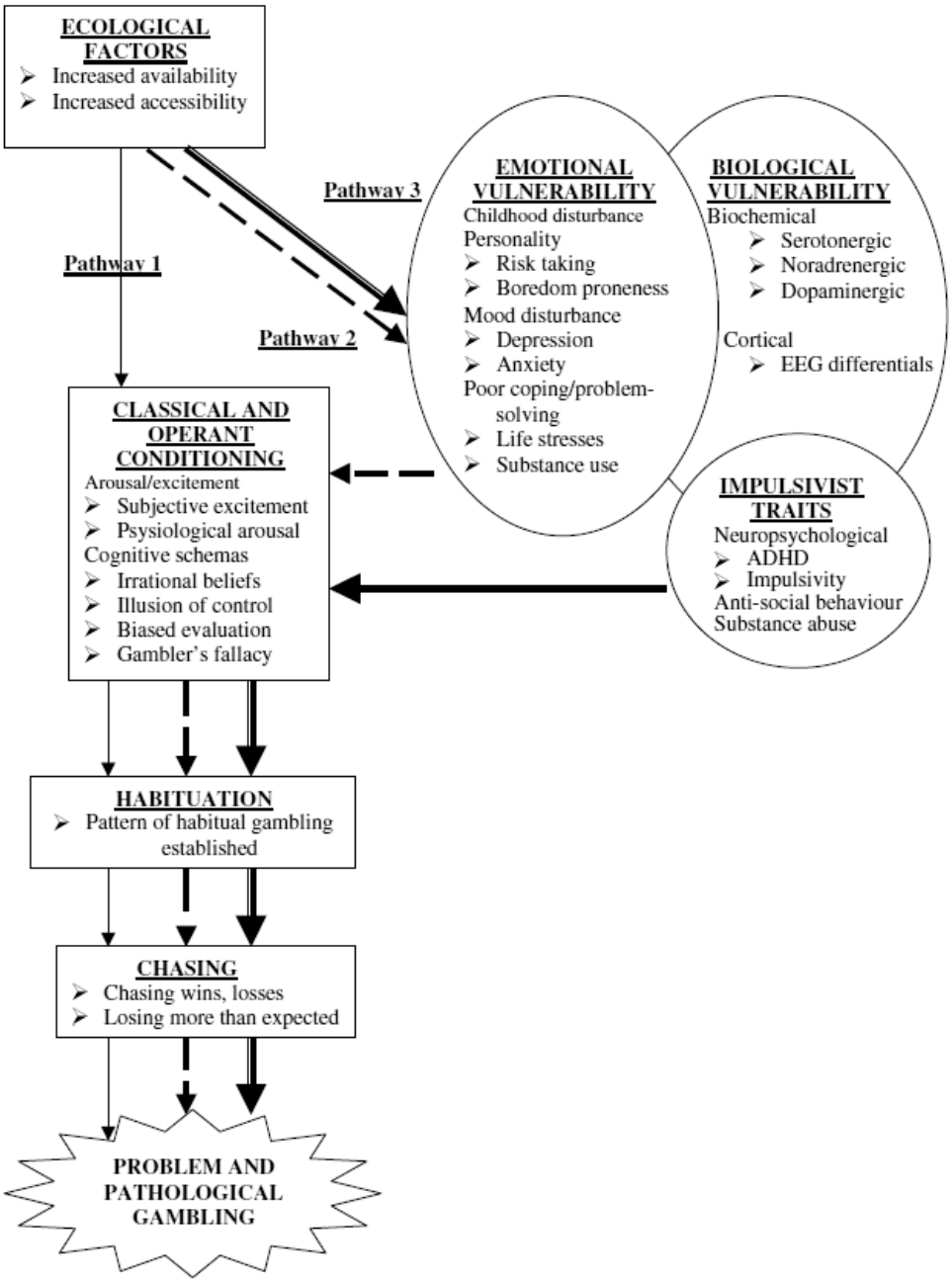

Figure 4. A pathways model of problem and pathological gambling (reprinted with permission from John Wiley and Sons).

continued gambling. Once gambling becomes a habit, cognitive distortions such as illusion of control and belief in perseverance become apparent.

According to Blaszczynski and Nower (2002) the first of the three pathways comprises behaviourally conditioned gamblers who do not have premorbid conditions that predisposes them to develop gambling problems. They mainly develop gambling problems because of the processes of classical and operant conditioning that accompany exposure to gambling and cognitive distortions surrounding probabilities of winning. As such, they are not characterised by impaired control over gambling behaviour.

The second pathway comprises emotionally vulnerable gamblers. This group is subjected to the same processes as in pathway one, but in addition they have premorbid emotional disorders, poor problem-solving skills and a history of poor family background and negative developmental background.

The third pathway comprises individuals who may have bio-behavioural dysfunction with subsequent psychological disorders and psychosocial interference that becomes apparent in their gambling behaviour. In addition to showing the same predisposition to becoming PGs as in pathway two, the pathway three gamblers also have impulse control disorders and/or antisocial personality disorder, which causes pathological gambling as well as poor psychosocial functioning.

Blaszczynski and Nower's (2002) model is similar to Sharpe (2002) and Shaffer et al.'s (2004) models in that the processes of classical and instrumental conditioning are implicated. Indeed, for the pathway one gamblers, these processes are the most important explanatory factors for the development of gambling problems. In pathways two and three, classical and instrumental conditioning are also viewed as important, but here underlying emotional and personality factors are also viewed as playing a role. The main point that sets Blaszczynski and Nower's (2002) model apart from the two other models is that individuals with gambling problems are not seen as one

homogenous group, and that the pathway to developing gambling problems can be different for individuals with different predispositional characteristics.

1.4 Relating Conditionability to PG

The processes of classical and instrumental conditioning are included in all three integrated models of the development of PG. Both Sharpe (Sharpe, 2002; Sharpe & Tarrier, 1993) and Blaszczynski and Nower (2002) mention classical and instrumental conditioning explicitly as a fundamental process pertaining to all individuals who develop PG. Shaffer et al. (2004) include the role of classical and instrumental conditioning implicitly, as they view the association between a pleasurable mood state and an object of addiction to be fundamental in the development of addiction. These models take into account that personality traits such as impulsivity can predispose some individuals for developing gambling problems, as impulsive individuals more readily seek positive reinforcement and are less affected by punishment. However, the models do not include individual differences in classical conditioning. Research has shown that there are considerable differences in the degree to which individuals show classical conditioning (Merrill, Steinmetz, Viken, & Rose, 1999). Eysenck (1959) has called an individual's ability to acquire classical conditioning "conditionability". A question that arises is whether individual differences in conditionability may predispose some individuals to be more likely to develop PG. Perhaps individuals with increased appetitive conditionability are more likely to develop gambling problems because they more readily learn to associate the gambling environment with excitement. Acquisition of this conditioned relationship is crucial for excitement to become a conditioned reinforcer for gambling behaviour.

As mentioned previously, gambling can become appetitive, as a result of successful gambling sessions. But gambling can also become aversive, as a result of unsuccessful gambling sessions. Little evidence is available that supports the hypothesis that individuals who develop gambling problems have stronger appetitive conditionability. However, a comparison between PGs and social gamblers found that on average the PG group showed greater arousal (measured by skin conductance level)

when imagining gambling scenarios compared to the group of social gamblers (Sharpe, 2004). This indicates that PGs have acquired stronger classical conditioning between gambling and excitement compared to social gamblers. However, it is unclear whether stronger conditionability was present prior to the development of gambling problems, or whether the PG group had developed a stronger association because of more exposure to gambling. Thus, more research that investigates this hypothesis is required.

Whether individuals who develop gambling problems have diminished aversive conditionability is also understudied, and only indirect evidence is available.. For instance, studies have found diminished aversive classical conditioning in individuals with alcohol-use disorder (McGlinchey-Berroth et al., 1995; McGlinchey-Berroth, Fortier, Cermak, & Disterhoft, 2002). As alcohol-use disorder is a condition often found to be co-morbid with PG (e.g. Petry, et al., 2005), it could be the case that individuals who have diminished aversive conditionability are vulnerable for developing PG as well as alcohol-use disorders. However, it is unclear whether diminished conditionability is a result of excessive alcohol intake, or whether individuals with alcohol use disorders would have shown diminished aversive conditionability before drinking excessively over time.

Research into psychopathy and criminal behaviour may also provide clues about the relationship between aversive conditionability and PG. A line of research has built on Eysenck's (1977) argument that repeated criminal behaviour can be seen in individuals who have difficulties associating the negative consequences of criminal behaviour with committing crime. According to Eysenck (1977) a person's conscience consists of classically conditioned associations. Since lacking a conscience is characteristic of psychopathy (currently referred to as antisocial personality disorder), a fundamental characteristic of psychopaths may be that they have diminished aversive conditionability. This has been supported in studies showing that psychopaths do indeed have diminished aversive conditionability (Hare, 1978). In relation to gambling, an interesting study was conducted where psychopaths were recruited from Gamblers Anonymous (Hare & Quinn, 1971). The study found that the psychopaths showed diminished physiological responding in the anticipation of presentations of a

loud burst of noise, compared to a control group. As the experiment group comprised psychopaths recruited from Gamblers Anonymous, they were problem gamblers as well as psychopaths, and this may suggest that diminished responding in the anticipation of an unpleasant event could also be true for PGs. However, it is difficult to determine the directionality of the relationship from this study, or whether the finding would also hold true for PGs who are not psychopaths.

Further indirect evidence is available in studies of persons with brain damage that disrupts the formation of physiological responses to specific events. For example, a study was conducted that investigated how patients with damage to the prefrontal cortex performed on the IGT (Bechara, et al., 1994). The results showed that the patients with prefrontal cortex damage compared to a control group were more likely to favour immediate high rewards in the face of great losses over time over low immediate rewards resulting in winning over time. The researchers referred this as "myopia for the future". Perhaps the brain damage had disrupted the ability to acquire or recall aversive classical conditioning, ultimately leading to the inability to learn to avoid making risky choices. This assumption is supported by the finding that aversive conditioning evokes neural responses in the frontal cortex (Gottfried, et al., 2002). Myopia for the future may resemble what PGs display throughout their gambling careers. They will sometimes take great risks in the hope that they win large sums of money in the immediate future rather than take smaller risks to avoid losing substantial sums over time. Indeed, this was supported in a study which showed that a PG group had poorer performance on the IGT compared to a control group (Goudriaan, et al., 2005).

In summary, there is some indirect evidence that PGs may have diminished aversive conditioning, which can help explain why they develop PG. However, as no studies have investigated this question directly, further research is warranted in order to ascertain whether this is indeed the case. One aim of this thesis is to further investigate this question.

1.5 A Note on Gambling and Risk-taking in Laboratory Studies

A venue for studying basic mechanisms in relation to gambling behaviour is the laboratory. As the laboratory can provide a great deal of experimental control, its use can provide important evidence for understanding the relationship between individual factors such as conditionability and personality, and gambling behaviour. Such evidence may in turn shed light on the dispositional factors and processes involved in the development of PG.

Laboratory studies of gambling behaviour in non-clinical samples have used several different tasks that are more or less analogous to commercial gambling games, for instance, the IGT (Bechara, et al., 1994) and a simulated slot machine (Demaree, et al., 2008). Whilst such gambling tasks may be analogous to commercial gambling games, they can perhaps also be construed as measures of risk-taking or risk-avoidance. In the risk-taking literature there has been a move from understanding risk-evaluation as a purely cognitive process (e.g. Anand, 1993) to also including the role of emotions. This move became evident with the risk-as-feelings hypothesis (Loewenstein, Weber, Hsee, & Welch, 2001). Here, behaviour is guided not as much by cognitive risk evaluation, but rather by anticipatory emotions. Anticipatory emotions may result from the process of classical conditioning. If a decision alternative becomes associated with positive emotions, it may become an appealing alternative. However, if a decision alternative becomes associated with negative emotions, it may be avoided. For individuals with a deficiency in making such associations, it has been found that risky decision making on the IGT may prevail (Bechara, et al., 1994).

It is important to bear in mind that laboratory tasks of gambling are measures of risk-taking. Gambling behaviour does not only involve the risk-taking behaviour gamblers display when gambling. An important part of gambling behaviour is the fact that gamblers chose to come back another day to gamble more, and this is not captured by using risk-taking tasks in the laboratory. Additional factors that set gambling apart from laboratory risk-taking tasks is the reallocation of wealth where

winners gain at the expense of losers, and that losses can be avoided simply by not taking part in the game (Griffiths, 1995). These factors are not accounted for in the laboratory, therefore laboratory studies of gambling are not entirely ecologically valid. Thus, gambling in the laboratory should be construed as measures of gambling behaviour when gambling, or more generally, risk-taking/risk-avoidance. Still, studies using laboratory gambling tasks can provide valuable knowledge about the relationship between for instance personality and gambling behaviour, but may not account for every aspect of gambling behaviour in real life.

1.6 Heart Rate Variability and Gambling Behaviour

Research has shown that gambling, both in the laboratory and in real gambling venues, is associated with elevated heart rate (Anderson & Brown, 1984). Also, imagining gambling scenarios has been found to cause larger increase in tonic levels of skin conductance in PGs compared to a control group (Sharpe, 2004). Tonic measures of heart rate and skin conductance are often referred to as arousal caused by increases in sympathetic nervous system activation (Dawson, Schell, & Filion, 1990). However, such a view may be rudimentary as it neglects the complexity of the autonomic nervous system. According to the "polyvagal theory" (Porges, 1995), the autonomic nervous system is made up of three stages, each resulting from demands that arouse during human evolutionary history. The first of the stages has the behavioural function of immobilisation seen in feigning death or passive avoidance of threat. This immobilisation system innervates the heart via the unmeyelinated vagus and it originates in the dorsal motor nucleus of the vagus. The second stage in the evolution of the autonomic nervous system comprised the addition of a mobilisation system, which could aid active avoidance responses to threats in the environment. This mobilisation system innervates the heart via the sympathetic adrenal system, which originates in the spinal cord. The third and most evolutionary recent stage added behavioural functions associated with social communication, inducing a calm state and the inhibition of arousal. This stage involves reduction of the heart rate and is mediated by the myelinated vagus, which originates in the nucleus ambiguus. The

Polyvagal theory proposes autonomic response strategies to challenges in the environment (Porges, 2007). When the environment is safe, the body state is regulated to save energy and promote growth. This is done by increasing myelinated vagus innervation to the cardiac pacemaker, which slows the heart down and inhibits the sympathetic nervous system's fight flight state.

Since only the myelinated vagus is capable of making fast paced adjustments to the heart rate, myelinated vagus innervation can be recorded by separating the high-frequency component of heart rate variability (the fluctuations in the inter-beat interval between successive heart beats) from the slower components (Porges, 2007). Heart rate variability (HRV) has consequently been used in recent years as an improved measure of autonomic trait and state. HRV usually refers to the high frequency band of HRV, and is also called respiratory sinus arrhythmia because it co-varies with spontaneous breathing. Low HRV indicates a state of low myelinated vagus innervation to the heart and indicates a shift from a relaxed state to a more stressed state due to sympathetic nervous system control of the heart. Low HRV is associated with increased risk of mortality from several causes (Dekker et al., 1997). HRV has also been found to be associated with prefrontal cortex activity (Thayer, Hansen, Saus, & Johnsen, 2009). Behaviourally, HRV has been positively associated with inhibition of impulses, as well as with increased adaptation to demands in the environment (Thayer & Brosschot, 2005). High resting HRV has been found to predict performance on tests of executive functioning (Hansen, Johnsen, & Thayer, 2003), as well as situational awareness (Saus et al., 2006).

In relation to gambling, studies have found that PGs show decreased activity in the prefrontal cortex when performing a simple gambling task, compared to a control group (Reuter, et al., 2005). Since prefrontal cortex activity has been associated with HRV (Thayer, et al., 2009) it can be assumed that PGs would also show lower HRV when gambling. A re-analysis of Anderson and Brown's (1984) comparison of pathological and social gamblers could provide support for this hypothesis. However, since such a comparison could be confounded by extraneous variables such as general health, smoking, anxiety and depression, lab studies of risk taking when gambling could shed light on the question of whether degree of HRV suppression is related to

risk-taking when gambling. One possibility is that individuals who show high HRV suppression perform better on a gambling task because they mobilise sufficient mental resources to perform the task sufficiently. Another possibility is that individuals who show low HRV suppression perform better on a gambling task because they do not go into fight-flight mode, which can be associated with heuristic thinking. As little research has been conducted which can be related to this question, more research investigating HRV in gambling behaviour may be warranted. One aim of this thesis is to further investigate this question.

1.7 Aims

1.7.1 Thesis Aims

Based the foregoing review of the literature, there is need for more research that investigates the relationships between conditionability and gambling behaviour, reinforcement sensitivity and gambling behaviour, and HRV and gambling behaviour. This thesis reports from three empirical studies, each with specific aims, and the studies build on each other successively. Since there is dearth of research that has studied aversive conditionability in relation to gambling, the first aim of this thesis was to investigate the relationship between aversive conditionability and risk-taking. The relationship between reinforcement sensitivity and risk-taking, and the relationship between HRV and risk-taking were also assessed to investigate whether these variables would explain variation in risk-taking alongside aversive conditioning. The next step was to also investigate whether appetitive conditioning was related to risk-taking along with aversive conditioning, and to investigate if reinforcement sensitivity also would contribute to explaining variation in risk-taking. The final step was to investigate whether aversive conditioning was diminished in PGs by comparing a group of PGs with a control group.

1.7.2 Specific Aims Study 1

The aim in Study 1 was to investigate whether individual differences in aversive conditionability as measured using a differential aversive classical conditioning

paradigm is associated with risk-avoidance on the IGT. Since this question is general in nature, a student sample was used. In addition, the study aimed at investigating associations between behavioural activation and behavioural inhibition, using the BIS/BAS scales, and risk-avoidance on the IGT. Also, the study aimed at investigating whether individual differences in baseline HRV and suppression of HRV during the IGT was associated with risk-avoidance on the IGT. To achieve these aims information about anxiety and depression, nicotine and alcohol use, gambling problems and executive functioning was collected in order to investigate whether aversive conditionability, behavioural activation and behavioural inhibition, and HRV would explain variation in risk-avoidance beyond these factors.

1.7.3 Specific Aims Study 2

The aim of Study 2 was to investigate whether appetitive and aversive conditionability, as measured using an EC paradigm, were associated with preferred bet-sizes on a simulated slot machine. Since this question is general in nature, a student sample was used. Also, study 2 aimed at investigating whether behavioural activation, behavioural inhibition and the fight-flight-freeze system, using the BIS/BAS scales with a parsing of BIS items that reflect Gray's updated reinforcement sensitivity theory, were associated with bet-sizes on the simulated slot machine.

1.7.4 Specific Aim Study 3

The aim of Study 3 was to investigate whether PGs would show diminished aversive conditioning, as measured using a differential aversive classical conditioning paradigm, by comparing a PG group with a control group. Information about age, gender, tobacco use, alcohol use, and anxiety and depression was collected in order to determine the equality of the two groups.

2. Method

The following section contains a description of the self-report measures, psychophysiological measures, and the experimental tasks and paradigms used in the three studies included in this thesis. The subsequent procedure section describes which measures were used in each study, and in which sequence.

2.1 Self-report Measures

2.1.1 Hospital Anxiety and Depression Scales (HADS)

HADS (Zigmond & Snaith, 1983) is a self-report measure of non-vegetative symptoms of anxiety and depression. Seven items are used to measure anxiety and seven items are used to measure depression. A four point scale is used in order for respondents to indicate their agreement with each item, ranging from 0 to 3. The composite score for each scale ranges from 0 to 21. The clinical cut-off point for both anxiety and depression is a score of 8 or above.

2.1.2 Tobacco Use

Smoking. Smoking habits was measured using two questions. The first asked "what are your smoking habits? Do you smoke daily, sometimes or never?" Participants indicated their response by ticking one of three boxes; daily, sometimes or never. The second question was: "If you smoke, how may cigarettes do you normally smoke per day? Indicate the number (both ready made cigarettes and rollies). There was a line underneath this question where participants could write the number of cigarettes.

Snus use. Snus is ground tobacco that comes in pouches or in loose form and is placed under the lip. Snus use was measured using two questions. The first question was "How often do you use snus?" Participants indicated their response by ticking one of three boxes; daily, sometimes or never. The second question was "If you use snus,

how many times do you use snus per day?" There was a line underneath this question where participants could write the number.

2.1.3 Fast Alcohol Screening Test (FAST)

FAST (Hodgson, Alwyn, John, Thom, & Smith, 2002) is used to assess the magnitude of alcohol use. It contains four items. The first asks how often a person drinks more than six (for women) or eight (for men) drinks. Respondents indicate the frequency on a five point scale ranging from *never* to *daily or almost daily*. The second question asks how often during the last year it has been impossible to remember what happened the previous night because of drinking. Respondents indicate the frequency on a four point scale ranging from *never* to *two to three times a week*. The third question asks how often he/she did not do things they were supposed to because of drinking. Respondents indicate the frequency by using the same response alternatives as for the previous question. The fourth question asks whether a family member, friend or physician has expressed worry about alcohol use and suggested a reduction in alcohol use. Here, participants respond by indicating *no*, *yes, on one occasion*, or *yes, on more than one occasion*. The composite score ranges from 0 to 16, and the cut-off point for hazardous drinking is a score of 3 or more.

2.1.4 South Oaks Gambling Screen – Revised (SOGS-R)

SOGS-R (Lesieur & Blume, 1993) is the most commonly used paper-and-pencil test to screen for gambling problems. It consists of eleven questions concerning gambling habits and negative consequences of gambling, and an additional nine questions concerning borrowing money from various people and institutions to finance gambling. Participants respond to each question by ticking off *yes* or *no*. Scores range from 0 to 20. A score of 0 indicates "no problem with gambling", a score of 1-4 indicates "some problems with gambling", and a score of 5 or more indicates that the individual is a "probable pathological gambler".

2.1.5 The Behavioural Inhibition/Behavioural Activation Scales (BIS/BAS Scales)

The BIS/BAS Scales (Carver & White, 1994) were developed to measure BIS and BAS according to Gray's original RST (Gray, 1982). The scale consists of twenty items. The BIS scale (seven items) is used to measure predisposition to avoid threatening or punishing stimuli, and the BAS scale (thirteen items) is used to measure predisposition to approach appetitive stimuli. Three BAS sub-scales are often used; BAS Drive (four items), BAS Fun Seeking (four items), and BAS Reward Responsiveness (five items). In order to comply with the updated Reinforcement Sensitivity Theory (Gray & McNaughton, 2000), an alternative parsing of the BIS items has been proposed (Heym, et al., 2008). The alternative parsing divides the BIS items into the fight-flight-freeze system (FFFS) (three items), which measures fear when facing aversive stimuli, and BIS-Anxiety (BIS-Anx) (four items), which measures behavioural inhibition. Participants respond to each item on a four point scale ranging from *very true for me* to *very false for me*. Scores on the scales therefore range from the number of items times 1 to the number of items times 4.

2.2 Psychophysiological Measures

2.2.1 Skin Conductance

Skin conductance is a measure of the skin's ability to conduct an electrical current. Skin conductance increases as the level of moisture in the skin increases, which is determined by activity of the eccrine sweat glands (Andreassi, 2007). The unit of measurement is micro-mho (also known as microSiemens). Skin conductance is highly correlated with bursts of sympathetic nervous system activity (Dawson, et al., 1990), and may therefore be used to measure phasic changes in sympathetic nervous system activity. The change in skin conductance following a stimulus presentation, such as in a classical conditioning paradigm, is referred to as the skin conductance response (SCR) (Andreassi, 2007).

To measure skin conductance, two unshielded 8 mm Ag-AgCl electrodes filled with isotonic gel were used. These were attached using an adhesive ring to the middle phalanx of the index and middle fingers on the non-dominant hand. The electrodes were plugged into a Biopac MP35 system, and recorded using BSL PRO software (BIOPAC Systems Inc., 2007). The skin conductance signals were analysed using Mindware EDA (Mindware Technologies Ltd, 2007). A SCR was determined as the largest change in skin conductance level during ten seconds following stimulus onset with a one second delay. The smallest response recorded was a .04 microSiemens change in skin conductance level. To adjust for individual differences in maximum skin conductance level, Lykken and Venables' (1971) range correction was used.

2.2.2 Heart Rate Variability (HRV)

HRV is a tonic measure of the variability in duration of the inter-beat-intervals between successive heart beats. At the level of the heart it reflects parasympathetic nervous system activity (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). To record cardiac activity, three disposable 10 mm electrodes (EL503, Biopac Systemts, Inc.) were used. Two of the electrodes were placed 15 cm below the left and right nipple, and the third was placed 20 cm below the left armpit. A Biopac MP-35 unit and Biopac Student Lab software (BIOPAC Systems Inc., 2007) was used to record cardiac activity at a rate of 1000 Hz. The power in ms^2 of the high frequency band (0.15-0.4 Hz) component of HRV was determined using autoregressive power spectrum analysis in Kubios (Biosignal Analysis and Medical Imaging Group, 2007). All values were transformed to their natural logarithms in order to obtain a normal distribution of scores.

2.2.3 Heart Rate Responses (HRR)

Heart rate responses are measures of phasic changes in heart rate. In response to a stimulus or event, two different components of HRR have been identified (Graham & Clifton, 1966; Thayer, Friedman, Borkovec, Johnsen, & Molina, 2000). One component is a deceleration of heart rate which occurs rapidly following stimulus onset. This deceleration is associated with orientation and increased attention. The

second component is an acceleration of heart rate often occurring after the orienting response. This acceleration is associated with a defence reflex (Graham & Clifton, 1966).

Cardiac activity was measured by the same means as heart rate variability, see section 2.2.2. To analyse HRR, Mindware HRV version 3.0.1 (Mindware Technologies Ltd., 2008) was used. Baseline inter-beat-intervals (IBIs) were calculated as the average of the three IBIs occurring before stimulus onset. Five consecutive IBISs (measured in milliseconds) following stimulus onset comprised the window of analysis. These were subtracted from the baseline to calculate delta scores (ΔIBI).

2.3 Experimental Tasks and Paradigms

2.3.1 The Iowa Gambling Task (IGT)

The IGT (Bechara, et al., 1994) is a card playing task where a participant is presented with four decks of cards (labelled A, B, C and D) on a computer screen. The participant is instructed to pick a total of 100 cards from the four decks by pressing corresponding keys on the computer keyboard. With each selected card the participant gains a set amount of money, $100 on decks A and B, and $50 on decks C and D. On deck A and B, the participant also loses large amounts of money, leading to monetary loss over time, whilst on decks C and D, the losses are smaller, and can lead to monetary gain over time. Therefore, the IGT measures preference for large immediate wins accompanied by large losses, resulting in monetary loss over time, versus preference for small immediate wins and small losses resulting in monetary gain over time. Selecting from the advantageous C and D decks rather than from the disadvantageous A and B decks can be regarded as indicating learned risk-avoidance. The IGT was programmed using E-Prime (Psychology Software Tools Inc., 2005) according to Bechara et al.'s (1994) specifications.

2.3.2 The Wisconsin Card Sorting Test (WCST)

The WCST (Berg, 1948; D. A. Grant & Berg, 1948) was developed as a technique for measuring flexibility in thinking. The standard computer version distributed by PAR Inc. was used (Heaton, PAR Staff, & Goldin, 2003). The test starts with the presentation of four cards on a computer screen. Each card is printed with a symbol; one red triangle, two green squares, three yellow crosses, or four blue circles. The participants' task is to sort the cards according to one of three rules. The rule can be the colour of the symbol on the cards, the shape of the symbol on the cards, or the number of symbols on the cards. The participant needs to figure out what the rule is by trial and error, and sort cards according to the rule. After a number of correct attempts at sorting, the rule will change, and the participants need to figure out what the new rule is. The WCST provides a number of performance indicators. The most used indicators are the number of errors made, and the number of perseverative errors (sorting cards according to the "old" rule). The perseverative errors are used as an index of flexibility in thinking, or executive functioning.

2.3.3 Aversive Conditioning

Two different aversive conditioning paradigms were used, the first in Study 1 and the other in Study 3. In both paradigms auditive stimuli were used and presented in mono to both ears via padded head-phones. Auditive stimuli were obtained using audio generator software. Two tones were used as CS, a 1500 Hz tone and a 850 Hz tone. These were played back at 65 dB. The US was white noise played back at 100 dB.

Aversive Conditioning Paradigm in Study 1

In the paradigm used in Study 1, the duration of the CS was 1350 ms, and the duration of the US was 1000 ms. For one half of the participants the CS+ was the 1500 Hz, and the 850 Hz tone was the CS-. For the other half of the participants, the 1500 Hz tone was the CS-, and the 850 Hz tone was the CS+.

The paradigm consisted of two phases. The first was an acquisition phase. Here, the CS+ and the CS- were presented pseudo-randomly eight times each with a 12 s

inter-trial interval. The CS+ was immediately followed by the US, whereas the CS- was never followed by the US. The second phase of the paradigm was an extinction phase. This was identical to the acquisition phase, but the US did not follow the CS+.

Aversive Conditioning Paradigm in Study 3

In the paradigm used in Study 3, the duration of the CS was 8000 ms, and the duration of the US was 1000 ms. For one half of the participants, the CS+ consisted of the 1500 Hz tone, and the 850 Hz tone comprised the CS-. For the other half of the participants, the 1500 Hz tone was the CS-, and the 850 Hz tone was the CS+.

The paradigm consisted of two phases. The first was the habituation phase. Here, the to-be CS+ and the to-be CS- were presented pseudo-randomly three times each with an inter-trial interval alternating randomly between 12 s and 18 s (with the rule that one should not be presented more than twice in succession).

The second phase of the paradigm was the acquisition phase. The CS+ and CS- were presented ten times each in a pseudo-random order. As in the habituation phase, the inter-trial interval alternated randomly between 12 s and 18 s. The CS+ was always followed by the US, and the CS- was never followed by the US.

2.3.4 Evaluative Conditioning

The evaluative conditioning (EC) paradigm comprised a version of the picture-picture paradigm (Levey & Martin, 1987). The picture stimuli used were eighteen pictures from the International Affective Picture System (IAPS) (Lang, Bradley, & Cuthbert, 2005). The IAPS includes a pre-rating of each picture on valence and arousal. Twelve of the pictures used were pre-rated as neutral in both valence and arousal. These were pictures of the shadow of a person (IAPS # 2880), an iron (7030), a dustpan, (7040), a rolling pin (7000), a spoon (7004), a bowl (7006), a lamp (7175), a bus (7140), an umbrella (7150), a fan (7020), wooden buckets (7041) and a storage room (7700). Three pictures were used that were pre-rated as positive in valence and high in arousal. These were pictures of a man and woman making love (4695), downhill skiers (8190), and children on a roller-coaster (8499). Three pictures were used that were pre-rated as negative in valence and high in arousal. These were

pictures of a crying baby (2800), a man pointing a gun at another man (3530), and a man carrying a burnt and lifeless child (9410).

The EC paradigm was programmed on a computer using E-prime software (Psychology Software Tools Inc., 2005), and run on a desktop computer with an 18 inch computer screen. The EC paradigm consisted of four phases. The first phase was the pre-evaluation phase where all eighteen pictures were presented in random order. Participants were instructed to look at each picture. Each picture was presented on the screen for 2 s, and the inter-trial-interval was 2 s.

The second phase was the pre-conditioning phase. Participants were instructed to rate each picture on a 100-point visual analogue scale (VAS), where the left hand extreme was labelled ”dislike” and the right hand extreme was labelled ”like”. All eighteen pictures were presented on the screen in random order, each presentation lasting 2 s.

The third phase was the conditioning phase. Here, the participants were instructed to look at the pictures on the screen. Nine CS-US picture pairs were assembled for this phase. Three pairs were neutral-positive, three pairs were neutral-negative, and three pairs were neutral-neutral. The CS was presented on the screen for 1 s, followed by a 100 ms inter-stimulus-interval, and subsequently the US was presented for 1 s. Each of the picture pairs were presented five times in random order.

The fourth phase was the post-conditioning phase. This phase was identical to the pre-conditioning phase, and participants rated all the pictures again.

2.3.5 The Hordaland Slot Machine

The Hordaland Slot Machine was created (by this author) in order to measure preferred level of risk-taking when gambling. It was programmed using E-Prime software (Psychology Software Tools Inc., 2005). The Hordaland Slot Machine comprises a screen showing a picture of a slot machine as shown in Figure 5. A display in the top left corner shows the bank, the amount that is left to play with. The amount is given in Norwegian kroner (NOK). The exchange rate is roughly 100 NOK = € 13 = $ 19.

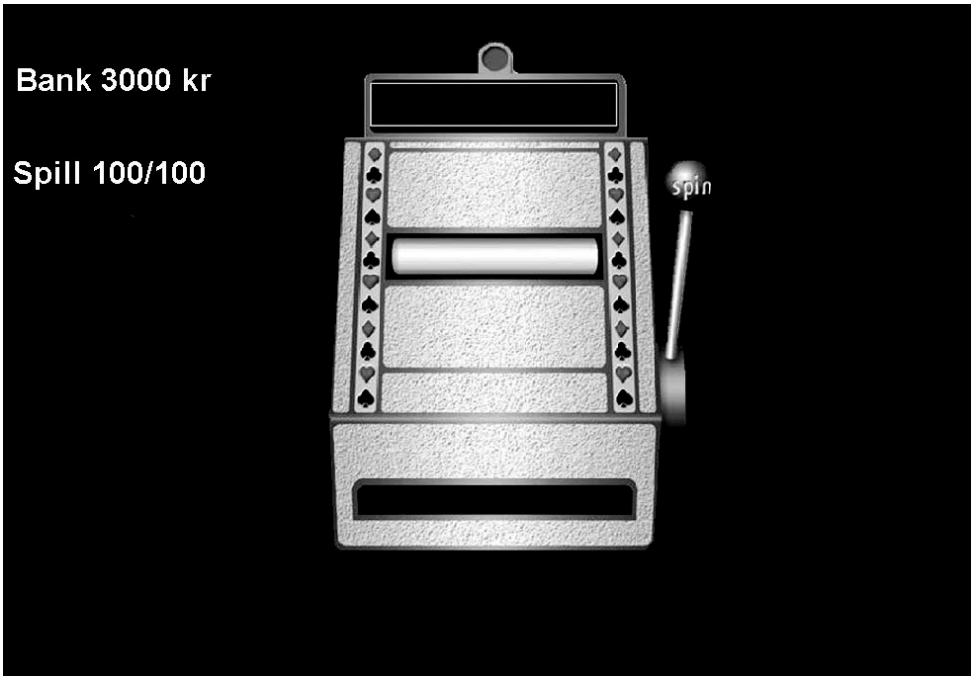

Figure 5. The Hordaland Slot Machine

The number of trials left is displayed below the bank. In the top centre of the slot machine is a display where the amount won on the current trial is presented. The game is played by pressing one of the number keys (1, 2 … 9) on a computer keyboard. When a key is pressed, a sound is played back which resembles the sound of the handle being pulled and released. The numbers on the keyboard determines the bet-size for each spin times 10. For example, pressing the "1" key equals a bet-size of 10 NOK, and pressing the "9" key equals a bet-size of 90 NOK.

Before gambling, participants are informed that the game resembles a slot machine, that they play 100 spins, that they start with 3000 NOK, that they can keep 10% of the amount left in the bank after the 100 spins, and that they decide the bet-size for each spin by pressing the number keys.

For each spin, three different outcomes can occur. The first is that the win is nil, and the wager is deducted from the bank. This has a 70% chance of occurring. The second possible outcome is a small win accompanied by a sound resembling a fast siren. The small win equals the bet-size times 2.25, and has a 20% chance of occurring. The small win is added to the bank, and the wager is deducted from the bank. The third possible outcome is a big win accompanied by a sound resembling the drop on thin metal of several handfuls of coins. The big win equals the bet-size times 4.5 and has a 10% chance or occurring. The big win is added to the bank, and the wager is deducted from the bank. The three outcomes are selected randomly by the computer software.

2.4 Samples and Procedures

2.4.1 Sample and Procedure Study 1

Sample

The sample in Study 1 consisted of 61 undergraduate students recruited during lectures for the subject "Introduction to Psychology" at the University of Bergen. A list with spaces for names and telephone numbers was passed around the lecture theatre. Those who wrote their names on the list were contacted via telephone to set up times for participation. The participants in the study were 31 female and 30 male students with an age range of 18 to 28 years. Three participants were excluded from the study, one due to abnormal cardiac activity, and two due to equipment failure. Thus, the final participants numbered 58 (29 females, 29 males).

Procedure

Participants were instructed not to consume caffeine and nicotine two hours prior to appearing at the testing facility. The testing facility was a sound attenuated and temperature controlled laboratory room where the temperature was kept at 22 degrees Celsius. It contained a comfortable armchair, which was separated from the laboratory equipment by an office cubicle wall. Upon entry, each participant read and signed an

informed consent form explaining the purpose of the study, the procedure, and that the data collected would be treated confidentially. Thereafter, the participant was fitted with electrodes and assigned to the armchair.

The procedure was as follows:

1. Five minutes recording of baseline heart rate.
2. Aversive conditioning paradigm, see section 2.3.3.
3. The IGT, see section 2.3.1.
4. The WCST, see section 2.3.2.
5. Five minutes recording of recovery heart rate.
6. Completing a questionnaire consisting of the HADS (section 2.1.1), Tobacco use (section 2.1.2), FAST (section 2.1.3), SOGS-R (section 2.1.4) and the BIS/BAS scales (section 2.1.5).

Before leaving the laboratory, all participants were paid 100 NOK (about € 13). The study was approved by the Regional Committees for Medical and Health Research Ethics in West-Norway, and by the Norwegian Social Science Data Services.

2.4.2 Sample and Procedure Study 2

Sample

The sample in Study 2 consisted of 100 undergraduate students recruited in the same way as the sample in Study 1. The participants were 51 women and 49 men with a mean age of 21.01 years (SD = 2.49). None of the participants were probable pathological gamblers, as none scored 5 or more on the SOGS-R.

Procedure

Data were collected in a multiple-testing laboratory at the University of Bergen. The laboratory contained five sound attenuated chambers. Each chamber contained a desk, a chair, and a desktop computer. Participants arrived in groups of up to five. Information about the study and assurance that the data collected would remain

confidential was given, and an informed consent sheet was signed prior to assignment to the chambers.

The procedure was as follows:

1. Completion of a questionnaire comprising the SOGS-R (section 2.1.4) and the BIS/BAS scales with the parsing of BIS items suggested by (Heym, et al., 2008) (section 2.1.5).
2. The EC paradigm (see section 2.3.4).
3. The Hordaland Slot Machine (see section 2.3.5).

Before leaving the laboratory, all participants were paid 10% of the amount that was left in the bank of the Hordaland Slot Machine, ranging from 168 NOK to 368 NOK. The study was approved by the Regional Committee for Medical and Health Research Ethics in West-Norway

2.4.3 Sample and Procedure Study 3

Sample

The sample in Study 3 consisted of two groups. One was a group of 20 PGs (17 male, 3 female). Mean age was 40.4 years (range 24-76, *SD* = 14.47). The PG group was recruited through advertisements in local newspapers, and from three treatment facilities, one in Western Norway and two in Eastern Norway. A control group (*n* = 20 [17 male, 3 female], mean age 39.3 years [range 22-71, *SD* = 14.51]) was recruited via advertisements in local newspapers, and among university students and staff. The control group was recruited with the aim to match each individual in the PG group on gender, and on age plus/minus 5 years. The average SOGS-R score for the PG group was 12.50 (range = 7 – 17, SD = 2.67). The average SOGS-R score for the control group was 0.60 (range = 0 – 4, SD = 1.05).

Procedure

The data collection took place in three locations. Thirteen of the individuals in the PG group and all individuals in the control group were tested in the same

laboratory room as was used in Study 1. Seven other individuals in the PG group were tested in offices located at one of two treatment facilities in Eastern Norway. These were the Borgestad Clinic divisions in Drammen and Skien. The offices were quiet and heated, however the exact room temperature was not recorded.

All participants read an information sheet explaining the purpose of the study and assurance that all data would be treated in a confidential manner. All participants signed an informed consent form. Before testing, participants were fitted with ECG electrodes and headphones, and were assigned to a comfortable arm-chair. The procedure was as follows:

1. A five minute resting period
2. A differential aversive classical conditioning paradigm, see section 2.3.3
3. Completion of a questionnaire consisting of Tobacco use (see section 2.1.2), FAST (see section 2.1.3), HADS (see section 2.1.1), and SOGS-R (see section 2.1.4)

Finally, participants were debriefed and electrodes were removed. All participants received a payment of 200 NOK (about €25) for their participation. The procedure was approved by the Regional Committee for Medical and Health Research Ethics in Western-Norway.

2.5 Statistics

2.5.1 Statistical Procedures Study 1

To determine aversive conditioning, for each participant the average magnitude of skin conductance responses following stimulus onset of CS- were subtracted from the average magnitude of skin conductance responses following stimulus onset of CS+. Inspection of the score distribution showed that 26 participants had a score of zero. Therefore scores were recoded into a dichotomous variable where these 26 participants comprised a group named aversive conditioning minus (AC-). The 32 participants who did show aversive conditioning according to the operational

definition comprised a group named aversive conditioning plus (AC+). The risk-avoidance variable comprised the number of cards each participant chose from the advantageous decks on the IGT.

Pearson's correlation coefficients were calculated for variables at interval level, whereas point-biserial correlation coefficients were calculated for relationships between variables measured at interval level and variables measured at dichotomous variables. Phi coefficients were calculated for relationships between dichotomous variables. Backward stepwise multiple regression analysis was used to investigate which variables could significantly predict levels of risk-avoidance. In addition, 2 x 5 repeated measures ANOVA was used to further investigate differences between the AC+ and AC- groups on risk-avoidance. Here, the number of cards chosen from advantageous decks on the IGT were divided into five stages each consisting of 20 consecutive trials. Group (AC+ vs. AC-) was a between group factor, Stage (5 blocks of 20 cards) was a within subjects factor, and the number of cards chosen from the advantageous decks was the dependent variable. Greenhouse-Geisser correction was used as a statistical correction as the Maulchy's test showed that the assumption of sphericity was violated.

2.5.2 Statistical Procedures Study 2

A manipulation check was performed in order to test whether the positive, negative and neutral pictures used as US in the EC paradigm were rated differently from one another. Repeated measures ANOVA was conducted where US type (positive US vs. negative US vs. neutral US) was the independent factor, and valence measured on the VAS comprised the dependent variable. The effect of EC with negative and positive US were tested separately using repeated measures ANOVA. In both analyses, Time (pre-conditioning vs. post-conditioning) was the independent factor and valence rating on the VAS was the dependent variable. Since a significant proportion of participants had change scores on the VAS that were smaller than one point for positive US or negative US, and because the scores lacked a normal distribution, dichotomous variables were constructed for use in the analyses (0 = did not show conditioning, 1 = did show conditioning).

The statistical methods used in the analyses were Pearson's product-moment correlations between variables at interval level, and point-biserial correlations for the relationship between dichotomous variables and variables at interval level. The interaction between positive EC and negative EC on average bet-size was tested using a 2 (positive EC: yes vs. no) by 2 (negative EC: yes vs. no) ANOVA with average bet-size as the dependent variable. Follow-up pairwise comparisons (*t*-test) were used to further explore the interaction effect.

The FFFS x BAS-RR interaction effect was tested using multiple regression where FFFS, BAS-RR and the FFFS x BAS-RR interaction term were entered as predictor variables, and where average bet-size was the dependent variable. The interaction effect was plotted graphically according to suggestions from Cohen, Cohen, West and Aiken (2003).

2.5.3 Statistical Procedures Study 3

Differences in self reported co-morbidity between the PG and the control group in Study 3 were analysed using Pearson's Chi-square and independent samples *t*-tests.

To test whether there were differences between the two groups in terms of habituation, the HRR following stimuli presentation for the three CS+ were collapsed into one block. The same was done for the three CS- presentations. A 2 (Group: PG vs. control) x 2 (Stimuli: CS+ vs. CS-) x 5 (IBIs) ANOVA was conducted where Group was a between-subjects factor, Stimuli and IBIs were within-subjects factors, and where ΔIBI comprised the dependent variable. Subsequently, pairwise comparisons (*t*-tests) of Stimuli on IBIs were calculated for each group to investigate differences between the two groups.

To test whether there were differences between the two groups concerning acquisition of aversive conditioning, HRR were collapsed into three blocks. The HRR on the first trial were left out because this was the first CS+-US pairing, and therefore no conditioning could have taken place. The subsequent nine trials were collapsed into three blocks. The first block comprised trials 2-4, the second block comprised trials 5-7, and the third block comprised trials 8-10. A 2 (Group: PG vs. control) x 2 (Stimuli:

CS+ vs. CS-) x 3 (Blocks: Block 1 vs. Block 2 vs. Block 3) x 5 (IBIs) ANOVA was conducted where Group was a between-subjects factor, Stimuli, Blocks and IBIs were within-subjects factors, and ΔIBI comprised the dependent variable. The ANOVA was followed up using pairwise comparisons (*t*-tests). Pairwise comparisons of Stimuli (CS+ vs. CS-) were made on each of the IBIs. This was done for each group separately.

For the ANOVA conducted to test differences in habituation and for the ANOVA conducted to test differences in acquisition of aversive conditioning, the assumption of sphericity was violated. Therefore, Greeenhouse-Geiser corretions were made to the degrees of freedom.

A-priori power analysis was computed. Expected effect size was set to $d = 0.08$, alpha was set to 0.05 (one-tailed) and power was set to 0.80. This power analysis showed that 21 participants were needed in each group to yield a power of 0.80 given the stated parameters.

3. Results

3.1 Results Study 1

Bivariate correlations showed that aversive conditioning was significantly and positively correlated with risk-avoidance, $r = .30$, $p <. 05$. Anxiety, depression, nicotine use, alcohol use, SOGS, WCST, resting HRV, HRV suppression and the BIS/BAS scales were not significantly correlated with risk-avoidance.

Multiple regression showed that aversive conditioning was the only variable that contributed significantly to explaining variance in risk-avoidance, $\beta = .30$, $p = .023$. The variance explained (R^2) was 9%.

Repeated measures ANOVA showed a significant main effect for the aversive conditioning factor, $F(1, 56) = 5.30$, $p = .025$. The AC- group ($M = 45.69$, $SD = 11.92$) chose fewer cards from advantageous decks on the IGT compared to the AC+ group ($M = 53.78$, $SD = 13.84$). A significant effect of Stage was also found, where the participants overall chose fewer cards from the advantageous decks on the first stage compared to the fourth and fifth stage, showing a gradual increase in card selection from the advantageous decks over time. The difference between the AC- and AC+ groups were greatest on the third, or middle, stage.

In summary, study 1 showed a positive association between aversive conditioning and risk-avoidance.

3.2 Results Study 2

Bivariate correlations showed that FFFS-Fear ($r = .23$, $p < .05$), BAS-Total ($r = .22$, $p = < .05$), and BAS-RR ($r = .24$, $p = < .05$) were positively correlated with average bet-size.

The ANOVA showed that there were no main effects of positive EC or negative EC on average bet-size. The positive EC by negative EC interaction effect was, however, significant, $F(1, 79) = 8.40$, $p < .01$. Follow-up pairwise comparisons

showed that the group of participants that did not show positive EC or negative EC had lower average bet-size compared to the three other groups (the group that showed negative EC but not positive EC, the group that showed positive EC but not negative EC, and the group that showed both negative and positive EC).

Multiple regression analysis showed that FFFS, BAS-RR, and the FFFS x BAS-RR interaction effect were significant predictor variables for average bet-size. The participants who had low scores on both BAS-RR and FFFS had lower average bet-size compared to the other participants. The model explained 14% of the variance in average bet-size.

3.3 Results Study 3

Bivariate correlations and chi-square analysis showed that the PG group, compared to the control group, comprised more daily smokers, and had higher average scores on symptoms of anxiety and depression. However, none of the participants had anxiety or depression scores within the clinical range. The PG group and the control group did not differ on snus use or alcohol use.

The ANOVA comparing the PG group with the control group on habituation showed that the two groups did not differ in habituation.

The ANOVA comparing the PG group with the control group on aversive conditioning showed that the conditioning phase had different effects on the PG group and the control group. The PG group did not show conditioning whereas the control group did show conditioning. This was corroborated by a significant interaction effect for Group x Stimuli x Blocks x IBIs, F (4.42, 167.97) = 3.37, p = 009. Follow-up pairwise comparisons showed that for the PG group there were no differences in HRR to the CS+ and CS-, apart from on IBI 5 in Block 3 (p = .038). However, for the control group significant differences between CS+ and CS- were found on IBI1 in Block 1 (p = .005). Significant differences between CS+ and CS- were also found on IBI 2 (p = .030), IBI 3 (p = .001) and IBI 5 (p = .010) in Block 2. Also, significant differences between CS+ and CS- were found on IBI 2 (p = .012), IBI 3 (p = .010), and IBI 4 (p = .005) in Block 3.

In summary, the PG group showed diminished aversive conditioning compared to the control group.

4. Discussion

One aim of this thesis was to explore the relationship between individual differences in classical conditioning and gambling behaviour. The three studies reported in this thesis addressed this question. Study 1 investigated whether individual differences in aversive conditioning was associated with risk-avoidance on the IGT (Bechara, et al., 1994). The results showed that participants who did not show aversive conditioning also showed less risk-avoidance on the IGT. This finding is in line with previous research that showed that individuals with diminished ability to develop aversive somatic reactions to losses on the IGT continue to display more risky decision-making compared to individuals who do not have such a deficiency (Bechara, et al., 1994). The finding is also in line with the risk-as-feelings hypothesis (Loewenstein, et al., 2001), which states that anticipatory emotions to risk alternatives may guide behaviour. Lacking the ability to acquire aversive anticipatory emotions may be why the non-conditioners showed less risk-avoidance.

Study 1 also investigated whether individual differences in baseline HRV and suppression of HRV during the IGT would be associated with differences in risk-avoidance on the IGT. The results showed that baseline HRV and HRV suppression were not associated with IGT performance. This finding was unexpected given that associations between HRV and executive functioning have previously been reported (Hansen, Johnsen, Sollers III, Stenvik, & Thayer, 2004; Hansen, et al., 2003). Since IGT performance in addition was also not associated with performance on the WCST, this may suggest that IGT performance is more contingent on aversive conditioning, rather than on executive functioning or cognition.

Study 1 also investigated whether BIS and BAS would be associated with risk-avoidance on the IGT (Bechara, et al., 1994). The results showed no associations between any of the BIS/BAS scales and IGT performance. This finding was not expected given that a previous study found associations between risk-taking and the BIS and the BIS/BAS ratio score (Demaree, et al., 2008). A likely explanation for this discrepancy concerns the operationalisation of risk-taking. Demaree et al. (2008) used

bet-sizes on a simulated slot machine, whereas the IGT was used in Study 1. Bet-sizes may be a more explicit measure of risk-taking compared to decision making on the IGT. With choosing bet-size, the game can immediately be switched from less risky to more risky, and vice versa, hence the individual has more control over the task. What constitutes risk taking on the IGT is perhaps less explicit, as it gradually becomes evident which cards should be avoided to avoid large losses. In that sense, the IGT is perhaps better construed as a measure of risk-avoidance rather than risk-taking. It could be speculated that individual differences in BIS and BAS may be more strongly associated with explicit rather than implicit risk decision making, and thereby explain the discrepant findings. The results from Study 1 are consistent with other studies that have reported no significant relationship between BIS and risk-avoidance (Franken & Muris, 2005; Suhr & Tsanadis, 2007). These studies have also reported both positive and negative relationships between BAS and risk-avoidance, and as Study 1 did not find a significant positive or negative relationship between any of the measures of BAS, more research is warranted to investigate this relationship, or to explain the discrepancies in findings.

Study 2 followed up on Study 1 by further exploring the relationship between conditioning and gambling behaviour, and BIS/BAS and gambling behaviour. In Study 2 appetitive conditioning was included in addition to aversive conditioning. In order to do this, an EC paradigm was employed that included both positive EC and negative EC. The outcome measure was also changed from the IGT (Bechara, et al., 1994) to a new simulated slot machine (the Hordaland slot machine, see section 2.3.5), since it may be more similar to commercially available gambling products. Study 2 investigated whether appetitive conditioning and aversive conditioning, were associated with preferred bet-sizes on the Hordaland slot machine. No main effects of appetitive or aversive conditioning were found on average bet-size. The interaction effect of appetitive conditioning and aversive conditioning was, however, significant and showed that the group of participants that did not show appetitive or aversive conditioning had lower average bet-size compared to the three other groups. A possible explanation for this finding may be that the participants who showed appetitive conditioning may have become influenced by positive events during

gambling, and this may have caused them to increase their bet-sizes in order to win even more money. Conversely, the participants who showed aversive conditioning may have become influenced by negative events during gambling, which may have caused them to increase their bet-sizes in order to chase their losses. The participants who showed both appetitive and aversive conditioning may have been influenced by both these processes, whilst the participants who did not show either may not have been influenced by either of these processes.

The results from Study 1 and Study 2 regarding the relationship between conditioning and risk-taking appear to contradict each other. However, the discrepancy in findings may be explained by some notable differences between the gambling tasks used in the two studies. The IGT involves gradually learning to avoid risky choices. On the Hordaland slot machine, however, there is no obvious strategy for avoiding loss of money. This suggests that the individuals in Study 2 who were conditionable tried to win or avoid losses by varying their bet-size, which resulted in higher average bet-size. In addition, there were some notable differences concerning the operationalisation of conditioning in the two studies. The physiological responses measured in the aversive conditioning paradigm in Study 1 may be qualitatively different from the self-reported changes in terms of liking in the EC paradigm. Skin conductance measurement bypasses cognitive control of responses and taps the physiological responses mediated by the sympathetic nervous system (Dawson, et al., 1990). Self-reported change in evaluation of pictures may be regarded as a more cognitive measure, as it has been shown to be influenced by contingency awareness (Walther & Nagengast, 2006). Thus, the two means of operationalising conditioning may be different. In addition, there is an ongoing debate concerning whether or not classical conditioning and EC measure the same underlying mechanism. For example, aversive classical conditioning may involve the learning of fear, whilst EC may involve the learning of disliking (Hofmann, De Houwer, Perugini, Baeyens, & Crombez, 2010).

Study 2 also built on the findings from Study 1 by further investigating the relationship between BIS/BAS and gambling behaviour. This time an alternative parsing of the items in the BIS/BAS scales were used that complies more closely with

the updated Reinforcement Sensitivity Theory (Gray & McNaughton, 2000; Heym, et al., 2008). In the updated theory, a distinction is made between the flight-fight-freeze system (FFFS) and BIS-Anxiety. FFFS is viewed as an individual's response to both primary aversive stimuli and conditioned aversive stimuli, whilst BIS-Anxiety represents inhibition of approach behaviour in circumstances where there might be aversive consequences. The results from Study 2 showed that FFFS was positively associated with average bet-size. This finding was somewhat surprising given that individuals who readily develop fear responses are assumed to avoid risk (Bechara, 2003). However, the finding was in line with findings from a previous study which found that punishment sensitivity was positively associated with risk-taking on the IGT (Davis, Patte, Tweed, & Curtis, 2007). A possible explanation for this is that individuals who react strongly to fear stimuli react strongly to losing money, which in turn may case them to increase bet-sizes in order to chase their losses.

The results from Study 2 also showed that BAS-scores were positively associated with average bet-size. Among the BAS sub-scales, only BAS-RR was associated with average bet-size, hence the BAS-average bet-size relationship may be attributed solely to this sub-scale. This finding was in line with findings from a previous study where BAS-RR was positively associated with risk-taking on the IGT (Suhr & Tsanadis, 2007). It is likely that high BAS-RR individuals should respond more positively to winning money during gambling compared to low BAS-RR individuals, since scores on BAS-RR reflects the degree to which a person experiences positive emotions when experiencing rewards (Carver & White, 1994). It is possible that the more positively individuals experience rewards, the more likely they are to gamble with high bet-sizes since high bet-sizes yield greater rewards.

The results from Study 2 also showed that there was an interaction effect between FFFS and BAS-RR on average bet-size. Among the participants who scored low on BAS-RR, only those who also scored low on FFFS scored lower on average bet-size compared to the other participants. This interaction effect indicates that FFFS may have moderating effect on the relationship between BAS-RR and risk-taking.

Both of the interaction effects in Study 2 suggest the operation of the same underlying process, that not showing appetitive conditioning or aversive conditioning, and scoring low on BAS-RR and low on FFFS, are associated with low average-bet size. Overall, it seems that individuals who are not easily affected by stimuli in general are also not affected by gambling to the extent that they play with high bet-sizes. It could be that individuals who are influenced by EC more easily develop illusion of control over the game and varied their bet-size more often, or conversely, that those who are unaffected by EC are more resistant to development of illusion of control. Individuals who are able to stay "cool" may play more rationally and play with low bet-sizes, since cognitively it may appear as if this is the best strategy for keeping as much of their money as possible.

Study 3 built on the findings from Study 1 and Study 2 by investigating whether a PG group would show diminished aversive conditioning compared to a control group. Since Study 1 showed that the not-conditioning group showed less risk-avoidance on the IGT, it was hypothesised that PGs would have diminished aversive conditioning. The results showed that the control group showed aversive conditioning, but that the PG group did not show aversive conditioning. This finding suggests that PGs may have deficiencies in neural structures that mediate aversive conditioning, such as the amygdala (Campeau, et al., 1992), the hippocampus, the anterior cingulated cortex, the insula and the medial temporal lobe (Sehlmeyer, et al., 2009). However, studies using neuroimaging are required to further investigate if this is the case. The finding in Study 3 is in accordance with previous indirect evidence for diminished aversive conditioning in PG. First, the results are in line with the finding from Study 1 showing that individuals who did not show aversive conditioning showed less risk-avoidance on the IGT. Individuals who have a deficiency in learning to associate losses during gambling with negative emotional responses are less likely to avoid risk, which means poor performance on the IGT, and perhaps also continued gambling behaviour that may develop into PG. The finding from Study 3 is also in line with the finding that psychopaths who were recruited from Gambler's Anonymous showed weaker physiological responses to repeated presentation of a neutral tone that preceded an aversive tone (Van Tharp, Maltzman, Syndulko, & Ziskind, 1980). Study

3 showed that a similar tendency was evident in a sample of PGs recruited via newspapers and treatment clinics.

The finding in Study 3 was also in line with previous research showing diminished aversive conditioning in alcohol use disorders (McGlinchey-Berroth, et al., 1995; McGlinchey-Berroth, et al., 2002). Alcohol use disorders are among the most common co-morbid disorders for PG (Petry, et al., 2005) and it could be the case that diminished conditioning is a pre-dispositional factor for the development of both alcohol use disorders, PG and perhaps also other forms of addiction. It is unclear whether diminished aversive conditioning is a cause of or a result of alcohol use disorders, as excessive alcohol consumption may cause brain deterioration (McGlinchey-Berroth, et al., 2002). However, The PG sample in Study 3 did not differ from the control group in hazardous alcohol use, and only 4 of the 20 participants in the PG group scored above the cut-off point for hazardous alcohol use. This suggests that the diminished aversive conditioning found in the PG group cannot be attributed to brain deterioration caused by excessive alcohol consumption. This indicates that that diminished conditioning may be pre-dispositional factor for the development of PG, however, more research is needed in order to further support this hypothesis.

In general, the finding from Study 3 may be in line with the suggestion that individuals with diminished aversive conditionability are likely to repeat harmful behaviour (Eysenck, 1977). It seems likely that individuals who fail to associate the harmful consequences of their behaviour with their behaviour will continue to display harmful behaviour. A question that arises is whether diminished aversive conditioning is a result of genetic predisposition, or whether it is a result of life experiences. Studies have shown that monozygotic twins show greater similarity in conditionability compared to dizygotic twins, which suggests a genetic influence on conditionability (Hettema, Annas, Neale, Kendler, & Fredrikson, 2003; Merrill, et al., 1999). Another possibility is that some individuals show emotional numbing as a result of traumatic childhood experiences, which may explain diminished aversive conditionability in adulthood. Neuro-imaging studies have shown that adults who experienced abuse during childhood had decreased hippocampus size (Bremner et al., 1997; Stein, Koverola, Hanna, Torchia, & McClarty, 1997). Since the hippocampus is one of the

neural structures involved in aversive conditioning (Sehlmeyer, et al., 2009), this suggests that childhood experiences may be a causal factor for diminished aversive conditionability in adulthood. Since there is evidence for both heritability in conditionability and for the role of experience in diminished conditionability, it is possible that an interaction between genes and environment determines level of conditionability.

The results of the three studies indicate that individual differences in conditionability may play a role in the development and maintenance of gambling behaviour and PG. Previous research has also suggested that individual differences in reinforcement sensitivity are associated with gambling behaviour and pathological gambling (e.g. Franken & Muris, 2005; Goudriaan, et al., 2005, 2006; Nordin & Eklundh, 1999; Potenza, Leung, et al., 2003; Potenza, Steinberg, et al., 2003; Reuter, et al., 2005). The process of classical and instrumental conditioning is included in the three integrated models that attempt to explain the development of PG. In Sharpe and Tarrier's (Sharpe, 2002; Sharpe & Tarrier, 1993) and Blaszczynski and Nower's (2002) models, classical and instrumental conditioning is mentioned explicitly as a fundamental process in the development of gambling problems. Shaffer et al. (2004) also includes the role of classical and instrumental conditioning in their model of the addiction syndrome, however more implicitly. They view pleasurable mood state associated with object of addiction (e.g. gambling) as fundamental in the development of addiction. Since the studies presented in this thesis show that individual differences in conditionability are associated with risk-taking when gambling and also that PGs show diminished aversive conditionability, classical conditioning may be viewed as a pre-dispositional factor for the development of PG in addition to being viewed as an important process in the development of PG. Individuals with increased appetitive conditionability may be more likely to develop gambling problems since they may more readily learn to associate the gambling environment with excitement. This hypothesis has gained some support (Sharpe, 2004). This suggested that PGs hold stronger classically conditioned associations between gambling and arousal compared to no-problem gamblers. The results from Study 3 adds to this field of enquiry by suggesting that PGs may less readily associate gambling with negative outcomes. In

summary, this research suggests that PGs may have pre-disposition for more easily associating gambling with positive rather than negative emotions. In combination with evidence that implicates the role of reinforcement sensitivity in gambling behaviour, the findings reported in this thesis suggest that the effects of the processes of classical and instrumental conditioning for gambling behaviour are contingent on individuals' conditionabililty and reinforcement sensitivity.

4.1 Implications

4.1.1 Theoretical Implications

The three studies may hold several implications for future research. Since the three studies were the first to investigate the role of aversive conditioning in gambling behaviour, the findings should be replicated by other research groups in order to corroborate the findings. Most importantly it is necessary to replicate Study 3. The enquiry into conditioning in PG may benefit from using larger sample sizes, and contrast a PG group with other clinical groups, such as patients suffering from anxiety disorders or alcohol use disorder in addition to a non-pathological group to investigate whether such groups differ in conditioning from the PG group. There is also further need to investigate the relative importance of appetitive and aversive conditioning in such an enquiry, though it may be difficult to find an appetitive US which is equal in magnitude to stimuli that is commonly used as aversive US. Future research may also investigate the relative importance of the acquisition of appetitive conditioning and the extinction of acquired appetitive conditioning since individual differences in both may be important for gambling behaviour and PG. With the advent of neuro-imaging techniques, a way forward is the use of functional magnetic resonance imaging (fMRI) in investigating conditionability in relation to gambling.

The relationship between BIS/BAS and gambling behaviour also requires further study. Since the BIS/BAS scales (Carver & White, 1994) are based on an outdated version of Reinforcement Sensitivity Theory (Gray, 1982), there may be a need to for new instruments. While an adjustment of the scoring of the BIS/BAS scales is available (Heym, et al., 2008), and instruments have been developed (Jackson, 2009)

to measure the updated theory (Gray & McNaughton, 2000), these measurements are in need of further validation. Nevertheless, using self-report measures to assess neural systems does require a leap of faith, and perhaps efforts should be concentrated on measures of behaviour rather than the underlying neural mechanisms that mediate behaviour.

Future studies should make efforts to ensure better gender balance in studies of conditionability and gambling, since there has been too little emphasis on female gamblers in the literature. This would also allow for investigation of possible gender differences in the development and maintenance of PG. Individual differences in conditioning can only be one part of the picture that attempts to explain the development and maintenance of gambling behaviour and PG. To determine the role of conditioning, studies are required that include several of the known factors for the development of gambling behaviour and PG in addition to individual differences in conditioning. If such studies employ longitudinal designs it is also possible to determine the directionality of conditioning and gambling, as well as possible mediating and moderating factors.

4.1.2 Practical Implications

The results of Study 3 may hold implications for treatment of PG. In the event that future studies corroborate the finding that PGs have diminished aversive conditionability, an implication concerning the use of aversion treatment for PG may become evident. If PGs do not readily acquire aversively conditioned associations, therapy that attempts to associate gambling with negative emotions may not prove sufficiently efficacious to warrant its use. Research has shown some effect of aversion treatment for PG, but also that aversion treatment may be less efficacious compared to cognitive behavioural therapy (Pallesen, Mitsem, Kvale, Johnsen, & Molde, 2005). A reason for this may be diminished aversive conditionability in PG.

Future studies may investigate the efficacy for extinction treatment for PG. This would involve extinguishing classically conditioned associations between gambling and excitement, and may be a promising avenue for further treatment research for PG as well as anxiety disorders and addictions (Quirk & Mueller, 2008)

4.2 Strengths and Limitations

4.2.1 Strengths

A particular strength of Study 1 was that the group that did show aversive conditioning and the group that did not show aversive conditioning did not differ on anxiety, depression, tobacco use, alcohol use, gambling problems, executive functioning, resting or suppression of HRV or BIS/BAS. Therefore, the differences between the two groups on risk-avoidance could not be attributed to any of these factors. In Study 2, a particular strength was the use of real money on the Hordaland Slot Machine. This strengthens the ecological validity of the task and may generate gambling behaviour similar to the gambling behaviour that would have been displayed in a naturalistic setting. A particular strength of Study 3 was that the PG group and the control group were matched on gender and age. The two groups did not differ in terms of hazardous alcohol use, and none of the participants were in the clinical range for anxiety or depression, therefore the differences in aversive conditioning may not be attributed to these factors.

All the three studies are among the first studies to directly investigate the role of classical conditioning in gambling behaviour and PG. Therefore they represent an important contribution to a field of enquiry that has been largely neglected empirically. Finally, a strength concerning all three studies was that they avoided the issue of common method variance, as independent and dependent variables were measured using separate methodologies (e.g. conditioning was measured using psychophysiology and gambling behaviour was measured using gambling tasks). This also avoided the reliance on self-report measures, which is characteristic of much of the research in psychology.

4.2.2 Limitations

The studies reported in this thesis have several limitations. The first limitation concerns the samples that were used in Study 1 and Study 2. Both samples were from populations of undergraduate university students and not randomly drawn from the community, and the sample comprised persons with restricted age range. Therefore,

the results cannot be generalised beyond student populations without reservations. In Study 3 sample size was low, but comparable to similar studies in the field (e.g. Sharpe, 2004; Van Tharp, et al., 1980). Larger sample sizes would have yielded greater statistical power, which would have allowed for the identification of group differences with smaller effect sizes. The PG sample in Study 3 was not randomly drawn from the population of PGs in the community and the control group was also not randomly drawn from the non-PG population, therefore the findings cannot be generalised beyond the sample without reservations. The sex distribution was skewed in favour of males, which also makes it difficult to generalise the results to the female population. Though the PG group and the control group were matched on sex and age, the PG group comprised more smokers, and it is possible that this confounded the results. Therefore, the study should be replicated with a sample of non-smokers, or a balanced number of smokers in the PG group and the control group. The PG group also had higher average scores on anxiety and depression. This is also a possible confounder, albeit these differences were rather small and none of the individuals in either group had anxiety or depression scores that were within a clinical range.

Study 3 did not differentiate between PGs who had different gambling preferences. Sharpe (2002) suggested that triggers for gambling may be different for gamblers who gamble to escape from their life circumstances and gamblers who seek excitement and avoid boredom. The inclusion of groups of PGs with different game preferences, for instance one group of slot machine gamblers, a group of horse race gamblers, and a group of poker players would have allowed for a comparison of these subsets of gamblers. Future studies may consider such a design if they have access to large enough groups of gamblers. Blaszczynski and Nower (2002) also suggested that PGs may have had different pathways to developing PG, and that the pathways to developing PG are determined by predispositional factors. The inclusion of groups of PGs who have followed the three pathways may have allowed for comparisons of these groups, and future studies may consider such a design if access to large numbers of PGs is granted.

Studies 1 and 2 have limited ecological validity as gambling took place in a laboratory. It has been shown that casino gambling and laboratory gambling can be

experienced differently (Anderson & Brown, 1984), therefore it is possible that conducting the study procedures in casinos or other gambling venues would yield different results. Studies 1 and 2 also employed gambling tasks that were different from available gambling games. This is especially the case in Study 1 where the IGT was used. While it has been claimed that the IGT can be considered an ecologically valid gambling task (Goudriaan, et al., 2005), it differs from commercial gambling games in that it is possible to lose more money than the value of the wager. Therefore, it was attempted to use a measure of gambling which more closely resembled available gambling games by constructing the Hordaland Slot Machine for Study 2. However, although real money was used with the Hordaland Slot Machine in order to improve ecological validity, this money was provided by the experimenter, and was not the participants own money, which is usually the case in real gambling.

There were also limitations concerning measurement. In Study 1, the focus in the aversive conditioning paradigm was on the extinction phase, and the design did not allow for scoring the acquisition of aversive conditioning since the interval between CS onset and US onset was too short for scoring of skin conductance responses. The conditioning paradigm in Study 1 did not include an habituation phase. Therefore, it was not possible to determine if the SCRs to the CS+ and CS- were different before the acquisitioning phase, or if the two groups had different responses to the CS before the acquisitioning phase. The focus in Study 1 concerned whether or not the participants had learned the association after the acquisitioning phase. If the study had compared differences in the acquisition phase, this could have made the results of Study 1 more directly comparable to Study 3, since Study 3 focused on the acquisition of aversive conditioning.

In Study 2 a total of eighteen participants were excluded from the EC analysis because they failed to rate the positive US more positively than the CS they were paired with, and the negative US more negatively than the CS they were paired with. Hence, the EC paradigm did not work for these participants. Therefore it is possible that the US could have been stronger (positive or negative) and it is also possible that some CS were not free from previous association. The loss of eighteen participants

could have influenced the results, however, it is difficult to forecast in which direction these participants would have influenced the results.

Another measurement issue, which is a possible limitation, is that in Study 1 and Study 2, the internal consistency (Cronbach's alpha) was low for the BAS-FS and BAS-RR subscales. This questions whether the items reflect underlying unidimensional concepts. If the scales had comprised more items and had greater internal consistency this could have yielded different results.

A limitation concerning the data collection for Study 3 is that some of the PGs were tested in office rooms at treatment clinics and not in the laboratory. It would have been ideal to test all participants under identical conditions, but this was not practically possible. The office rooms had a similar set-up and were quiet and heated to normal office temperature, therefore conditions were similar albeit not identical. Finally, the three studies did not use longitudinal methodology, but were more akin to cross-sectional research. Therefore it was not possible to determine directionality or causality.

4.3 Conclusion

This thesis reports the results from three studies that investigated the relationship between conditioning and gambling. Study 1 used a student sample and found that the participants who did not show aversive conditioning showed less risk-avoidance on a gambling task compared to the participants who showed aversive conditioning. In addition, Study 1 found no differences between the two groups on behavioural activation and behavioural inhibition. Study 2 also used a student sample, and found that participants that did not show appetitive or aversive conditioning on an evaluative conditioning paradigm showed less risk-taking on a simulated slot machine. In addition, Study 2 found an interaction effect between the FFFS and BAS-RR on risk-taking, where the participants with low scores on both scales showed less risk-taking. Study 3 compared a PG group with a control group on results of an aversive conditioning paradigm. The results showed that whilst the control group showed aversive conditioning, the PG group did not show conditioning. The results from these

three studies contribute to the extant literature by suggesting that conditionability plays a role in gambling behaviour and PG. The results also suggest that BIS and BAS may play a role in gambling behaviour. The processes of classical and instrumental conditioning are included as important in the development of gambling behaviour and PG. This thesis suggests that the effects of the processes of classical and instrumental conditioning for gambling behaviour are contingent on individuals' conditionability and reinforcement sensitivity.

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