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Conceptualizing problem gambling via two neuro-motivational systems.

Nicola E. Fitzgerald
University of Windsor

NOTE TO USERS

Conceptualizing Problem Gambling via Two Neuro-motivational Systems

by

Nicola E. Fitzgerald, M.A.

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ABSTRACT

Although impulsivity is commonly associated with problem gambling (PG), relatively little is known about the mechanisms that drive gambling behaviour. The Behavioural Activation System (BAS) and Behavioural Inhibition System (BIS) are widely used components of Jeffrey Gray's (1981) sensitivity to reinforcement model used to study disinhibitory behaviour. These constructs were applied to further the understanding of the disinhibition mechanisms underlying PG. Eighty-eight individuals who endorsed having gambled in the last 12 months were recruited and combined in the study from a variety of community agencies (n = 18) and from an undergraduate psychology pool (n = 70). A number of self report measures of the BIS and BAS and two computer implemented disinhibition tasks (go/no-go [Patterson & Newman, 1993], stop-signal [Logan & Cowan, 1984]) were employed as dependent variables. The Problem Gambling Severity Index (PGSI; Ferris and Wynne, 2001) was the measure of PG. When all BIS and BAS measures were entered simultaneously into a regression equation, both high BAS (novelty seeking and reward expectancy) and high BIS (harm avoidance) scales were positively predictive of gambling severity. Consistent with Newman's views of passive avoidance learning deficits in syndromes of disinhibition, the PGSI, as a continuous scale was correlated with commission errors in the mixed reward and punishment condition. When the PGSI was used to define ordinal groups as prescribed by Ferris and Wynne (2001), PG was linearly and monotonically associated with errors of commission when errors of omission were first subtracted out. Contrary to Gray's theory of disinhibition, errors of commission in the punishment only condition of the go/no-go task were not associated with low BIS or gambling category.

An exploratory analysis indicated that stop signal reaction times were not a better predictor of gambling severity than the BIS/BAS measures, suggesting that disinhibition in gamblers is not due to a general deficit in the ability to stop ongoing behaviour. Finally, TPQ-NS and TPQ-HA, measures of reward and punishment sensitivity, were the best predictors of problem gambling severity of the variables included in this study. Results and their implications for treatment were discussed.

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CHAPTER I

INTRODUCTION

The constellation of features that comprise the definition of pathological gambling could have described the behaviour of individuals dating back to the time of the Roman Emperors (Wildman, 1997). Nonetheless, pathological gambling was not a recognized, diagnosable entity until its debut in the *Diagnostic and Statistical Manual of Mental Disorders, Third Edition* (*DSM-III*) in 1980 (American Psychiatric Association, 1980). Since that time, estimated prevalence rates in community samples typically range between 1-2% (Walker & Dickerson, 1996; Petry & Armentano, 1999), while lifetime prevalence rates of pathological gambling are reported to be as high as 5.1% (Petry & Armentano, 1999).

Perhaps more troubling is the significant increase in the prevalence of pathological gambling in the last 20 years (Shaffer, Hall, & Vander Bilt, 1999), given that the negative consequences of gambling are not solely borne by the gamblers themselves, but also by their families and by society. Pathological gambling has been associated with increased suicide risk (Thompson, Gazel, & Rickman, 1996); exacerbation of other mental disorders and stress-related illnesses (Lorenz & Yaffe, 1986); and job loss (Ladouceur, Boisvert, Pepin, Loranger, & Sylvain, 1994). Spouses of pathological gamblers are reported to experience increased rates of emotional and physical illnesses (Lorenz & Yaffe, 1988), as well as physical and emotional abuse (Bland, Newman, Orn, & Stebelesky, 1993; Lorenz & Shuttlesworth, 1983). Moreover, pathological gambling touches society in terms of cost to employers (Thompson et al., 1996), increased rates of bankruptcy claims (Ison, 1995), and increased rates of crime (Blaszczynski, 1994).

Canadian studies of pathological gambling are necessary since research has suggested unique aspects of the Canadian gambling experience (Beaudoin & Cox, 1999; Wynne, 2002). For instance, in one Canadian sample of problem gamblers, one-third of treatment seekers were between the ages of 18 and 34 years and women composed almost half of the sample (47.5%). This is in comparison to some US studies which have examined problem gambling in older male samples (Kaplan, 1996).

Furthermore, gambling in Canada is expected to increase. According to a report of forecasted trends (Wynne, 2002), gambling in Canada is expected to expand with the growth and popularity of machine-based gambling (i.e., video lottery terminals, electronic Keno and bingo, etc.) and the legalization of Internet gambling due to the difficulty enforcing the current laws. Moreover, if lobbyists of the Canadian tourism and hospitality industry are successful, then the appearance of special gaming rooms and mini casinos in hotels, convention centres, and tourist facilities is expected. Lastly, it is predicted that gambling revenues will increasingly become part of the fundraising strategies of charitable organizations. The study of pathological gambling is thus clearly clinically and socially relevant.

The classification of pathological gambling as a disorder of Impulse-Control in the *DSM- Fourth Edition – Text Revision* (*DSM-IV-TR*: American Psychiatric Association, 2000) implies that impulsivity is a key component of the disorder. While various studies support this classification, research in the area has tended to remain at a more descriptive level, without much investigation into the mechanisms or processes that contribute to, maintain, or exacerbate the problem. One way of addressing this limitation of the research is to look at other ways that impulsivity has been examined, as both a

construct in its own right, as well as a characteristic of other disorders (i.e., psychopathy and attention deficit disorder).

For decades, researchers whose interests span both learning theory and the neuronal bases of personality and psychopathology have conceptualized impulsivity and disinhibition by reference to two neuro-motivational systems (Cloninger, 1998; Eysenck, 1957; Fowles, 1980, 2001; Gray, 1970, 1982), one involved with activating behaviour, the other with inhibiting behaviour. One of the leaders in this area, Jeffrey Gray (1970, 1982), applied his results from drug and lesion studies in animals to human behaviour by describing two motivational systems, the behavioural inhibition system (BIS) and the behavioural activation system (BAS). According to Gray, the BIS was described as being associated with punishment sensitivity. He proposed that a less sensitive BIS is associated with disinhibited behaviour and deficits in passive avoidance learning. He also aligned this construct with anxiety. In contrast, the BAS was proposed to be associated with sensitivity to reward and approach behaviour. He also aligned it with impulsivity.

The BIS and BAS constructs have been used to understand and describe the disinhibited behaviour exhibited by individuals with psychopathy (Patterson, Kosson, & Newman, 1987; Newman, 1987), attention deficit-hyperactivity (Quay, 1988), as well as extraversion (Newman, Widom, & Nathan, 1985), and alcohol abuse and dysfunctional eating (Loxton & Dawe, 2001). They have not, however, been explicitly applied to the study of problem gambling. Given the reported relation between impulsivity and gambling and the inherent rewards and punishments associated with gambling, the application of the BIS/BAS constructs to further the understanding of mechanisms underlying problem gambling seems appropriate.

While alternative theories of human disinhibition have been proposed, they tend to share a number of common features with Gray's model. For instance, in each case, one system is thought to cue the activation of movement (primarily approach toward a cue for reward, but secondarily escape from an approaching pain), whereas the other system governs inhibitory processes involved in avoidance (primarily of a cue for punishment, but secondarily for frustrative non-reward[1]).

These two systems operate independently of one another, although in any particular situation, both activation and inhibition of a response may be triggered by the same stimulus. Both systems are best understood in the context of learning theory. The BAS triggers an activating impulse consisting of approach towards a cue for reward (secondarily, fleeing an approaching pain). This occurs as a result of prior learning in which the cue has come to be associated with reward contingent on a response. The BIS, conversely, triggers an inhibitory impulse consisting of a "do not approach" response in the presence of a cue for punishment (secondarily, a cue for frustrative non-reward) that occurs as a result of prior learning in which the cue has come to be associated with punishment contingent on response.

In contrast to many recent and current "network" (Anderson, 1995) or "connectionist" (Rumelhart, 1989) models, the theory is offered not simply as an abstract model, but is hypothesized, and in the case of Gray's (1970) model has been empirically tested, to be implemented in actual neuronal circuitry (i.e., in the brain). Each theory suggests that there are individual differences in the strength of these systems, that is, in the efficiency of the systems to profit from learning experiences. Some individuals may

[1] Frustrative non-reward refers to situations in which a previously rewarded approach response to a cue was not rewarded.

have too highly calibrated an inhibition system, and so the individual becomes overly cautious for fear of being harmed (i.e., harm avoidance). Others may not be able to benefit from punishment, and so they may fail to learn to avoid punishing experiences.

Lastly, in addition to this anchoring of the theories in both neural mechanisms and in learning theory as sketched above, each theory also supports major aspects of personality theory, at least those aspects concerning neuroticism, extraversion, negative and positive emotionality, and emotional arousal. As might have been gleaned from the above description, various configurations of BIS/BAS sensitivities have been proposed to account for disinhibited behaviour. Some argue that people who are less sensitive to punishment might appear disinhibited. Alternatively, those who are more sensitive to cues of reward might appear disinhibited as their BAS overrides their BIS. Moreover, disinhibition may be contingent on what types of stimuli are present.

Presently it is unknown where exactly gamblers fall along this spectrum. One could easily imagine how any of the above BIS/BAS configurations could account for gamblers' seemingly disinhibited behaviour. For instance, it could be argued that problem gamblers are less sensitive to punishment cues given the substantial losses that they often accrue, both financially and personally, in the form of job, relationships, etc. It might also be argued that due to overly sensitive BASs, the prospect of winning leads problem gamblers to behave in a disinhibited manner. Alternatively, it might be that problem gamblers act in a disinhibited fashion only when confronted with situations in which both reward and punishment cues are salient. Or finally, problem gamblers' disinhibited behaviour might also stem from a more general deficit in their stopping process, specifically one that is slower, and that is not overtly related to cues of reward or punishment. The goal of this study is to address these questions.

As it stands presently, there seems to be an emerging consensus (Fowles, 2001) that Gray's (1981, 1987) work in the study of the behavioural inhibition and activation systems is the most accurate description of these constructs. For this reason, most of the paper and pencil measures used in this study as measures of activation and inhibition have targeted Gray's constructs. Of course, it is recognized that paper and pencil measures simply lack the specificity to target any one of the conceptualizations to the exclusion of another. Hence, although it is tempting to say that the study tests Gray's theory, it is more accurate to say that it tests a more general, non-specific version of the theory. Most particularly, the study contains no measures that are pharmacologically based, these measures being perhaps Gray's greatest contribution to the advancement of the theory. For this reason, the study is probably most accurately described as an investigation of the relation of the two neuronal system theory to problem gambling.

Although it might be intuitively expected that gamblers have increased sensitivity to rewarding stimuli, very few studies have explicitly applied Gray's concepts to further the understanding of impulsivity in problem gamblers. Using self-report measures and computer-based tasks, this study sought to examine the applicability of the BIS and BAS constructs to problem gambling. Specifically, the study sought to determine: (a) whether increased gambling severity is associated with increased responsivity to rewarding stimuli (i.e., winning or the potential of winning) as reflected by high scores on measures of BAS (b) whether gambling severity is associated with decreased sensitivity to punishment (i.e., when losing) as reflected by an underactive BIS and low scores on related measures; or (c) whether there are certain circumstances in which gamblers are more likely to act in a disinhibited manner, such as when both cues of reward and punishment are present.

As the application of BIS/BAS concepts to problem gambling is relatively new, there were some areas that we were less certain about and thus these questions were approached on a more exploratory basis. Specifically, along the lines of a response modulation hypothesis, we explored whether the difference between errors of commission and errors of omission made in the mixed reward and punishment condition differed on the basis of problem gambling category. Furthermore, we explored whether problem gambling severity was more related to general deficits in stopping processes, rather than to a specific oversensitivity to reward or undersensitivity to punishment. Finally, we examined which of the variables that predicted or were correlated with problem gambling severity or category best predicted problem gambling severity when tested as a group.

Pathological Gambling – Some Definitions

Before embarking on a brief review of gambling through the ages, it is important to establish a working definition of gambling. Unlike the British Royal Commission on Gambling (1978, as cited in Blaszczynski, 1996) which defined gambling rather flippantly by saying, "almost everybody knows intuitively what gambling is," Blaszczynski (1996) took a more formal approach to the task and looked to what others in the field saw as the core elements of gambling. This led him to a definition of gambling that described it as involving the following features: (a) the existence of an agreement between at least two parties (b) to exchange items of value (c) on the basis of the outcome of an uncertain event (d) where participation is voluntary. Blaszczynski (1996) added a motivational component to the above definition: participants might be driven to risk items of value in order to gain profit and/or induce some subjective state of arousal.

A challenge in the gambling literature has been the consistent use of a term to apply to gambling behaviour that is problematic and/or excessive. Shaffer, Hall, and Vander Bilt (1999) note that "conceptually equivalent categories have been given different names by different authors" (p. 1370). To demonstrate their point, various labels have been used to describe gambling behaviour that is problematic including pathological gambling, compulsive gambling, problem gambling, potential pathological gambling, probable pathological gambling, etc. Compulsive gambling was the original lay term used to describe pathological gambling and it continues to be used by Gamblers Anonymous and other self-help groups (National Research Council, 1999). Pathological gambling is the term preferred by the medical fields and denotes a mental disorder (National Research Council, 1999; Raylu & Oei, 2002). The term problem gambling has been applied to the gambling behaviours of people whose problems, as measured by one screening tool, the South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987), are less than those of people classified as probable pathological gamblers. The term has also been used to refer to gambling behaviour that results in any harmful effects, though it might not meet strict diagnostic criteria (National Research Council, 1999). Others, however, have used it almost as a wastebasket term to include all problematic gambling behaviour, including pathological behaviour. The problem gambling label is preferred by some because it is thought to avoid "negative judgements and conceptual issues" that are often associated with pathological gambling (Allcock, 1994, as cited in Walker & Dickerson, 1996, p. 243), as well as the medical and negative connotations of the word "pathological" (Walker & Dickerson, 1996).

Others have attempted to move beyond naming categories of gamblers and instead classified those who gamble according to three "generic" levels (Shaffer et al., 1999,

p.1370). According to this classification system, Level 1 gamblers represent individuals who do not exhibit problems related to their gambling; they are essentially the nonproblem gamblers and nongamblers. Level 2 captures individuals who would be considered subclinical and have been referred to as "problem," "at risk," or "potential pathological." Finally, those in the Level 3 are likely to be those who meet DSM criteria for pathological gambling and experience the most severe problems related to gambling. In this paper, PG will be used to refer to problematic gambling behaviour that might or might not meet diagnostic criteria for pathological gambling, as the study is a preliminary application of Gray's constructs to gambling behaviour.

In addition to the *DSM* criteria, various other measures were developed to identify problem gambling. One of the earliest and most widely used measures was the SOGS (Lesieur & Blume, 1987). The measure, based on *DSM-III* criteria (American Psychiatric Association, 1980), was initially developed for use in clinical populations. Over time, however, it soon became widely used in surveys of the general population where its sensitivity and specificity were less clear (Volberg, 1996). One recent study found that the SOGS had a 50% false positive rate in general populations (Stinchfield, 2002). To overcome this potential problem, the Canadian Problem Gambling Index (CPGI; Ferris, Wynne, & Single, 1999) was created for use in general populations. This measure consists of both an index of gambling severity (i.e., Problem Gambling Severity Index; PGSI) and a "softer" section intended to capture aspects of gamblers' experience, such as type of gambling activity engaged in, family history of gambling problems, etc. An additional advantage of the CPGI is the inclusion of questions placing gambling into a social context. The hope was that such questions would allow for the better capture of previously missed or "non-traditional" problem gamblers, such as women, ethnic

minorities and those from low socioeconomic brackets (Ferris & Wynne, 2001). As such, the CPGI was administered to study participants and problem gambling severity was operationalized by the PGSI.

Historical Overview

Despite its only recent inclusion in the *DSM-III* in 1980, gambling and problems associated with it have existed through the ages in almost every race and culture (Blaszczynski, 1996). One of the earliest depictions of gambling was found in an Egyptian mural displaying various types of board games, including one that looked like checkers (Blaszczynski, 1996). Six-sided dice were reportedly used by Etruscans and given to the Romans in 600 B.C. (Blaszczynski, 1996) and lotteries were known to exist in Roman times. There is also evidence to suggest that Homer, Ovid, Herodatus, and Xenophon were likely horserace enthusiasts (Blaszczynski, 1996).

Just as sure as gambling existed, so too did problems of excessive gambling and attempts by societies and governments to control gambling behaviour. One early report of the substantial losses that can result following a round of gambling involved an Indus River tribesman who in 1500 B.C. reportedly gambled away 200, 000 slaves, his kingdom, his brothers, and his wife (Blaszczynski, 1996). Attempts to curtail gambling included Aristotle categorizing gamblers amongst thieves and robbers. Aristotle was of course no stranger to the principles of classification and this way of grouping gamblers suggests that he as well thought of it as a disorder stemming from disinhibitory problems. By 1822, most European states had laws prohibiting gambling due to its negative impact on social and personal welfare (Blaszczynski, 1996).

Descriptions of the addictive and compulsive processes involved in gambling can be found as early as 1619 in a book passage that states, "most gamesters begin at small

games; and by degrees, if their money or estate holds out, they rise to great sums; some have played first all their money, their rings, coach and horses, even their wearing clothes and perukes; and then such a farm; and at last perhaps a lordship" (as cited from Blaszczynski, 1996, p.4). Moreover, in *The Gambler* (1866), Dostoevsky describes the all consuming nature of gambling, stating that one can feel "powerless in the clutches of this terrific gambling mania" that can "blunt (the) sense of moral responsibility as effectively as extreme alcohol addiction could" (as cited in Blaszczynski, 1996, p.6).

Some of the early thinking about gambling has conceptualized it as an addiction, much like alcoholism or substance abuse. In 1957, the modern Gamblers Anonymous was founded and modeled after Alcoholics Anonymous. Those lobbying for the inclusion of PG in the *DSM-III*, such as Gamblers Anonymous, thought it should be included amongst the other substance use disorders. Given its inclusion amongst the Impulse Control Disorders, evidently the *DSM* task force did not concur. Interestingly, however, PG criteria appearing in the latest version of *DSM-IV-Text Revised* (*American Psychiatric Association*, 2000), is closely modeled after the criteria seen in the substance use section.

Proposed Conceptualizations of PG

As alluded to above, different conceptualizations of PG have been proposed and argued for by various groups. One principal components analysis of measures completed by 115 pathological gamblers produced a four factor model that included the following components: psychological distress, sensation seeking, crime and liveliness, and impulsive antisocial (Steel & Blaszczynski, 1996). When examining the gambling correlates of these factors, the impulsive antisocial was argued to have the most clinical utility because it seemed to be more related to gambling-related consequences, such as a

greater likelihood of being divorced or separated from a spouse due to gambling and having a high number of jobs with a short period of employment. Later, in an attempt to integrate elements of biology, personality, developmental theory, learning theory, and environmental factors, Blaszczynski and Nower (2002) proposed three pathways to PG that were labelled "normal," "emotionally disturbed," and "biological correlates." According to Blaszczynski and Nower (2002), the normal group was characterized by an absence of premorbid psychopathology (e.g., substance abuse) and less severe difficulties resulting from gambling relative to the other PG groups. In contrast, the emotionally disturbed group, as the label suggests, exhibits a history of psychological vulnerability factors that included a history of problem gambling in the family, negative developmental experiences, neurotic personality traits, and stressful life events. Lastly, the biological correlates group was proposed to manifest such biological factors as neurological or neurochemical dysfunction suggestive of impulsivity or attention deficit features.

In a recent review article (Moreya, Ibanez, Saiz-Ruiz, Nissenson, & Blanco, 2000), four competing conceptualizations of gambling were presented to account for the phenomenology of PG. These bear some similarity to Blaszczynski's (1996) pathways. These conceptualizations propose that PG should be considered as either: (a) a non-pharmacological addiction; (b) a form of affective disorder; (c) part of the obsessive-compulsive spectrum; and lastly (d), as an impulse control disorder.

Proponents of the addiction theory cite various similarities between the experience of PG and other addictions (Langewisch & Frisch, 1998; 2001). For example, withdrawal symptoms such as irritability, psychomotor retardation, difficulty concentrating, and somatic complaints have been documented in a group of gamblers (Dickerson, 1989). In another study, 65% of 222 PGs reported at least one somatic complaint when trying to

reduce their gambling (Rosenthal & Lesieur, 1991). Similar to alcoholics who develop a physiological tolerance to alcohol and need to drink more to get the same desired effect, increases in gambling activity and preoccupation with gambling have also been reported (Jacobs, 1988; Lesieur, 1979). Moreover, various studies have reported increased rates of alcohol abuse in gamblers. For example, 39% of 51 successive individuals seeking treatment for problem gambling also met criteria for alcohol abuse in the past year (Ramirez, McCormick, Russo, & Taber, 1983), while in another study it was reported that 48% of females in a sample of Gamblers Anonymous members met criteria for substance abuse or dependence during their lifetime (Lesieur & Blume, 1991).

Evidence supporting the view that PG lies on the obsessive-compulsive spectrum includes reports of increased rates of obsessive traits in individuals with PG (e.g., Blaszczynski & McConaghy, 1988; Petry, 2000b, Blaszczynski, 1999; Bazargan, Bazargan, & Akanda, 2000; Frost et al., 2001). Those opposing this view state that unlike the behaviours of those diagnosed with Obsessive Compulsive Disorder (OCD), the behaviour of PGs is not ego-dystonic (American Psychiatric Association, 2000), nor do individuals with PG exhibit the excessive self-doubt characteristic reported by people with OCD (Rasmussen & Eisen, 1992).

The view of PG as an affective disorder was proposed to account for the high incidence of depression reported during the lifetime of problem gamblers (Roy, Custer, Lorenz, & Linnoila, 1988). Others have suggested gambling might serve as a means of dealing with feelings of depression. McCormick, Russo, Ramirez, and Taber (1984) diagnosed 76% of gamblers seeking treatment with a major affective disorder using the Research Diagnostic Criteria. Moreover, 14% reported that the depression had preceded the gambling behaviour.

Lastly, PG has also been characterized as an Impulse Control Disorder, Not Otherwise Specified (American Psychiatric Association, 2000). As a major component of this study involves the relation between impulsivity and gambling, the literature supporting this view will be reviewed more fully shortly.

To examine the strength of the relation between PG and the substance-use disorders, OCD-spectrum, and impulsivity, a meta-analysis that included 54 published studies up to June 15th, 2004 was recently conducted (Wilkie, 2004). Results indicated that PG had the strongest relation with impulsivity ($d = .69$), followed by the obsessive-compulsive spectrum ($d = .61$), but because the difference between these values was not statistically significant, they could be considered equal influences. This finding supports previous assertions that impulsivity is at the very least a component of PG (Raylu & Oei, 2002; Sharpe, 2002; Blaszczynski & Nower, 2002), and thus is an area deserving of continued study to better understand the role it plays in gambling behaviour.

Impulsivity and Gambling

Impulsivity has been cited as a "major characteristic" of Pathological Gambling (Raylu & Oei, 2002, p.1023; Sharpe, 2002) and thus it seems appropriate that it is classified in the *DSM* amongst the Impulse-Control Disorders Not Elsewhere Classified. Other disorders included under this heading are: Intermittent Explosive Disorder, Kleptomania, Pyromania, and Trichotillomania. One of the defining characteristics of these disorders is a failure to resist an impulse, drive, or tension that results in harm to the person or to another (American Psychiatric Association, 2000). Other core features include an increase in tension prior to committing the impulsive act, and pleasure and/or

gratification and/or relief of tension during the act, which can be followed by regret, guilt, or self-reproach (American Psychiatric Association, 2000).

Supporting the relation between gambling and impulsivity are findings that self-report measures of impulsivity differentiate individuals identified as problem gamblers from control groups (Carlton & Manowitz, 1994; Blaszczynski, et al., 1997). Castellani and Rugle (1995) compared impulsivity levels of individuals with a primary diagnosis of PG, alcohol dependence, or cocaine dependence. They found that the gamblers scored significantly higher on the Barratt Impulsivity Scale-10 (Barratt, 1985), both on the total score and on subfactor scores (i.e., cognitive, motoric, and non-planning impulsivity). In addition, they exhibited an inability to resist cravings as measured by Costa and McCrae's N5 Impulsivity subfactor.[2] No significant differences were reported for levels of sensation seeking, a variable often reported in studies of impulsivity.

In another study, impulsivity, measured by both a self-report measure (i.e., subset of questions from the Eysenck Impulsiveness scale) and a card-sorting task (based on Newman, Patterson, & Kosson, 1987,), predicted problem gambling in a sample of adolescents (Vitaro, Arsenault, & Tremblay, 1999). Participants who scored in the 70th percentile on the Eysenck impulsivity questions were six times more at risk of becoming problem gamblers than those who scored below the 70th percentile. In addition, performance on the card sorting task increased risk of problem gambling an additional three times, above and beyond the effect of the impulsivity score. Impulsivity has also been associated with pathological gambling severity (Alessi & Petry, 2003), treatment

[2] Castellani & Rugle (1995) argued that the N5 Impulsivity subfactor was better defined as a craving scale as those with low scores were reportedly more easily able to resist temptations for food, cigarettes, etc, while high scorers had low frustration tolerances.

drop-out (Leblond, Ladouceur, & Blaszczynski, 2003) and problem severity (Steel & Blaszczynski, 1998).

Biological correlates of impulsivity have also been linked to gambling behaviour. Serotonin is a neurotransmitter frequently associated with disorders of impulse control, such as borderline personality disorder (Paris, et al., 2004) and bulimia nervosa (Steiger, et al., 2001), thus it is not surprising that links have also been made between gambling and serotonin dysfunction (i.e., deficit). Assessing the functioning of serotonin can be accomplished by using various methods such as measuring the activity of platelet monoamine oxidase (MAO), measuring levels of 5-hydroxyindoleacetic acid, a serotonin metabolite found in the cerebrospinal fluid and examining the effect of pharmacological treatment, for example determining whether serotonin reuptake inhibitors (SRI) reduce gambling behaviour. Impulsive behaviours have been associated with lowered platelet MAO activity (Schalling, Edman, Asberg, & Oreland, 1988), a finding that has also been reported in two samples of male pathological gamblers (Blanco, Orensanz-Munoz, Blanco-Jerez, & Saiz-Ruiz, 1996; Carrasco, Saiz-Ruiz, Hollander, Cesar, & Lopez-Ibor, 1994). Studies examining CSF 5-HIAA level, however, have been less successful in differentiating gamblers from control groups (Bergh, Eklund, Soedersten, & Nordin, 1997; Roy, et al., 1988). Pharmacologically, fluvoxamine, a selective SRI, significantly decreased gambling behaviour and urges in a 16-week randomized double-blind crossover study (Hollander, et al., 2000); clomipramine, a partially selective SRI blocker, was also reported to decrease gambling behaviour in a case study (Hollander, Frenkel, DeCaria, Trungold, & Stein, 1992).

Despite the existence of research supporting the link between impulsivity and gambling, not all findings have been so positive. For example, impulsivity scores failed

to differentiate between gamblers and a control group in a study by Allcock and Grace (1988). The study's small sample size and unknown criteria for classifying the gamblers, however, might have contributed to this result. Langewisch and Frisch (1998, 2001) reported that although pathological gamblers had significantly higher impulsivity scores than non-pathological gamblers, impulsivity scores did not predict problem severity as previously suggested (Steel & Blaszczynski, 1998).

Comorbidity with other disorders associated with impulsivity. PG has been associated with other disorders characterized by impulsivity such as Attention Deficit-Hyperactivity Disorder (ADHD). For instance, Rugle and Melamed (1993) reported that gamblers endorsed a greater number and intensity of childhood behaviours associated with ADHD when compared to those in a control group. Moreover, those in the gambling group used more trials on the Wisconsin Card Sorting Task, took longer on the Embedded Figures Test, and tended to make more errors on the Porteus maze task, suggesting deficits in the cognitive area of impulsivity. Interestingly, however, childhood history did not correlate with current neuropsychological functioning. Lastly, Goldstein, Manowitz, Nora, Swarzburg and Carlton (1985) reported a pattern of hemispheric dysregulation in a sample of men with a history of pathological gambling. This profile is similar to that found in a sample of unmedicated children diagnosed with ADHD.

As several of the personality disorders (PDs) include impulsivity as a diagnostic criterion, particularly those in cluster B (i.e., Borderline, Anti-Social, Narcissistic, and Histrionic Personality Disorder), it should not be surprising that associations between PG and these disorders have been reported. In one study, as many as 93% of individuals recruited from a gambling treatment program met criteria for at least one PD, with the most common diagnoses being from the cluster B group (Blaszczynski & Steel, 1998).

Not surprisingly, those who received a diagnosis from this cluster also tended to have heightened impulsivity scores. In another sample of pathological gamblers, 40% met criteria for antisocial PD (Bland, Newman, Orn, & Stebelesky, 1993).

If impulsivity levels are elevated in individuals meeting criteria for PG, one might expect for this group to exhibit a higher rate of comorbidity with the other Impulse Control Disorders, relative to normal control groups. Indeed, when this question was addressed, 35% of a PG group met criteria for a comorbid Impulse Control Disorder, compared to only 3% of the normal control group (Specker, Carlson, Christenson, & Marcotte, 1995). Support for an increased rate of PG comorbidity with the other Impulse Control Disorders is reported elsewhere (Black & Moyer, 1998). In addition, Grant and Kim (2003) reported that individuals with a comorbid Impulse Control Disorder had greater thoughts and urges related to gambling and reported more interference and distress than PG without the comorbid Impulse Control Disorder.

The Construct of Impulsivity

To this point, the focus has been on demonstrating an association between impulsivity and problem gambling and based on the literature reviewed above, there is evidence to support this argument. The question remains, however, just what is impulsivity? Even the *DSM-IV-TR* (American Psychiatric Association, 2000) with its entire category devoted to disorders of impulse control fails to define just what this thing called "impulsivity" is. Even in the impulsivity literature itself, there is controversy about how to define the object of their study.

One reason for this conundrum is the multidimensional nature of impulsivity. Kindlon, Mezzacappa, and Earls (1995) indicate there are many ways in which

impulsivity can be manifested, such as cognitive, emotional, and motoric, to name a few. Conceivably then, there are also various physiological pathways that might lead to what appears to be same overt impulsive behaviour. Evendon (1999) echoes these concerns, stating that a problem of the impulsivity literature is the fact that researchers adopt different definitions of impulsivity. He also argues that psychological diagnoses that focus on the nature of impulsive acts instead of the underlying processes might actually impede the study of impulsivity (Evendon, 1999). One of the few points of agreement in the impulsivity literature is that it appears to be a multidimensional construct (Barratt, 1985; Kindlon, et al., 1995; Evendon, 1999; Moeller, Barratt, Doughtery, Schmitz, & Swann, 2001).

So how has impulsivity been defined? Barratt (1985) developed a three subtrait model of impulsivity based on an item-analysis of self-report questionnaires. According to his findings, impulsivity consists of a motor component related to acting without thinking (e.g., "I do things without thinking"); a cognitive factor that entails making quick decisions (e.g., "I make up my mind quickly"); and a non-planning element that is associated with a lack of future-orientation (e.g., "I am more interested in the present than the future"). Barratt's (1985) non-planning subtrait is similar to Eysenck and Eysenck's (1977) Impulsivity subfactor of Extraversion that involves a lack of thought about consequences of behaviour. Barratt (1985) later translated these subtraits into a self-report questionnaire, the Barratt Impulsiveness Scale (BIS-10). Similarly, Moeller, Barratt, et al., (2001) take a biopsychosocial approach and see the core features of impulsivity as a predisposition for a pattern of behaviour, rapid and unplanned action, and a disregard for consequences.

Other terms associated with impulsivity include risk taking and sensation seeking. Buss and Plomin (1975) suggested that the core of impulsivity is a lack of inhibitory control, which Barkley (1997) identified as a fundamental problem of ADHD, a disorder associated with impulsivity. Along these same lines, Kindlon et al. (1995) focused on the motivational and cognitive domains of impulsivity and how they might be assessed. They saw the motivational component of impulsivity as involving individual differences in sensitivity to reward and punishment and passive avoidance (i.e., learning to inhibit responses that are no longer adaptive), which are very similar to the work of Gray (1970, 1982) and Patterson and Newman (1993). Kindlon et al., (1995) further argued that the cognitive area encompasses inhibitory control processes such as modulation, planning, and the ability to withhold behaviour. This area received considerable attention in the area of ADHD (Schachar & Logan, 1990).

Identifying subtraits/ behaviours/characteristics, etc., is a useful and necessary beginning to the task of differentiating impulsive from non-impulsive individuals. It is, however, just a starting point towards creating a deeper understanding of what drives these differences. Studying the motivational and cognitive domains of impulsivity is appealing because it seems to address the underlying mechanisms and processes that manifest as what are described as impulsive behaviours. To date, however, the study of the motivational and cognitive domains of impulsivity has been limited in the area of PG. An increased understanding of these processes should lead to an enhanced ability to address these areas of difficulty in treatment.

Disinhibition and the Behavioural Activation and Inhibition Systems

As mentioned above, a concept related to impulsivity is disinhibition. Gorenstein and Newman (1980) defined disinhibition as "human behaviour interpreted as arising from lessened controls on response inclinations" (p. 309). The study of disinhibition is rooted in neuropsychology and animal studies and has been useful in understanding areas often associated with impulsivity such as psychopathy, alcoholism, and childhood hyperactivity (Gorenstein & Newman, 1980). Gray (1981, 1987), whose work has been described as "one of the most powerful frameworks for the study of human disinhibition" (Avila, 2001, p.311), proposed the existence of two independent motivational systems to account for human disinhibition: the BIS and the BAS. According to Gray's (1980) theory, the BIS is activated by the presence of conditioned signals of punishment, frustrative nonreward, and novel stimuli. When activated, the BIS stimulates cortical arousal, inhibits ongoing behaviour, and refocuses attention. In other words, it stops behaviour, evaluates the environment and new stimulus, and then the old behaviour is either continued or another is emitted. Gray posited that an underactive BIS is responsible for disinhibition, which he based on countless lesion and anxiolytic drug studies in animals (see Gray, 1982, for a complete review).

Gray also proposed the existence of an opposing system, the BAS, which he hypothesized was responsible for approach behaviour. He suggested that this system is engaged by signals of reward or nonpunishment. When activated, the BAS can lead to increased cortical arousal and movement towards an appetitive stimulus.

The model suggests these systems can operate in one of two different modes: checking and control. In the checking mode, the systems are believed to function

independently with each scanning the environment for stimuli to which to respond. In the control mode, however, the systems are proposed to exhibit reciprocal inhibition. Whether someone is considered high BAS/low BAS or high BIS/low BIS is important in that it is thought to affect the probability that a system will be activated. For example, someone who has highly sensitive BAS is thought to have a greater likelihood of detecting rewarding stimuli than someone who's BAS is less sensitive. Likewise, someone with a sensitive BIS will have a greater probability of detecting a punishing stimulus and inhibiting response to it. Conversely, an underactive BIS would result in less sensitivity to punishment cues (due to decreased ability to learn the associations from punishment), which can then lead to impairments in passive avoidance learning, i.e., learning to "not go there." (Note: for purposes of completeness, this inhibition of responding is in contrast to fleeing from an approaching aversive stimulus, a learned response activated as part of the BAS.)

Development of the BIS and BAS. Gray's model originally began as a modification of Eysenck's theory of introversion (I) and extraversion (E), but over time it developed into an alternative theory of personality (Gray, 1970). Eysenck (1967) proposed that personality could be conceptualized according to two orthogonal dimensions: extraversion and neuroticism. In addition, he postulated that the I/E dimension was a reflection of cortical activity, specifically of the ascending reticular activating system. In his view, introverts were more highly aroused cortically than extraverts, and as a result, were expected to show better conditioning performance since it was assumed that high arousal facilitated conditioning. Consequently, introverts were expected to be more responsive to societal norms, compared to extraverts who were not as fearful of punishment following a social transgression. With respect to neuroticism,

Eysenck (1967) conceived of it as an emotionality or lability factor associated with the autonomic nervous system (as opposed to the motor system which is thought to be under voluntary control) that could serve to either mute or heighten an individual's level of introversion or extraversion.

One method commonly used to test Eysenck's hypotheses about introverts and extraverts involved eye conditioning under various reinforcement schedules, unconditioned stimulus intensities, and conditioned-unconditioned stimulus intervals. In his own experiments, Gray (1970) observed, however, that it tended to be only under certain conditions, specifically those that involved fear or threat, in which introverts conditioned better than extroverts. This led Gray (1970) to suggest that introverts are really more sensitive to punishment and frustrative nonreward than extraverts and *this* is the reason they condition better.

Through a series of animal studies, Gray was able to chemically "create" extraverted behaviour in rats through the administration of sodium amylobarbitone (amytal) (Gray, 1970). He observed that animals became less sensitive to the effects of punishment (Miller, 1959) and frustrative nonreward (Miller, 1964) following administration of a low dose of amytal, while the effects of reward were unaffected. This led to the idea that there are two independent mechanisms in the brain associated with reward and punishment.

Gray soon discovered that lesioning the septal hippocampal system also produced similar behavioural effects to amytal (i.e., reducing the effects of punishment and frustrative nonreward exhibited by impaired passive avoidance learning, extinction and partial reinforcement acquisition). This finding led to a hypothesis that deficits in the behavioural activation system were related to disinhibited behaviour. Simple reward

learning and active avoidance, however, were not affected by these lesions or by administration of amytal. Taken together on the basis of these results, Gray (1970) not only inferred that there are two mechanisms associated with reinforcing stimuli, one mediating reward and relief of punishment, the other mediating punishment and frustrative nonreward stimuli (i.e., the septal hippocampal system), but that the degree of introversion is specifically associated with the latter system (i.e., the septal hippocampal system).

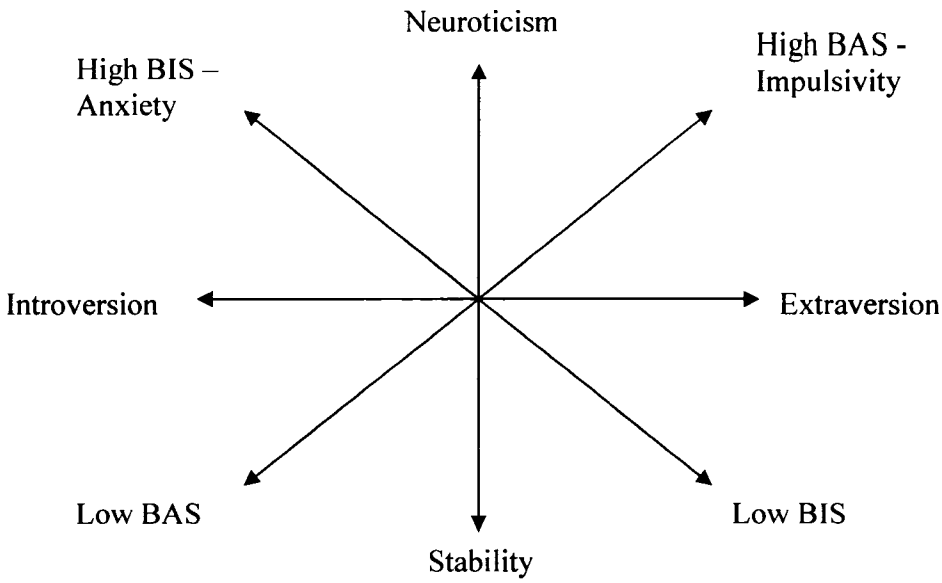
Over time, Gray elaborated on the functioning of the septal hippocampal system, suggesting that it functioned as a comparator that was constantly evaluating incoming stimuli with expected stimuli. He proposed that in the checking mode, behavioural control is left to other brain areas if there is a match between actual and expected stimuli. If there is not, however, he argued that the septal hippocampal system goes into control mode and operates the output mechanisms of the BIS, whereby the behaviour at the time is stopped, there is an analysis of the environment, and a decision is made about how to proceed.

Over time, the "punishment mechanism" was referred to as the BIS and was associated with anxiety and decreased BIS activity was associated with disinhibition and deficits in passive avoidance learning. The other "reward mechanism" initially received less attention, but ultimately became known as the BAS and was associated with reward-oriented behaviour. Also included in this model was a third system, the fight/flight system that is sensitive to conditioned aversive stimuli and mediates rage and panic. Lastly, the non-specific arousal system receives input from both systems and that was thought to invigorate the behavioural output of each of the respective systems. In Gray's

model, arousal is associated with the reticular activating system and serves to increase the speed and vigour of behaviour.

As his theory initially began as a reformulation of Eysenck's theory, Gray (1981) plotted his BIS (which he associated with anxiety) and BAS (associated with impulsivity) constructs onto Eysenck's dimensional factor space (see Figure 1). With the introversion-extraversion dimension lying horizontally and the neuroticism/stability dimension vertically; Gray proposed that his anxiety dimension could best be captured

Figure 1
BIS and BAS in Eysenck Factor Space



running from the stable extravert quadrant to the neurotic introvert quadrant since it would be expected that highly neurotic introverts would be the most sensitive to punishment and conditionable and stable extraverts the least. In contrast, the impulsivity dimension was hypothesized to run from the stable introvert quadrant to the neurotic extravert quadrant, with the neurotic extraverts hypothesized to be the most sensitive to

rewarding stimuli. Gray (1981) postulated that his anxiety and impulsivity dimensions were actually the fundamental factors, or as he said about his anxiety and impulsivity dimensions, they are "the biologically real line of causation" (p. 353), as they resulted from two relatively separate subsystems in the brain. Extroversion and neuroticism were then seen as arising from the interaction of the impulsivity and anxiety dimensions. Gray (1970) viewed the introversion/extroversion dimension as reflecting the relative strength of the BIS and BAS, while the neuroticism dimension was thought to reflect the degree of sensitivity to both reward and punishment.

Originally, anxiety and impulsivity were positioned at 45 degree angles to Eysenck's dimension; however, this positioning has since been revised so that the Anxiety and Neuroticism are angled at 30 degrees. In order to maintain the orthogonality between the anxiety and impulsivity dimensions, Gray's impulsivity was positioned 30 degrees from extraversion (Pickering, Corr, & Gray, 1999).

Alternative Means of Conceptualizing Disinhibition via BIS/BAS

Gorenstein and Newman (1980) were another group of researchers interested in the behavioural effects of septal lesions as a means of understanding disinhibition in humans. They felt that the Septal Syndrome, a set of symptoms/behavioural manifestations that commonly resulted from lesioning the septal area in animal studies, could serve as a useful analogue in providing insight into the psychological processes of disinhibition in human beings. These researchers defined disinhibition as "human behaviour that has been interpreted as arising from lessened controls on response inclinations" (Gorenstein & Newman, 1980, p.302). Gorenstein and Newman (1980) cite numerous dimensions shared by human disinhibition (with psychopathy being considered the prototypical syndrome in humans) and the syndrome resulting from a septum lesion to

support their contention that although not necessarily stemming from the same cause (i.e., lesions to the septum) the two could be thought of as "functionally equivalent hypothetical constructs" (p. 309). Specifically, they note that both psychopaths and animals with septal lesions share similarities in deficits in avoidance learning, anticipation of noxious events, inhibition of appetitive responding, and mediation of temporal intervals (Gorenstein & Newman, 1980). This group focused on passive avoidance learning or withholding an appetitively motivated response in the face of potential punishment, as well as response modulation or the ability to stop what one is doing, shift one's attention from the "organization and implementation of goal-directed responding to its evaluation" (p.717) and then potentially adjusting the behaviour in response to this. Patterson and Newman (1993) further elaborated on the ideas of Gorenstein and Newman (1980) and proposed their own 4-stage theory of disinhibition.[3]

According to this model, in the first stage, a response set to rewarding stimuli is likely to develop for all individuals, regardless of impulsive style, whereby individuals are expected to emit behaviour so long as the opportunity for reward exists. The difference between disinhibited and inhibited individuals is that disinhibited individuals are likely to develop response sets to appetitive stimuli more quickly, easily, and hold on to them more strongly than nondisinhibited individuals. Responding continues until the goal is obtained or it is interrupted. The second stage involves the occurrence of an aversive event that interrupts responding, which results in "an automatic call to process the unexpected event," (Patterson and Newman, 1993, p. 721), as well an increase in arousal results, regardless of BIS or BAS level. The difference, they posit, is in the

[3] For the sake of parsimony, subsequent reference to the response modulation theory will be referred to as Newman's theory.

subsequent behaviour which leads to the third stage. According to Newman (Patterson &Newman, 1993), the resulting increase in arousal allows for an "effortful, adaptive switch to passive, information- gathering set" (p. 721). They state, however, that this might be an area in which disinhibited individuals have difficulty: instead of pausing to reflect, they tend to continue in the same manner. This is in contrast to nondisinhibited individual who might stop to take in the new environmental information. Their final stage discusses an associative deficit that is the consequence of disinhibited individuals' failure to stop and reflect on new environmental information. For these people, the associations between aversive cues and their behaviour do not have the opportunity to form, meaning that future encounters with similarly aversive stimuli will not generate even a pause in behaviour because they are not recognized as possibly dangerous.

To test their theory, Newman (Patterson &Newman, 1993) used cognitive and behavioural measures and found support for their hypothesis in studies of groups characterized by impulsivity, such as extraverts and psychopaths (Patterson, et al., 1987; Newman, 1987). A commonly used paradigm to test reward dominance presents a possible 100 prearranged cards to participants. The participants receive a number of points or sum of money with which they can play. For each face card (i.e., jack, queen king), participants are rewarded; for every number card, they are punished and lose a point or a given amount of money. Initially, the probability of reward is set high, but with each set of trials, the probability of reward decreases, while the probability of punishment increases. Participants are informed that they may stop at any time. Number of trials played is generally the variable of interest and the expectation is that people who are less sensitive to the changing contingencies will play more trials.

Using this paradigm, Newman, Patterson, and Kosson (1987) with a prison sample of 36 psychopaths and 36 controls indeed found that psychopaths chose to play more cards in a reward dominance task and also lost more money in the task compared to a control group. This was interpreted to mean that those who were identified as psychopaths were unable to change a dominant response set as the environmental conditions changed, making their formerly adaptive behaviour maladaptive. Perhaps more interesting, however, was the finding that once a 5-second delay was imposed in conjunction with a visual reminder of the cumulative feedback (i.e., money won, money lost), differences between the psychopathic group and the control group were reduced in both the number of cards played and in the amount of money earned. This finding is consistent with other work suggesting that longer pauses after negative feedback are associated with better modulation of behaviour (O'Brien & Finck, 1996). This is likely because the time delay allows for deactivation of the BAS.

Another paradigm frequently used to test the response modulation hypothesis places emphasis on assessing passive avoidance learning in a situation in which the opportunity for earning rewards and punishments exist. The paradigm involves a go/no-go discrimination task in which participants are required to learn by trial and error learning, which stimuli are "good," resulting in a monetary reward and which are "bad," resulting in a monetary "punishment." Typically, there is one condition in which participants are both rewarded and punished. In other words, there is one condition where participants can either win or lose money. To receive a reward (generally a small monetary amount, such as a dime), participants must either actively respond to a "good" number or withhold responding to a "bad" number. Responding to a "bad" number constitutes making a passive avoidance error. Participants are "punished" (i.e., lose a

dime) if they respond to a "bad" number or if they do not respond to a "good" number, thus making an error of omission. According to Newman's (Patterson &Newman, 1993) conceptualization of disinhibition, it is in situations where both rewards and punishments are available that deficits in response modulation, as expressed by a greater number of passive avoidance errors in this paradigm are expected. This hypothesis has been supported in various studies (e.g., Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985).

In order to demonstrate that it is the presence of *both* reward and punishment that result in an increased number of passive avoidance errors and suggest a deficit in response modulation, two other response contingencies are often administered. In the reward only condition, the only reinforcement participants receive is a reward when they correctly identify a number as "good" and when they correctly withhold a response to a "bad" number; they do not receive a punishment if they make a mistake and respond to a "bad" number or fail to respond to a "good" number. In the punishment only condition, participants can only receive punishments; there is no opportunity for reward. This passive avoidance task was used to examine whether problem gamblers also exhibit impairment in their passive avoidance ability in the presence of rewards and punishments.

Empirical Support of the BIS/BAS

Studies have tested Gray's theory in a variety of ways. Some have demonstrated differential processing of threat-related cues based on anxiety levels. For instance, one study reported that introverted individuals took longer to shift their attention from negative locations, while extraverts were slower to shift their attention from positive locations (Derryberry & Reed, 1994). In another study, individuals who were low in anxiety demonstrated less interference on the emotional Stroop task when threatening

words were used (Mathews & MacLeod, 1994). Slower learning of responses to aversive stimuli by low anxious individuals has also been reported (Corr, et al., 1995; Zinberg & Mohlman, 1998).

Avila (2001) conducted a series of four experiments and overall, results indicated that those who were more sensitive to reward were less cautious when warned of possible punishment cues and exhibited poor inhibitory learning and deficient maintenance of inhibitory learning once acquired. Results also indicated that high sensitivity to reward was associated with faster reaction times in the context of a mixed reward and punishment condition, which was posited to be a contributory factor in response modulation during passive learning. With respect to those low in punishment sensitivity, they extinguished previously punished behaviour faster than those low in punishment sensitivity, as has been reported elsewhere (Avila, 1994; Avila et al. 1999; Newman et al., 1993). Moreover, low sensitivity to punishment was associated with less stimulus generalization and less response suppression of reward-directed behaviour.

Application of the BIS and BAS to other areas of psychopathology

In addition to psychopathy, the BIS/BAS conceptualizations have been applied to a wide range of psychological disorders including anxiety (Turner, Beidal, & Wolff, 1996); depression (Henriques, Glowacki, & Davidson, 1994); and hypomania (Meyer, Johnson, & Carver, 1999). Moreover, the BIS/BAS constructs have been used in the study of Attention Deficit Hyperactivity Disorder, which is relevant to the present study, given the reported comorbidity between gambling and ADHD (Sood, Pallanti, & Hollander, 2003), as well as the findings suggesting deficits common to both disorders (Rugle & Melamed, 1993).

Quay (1988) proposed that ADHD was the result of an underactive BIS, while conduct disorder children tended to be reward or BAS dominant. According to his model, one would then expect for ADHD children to respond more impulsively in punishment only or mixed punishment and reward. Tasks used to test these hypotheses are similar, if not the same as the Newman-based tasks used in the study of psychopathy (i.e., door opening task, card sort). In support of Quay's hypothesis regarding the behaviour of conduct disordered children, Shapiro, Quay, Hogan, and Schwartz (1988) reported that their sample of children with conduct disorder performed similarly to psychopaths and extraverts in a card-sorting task, as they played significantly more cards than a normal comparison group.

With respect to Quay's hypothesis regarding ADHD and low BIS functioning, the supporting evidence is more limited. For instance, Oosterlaan and Sergeant (1998) found in their study involving a reward only and punishment only condition that ADHD children were more impulsive in both conditions compared to a sample of normal control children and that the differing reinforcement contingencies had no differential effect on the impulsivity of the ADHD children. In contrast, Milich, Hartung, Martin, & Haigler (1994) found that ADHD symptoms correlated positively with impulsive responses in a joint reward and punishment condition, but not in the reward only condition. In a study using all three reward/punishment contingencies (i.e., reward-only, punishment-only, and mixed reward and punishment), the ADHD group was found to be more impulsive across all three conditions (Iaboni, Douglas, & Baker, 1995).

Newman's (Newman & Patterson, 1993) response modulation paradigm has also been applied to the study of ADHD. Recently, Gomez (2003) pitted three proposed models of BIS/BAS functioning in ADHD against one another using Newman's go/no-go

discrimination task. The three models included Quay's (1988) proposition that ADHD was characterized by low BIS responsivity, Newman's model which suggests that one of the core deficits in ADHD is poor response modulation in conditions where there is the possibility of either reward or punishment, and lastly the generalized deficit model of ADHD was tested in which it was expected that those with ADHD would demonstrate more impulsive behaviour across all reinforcement conditions. Participants were a sample of boys, who were either considered to be a normal control or who met criteria for ADHD and did not meet criteria for comorbid conduct disorder or an anxiety disorder. Gomez's (2003) findings provided support for the generalized deficit model as the ADHD group was generally more impulsive. In addition, the ADHD group did show the greatest impulsivity on the mixed reward and punishment condition, suggesting that those with ADHD might have difficulty with go/no-go stimuli. No support for Quay's (1988) model was found.

The BIS/BAS concepts have also been applied in the area of alcohol research. A study by Franken (2002) indicated that BAS scores in a sample of inpatient alcoholics predicted strong desires and intentions to drink. Drinking frequency, quantity and variability were not related to desire or intention to drink.

Alternative theories of inhibition and their experimental paradigms

An alternative paradigm used to study inhibitory processes is the stop signal task developed by Logan, Cowan, and Davis (1984). They too, argue that a key feature of impulsivity is increased difficulty inhibiting responses or behaviours The key differentiator is that unlike Newman's paradigm, which is intrinsically motivational because of it dependence on positive reinforcement and punishment, the Logan paradigm

rests simply on the notion that there are individual differences in the time it takes to inhibit or stop an ongoing action or a prepotent action, and that extremes of this difference result in syndromes of disinhibition. The focus of their work has concentrated on inhibitory processes in the context of an ongoing behaviour that is no longer adaptive or appropriate (i.e., braking when a person runs into the road). Like Gray's suggestion of independent BIS and BAS systems, they suggest that response inhibition and "go" behaviour are independent processes that can be conceptualized via a *horse-race model.* According to this model, processes responding to a stimulus race against the processes responding to the stop signal, with the first one to completion being the "winner" (i.e., if the processes responding to the stop signal finish first, the response will be inhibited. Conversely, if the processes responding to the stimulus finish first, the response will be emitted).

The task involves a choice reaction time task that requires participants to distinguish between two letters and respond, with the instruction to respond as quickly and accurately as possible – creating the "go" or primary task process. When a tone is sounded, participants are instructed, however, to withhold their response, initiating the "stop-signal" process. According to the model, a race ensues between the primary task process and the stop signal process, with the winner, resulting in either response inhibition, in the case of the stop signal winning, or the emittance of the behaviour associated with the primary task. Variables that influence the outcome include the latency of response to the "go" signal (go reaction time, RT); latency of the "stop" signal (stop signal reaction time, SSRT); the delay between the onset of the go stimuli and the onset of the stop signal, with a longer delay associated with a greater probability of inhibiting

the response; and the variance of the reaction time to the go task (Schachar & Logan, 1990).

Poor inhibitory control can thus conceivably occur due to an accelerated "go" process in which case the response would be emitted before the stop signal even occurs or before the stop signal was processed. In contrast, poor inhibitory control can also occur due to a slower stop process, resulting in less inhibition of a normally speeded go process. This question was addressed in a sample of children diagnosed with ADHD and in this sample, it appeared that deficits in inhibitory control was due to the latter explanation, slower stop signal response time in the presence of a pre-established response, not an overly speedy go response (Schachar & Logan, 1990; Oosterlaan, Logan, & Sergeant, 1998).

The horse-race model, as captured by the stop signal task, bears similarities to Newman's (Newman & Patterson, 1993) response modulation model of inhibitory function. The first step of this model is the establishment of a dominant goal-directed or approach behaviour. In the stop signal task, the choice reaction task without the stop signal can be thought to act in a similar manner, establishing a dominant behaviour. The stop signal, which serves to interrupt the primary task process, is thought to create an approach-avoidance conflict. Those with an overactive approach system would be expected to have greater difficulty inhibiting a prepotent behaviour. Gray, in contrast, might propose that an underactive BIS would be associated with inhibitory dysregulation.

Avila and Parcet (2001) tested these hypotheses by administering the Eysenck Personality Questionnaire, the Sensitivity to Punishment/Sensitivity to Reward Questionnaire, and the stop signal task to a sample of female undergraduates. As predicted, increased sensitivity to reward and decreased sensitivity to punishment were

had been ongoing since the boys were in kindergarten, completed the Eysenck impulsivity questionnaire, and did so again one year later. Teacher ratings of impulsivity were taken when the boys were 12 and 13 years of age. Two laboratory tasks were administered as well. One of these was a card playing task developed by Newman et al. (1994) to measure response perseveration. This is the same task used in their research with psychopaths. This task was completed by the boys at age 14. The second task, a delay of gratification task, was also one developed by the Newman group (Newman et al., 1992), and administered to the boys when they were 13 years old. This task was developed to measure the ability to inhibit immediate responding to increase the probability of positive reinforcement. At age 17, a measure of gambling behaviour was administered (i.e., the SOGS).

Results of the study indicated that both self-reported impulsivity and perseveration on the card playing task predicted classification of problem gambling at age 17. More specifically, those who scored above the 70th percentile on the Eysenck impulsivity measure at age 14, were 6 times as likely to fall into the gambling group at age 17, while being high on the card playing task increased the risk of becoming a problem gambler by an additional three times. Thus, the combination of a high impulsivity score *and* response perseveration on a card playing task significantly increased the probability that one of these boys would become a problem gambler.

The study findings are consistent with a response modulation deficit as conceptualized by Newman and Wallace (1993) and are the first step in adding PG to the list of disinhibited syndromes. However, neither the study by Breen and Zuckerman (1999) nor Vitaro et al. (1999) explicitly examined PG in Gray's theoretical framework. Do gamblers score higher on measures of reward sensitivity or lower on measures of

punishment sensitivity? Another limitation of these studies is the high ecological validity of the card-playing task (i.e., participants are essentially gambling and they are given the choice whether to continue to or to stop gambling, as in actual gambling). While suggestive of a general response modulation deficit, given that participants are actually betting and gambling, the exhibited deficit might be limited to gambling situations. The go/no-go task, however, which admittedly has less ecological validity, might allow for the assessment of a more generalized deficit as it does not involve betting, but is a task of passive avoidance learning in the context of reward and punishment. The stop-signal task goes one step further in the study of response inhibition as it strips away all explicit rewards and punishments and thus offers another examination of the ability to inhibit a prepotent response when it is no longer adaptive.

Summary of Relevant Research and Rationale for Proposed Study

As discussed, impulsivity can, and has been, conceptualized in numerous ways which are thought to be captured by measures developed to quantify the construct in its various forms. In the area of gambling, impulsivity is presently considered to be, at the very least, a core feature of the disorder (Raylu & Oei, 2002; Wilkie, 2004; Sharpe, 2002). For the most part this conclusion is based on results from studies using a few different self-report questionnaires of personality (Alessi & Petry, 2003). Given the multifaceted nature of impulsivity (Barratt, 1985; Kindlon, et al., 1995; Evendon, 1999; Moeller, et al., 2001), using one measurement strategy is certainly not adequate to fully capture the complexities of the construct (Oas, 1985). It is thus time for those researching gambling to explore other means to provide convergent validity that impulsivity is truly a

core feature of the disorder or to better define what specific types of impulsivity are associated with it.

Gray's model of reward and punishment sensitivity is considered by some to describe a motivational conceptualization of impulsivity (Avila, 2001). According to this model, impulsive or disinhibited behaviour might result if individuals are less sensitive to cues of punishment meaning that there is less to deter them from acting. On the other hand, individuals who are relatively more sensitive to rewards might appear impulsive due to an exaggerated approach response in the presence of appetitive stimuli. Alternatively, Newman (Patterson &Newman, 1993) argues that impulsive behaviour might actually stem from deficits in response modulation, or difficulties disengaging from a prepotent, appetitive response in the presence of both punishment and reward. Personality questionnaires exist to measure individuals' levels of BIS and BAS, such as the BIS/BAS (Carver & White, 1994) and the GRAPES (Ball & Zuckerman, 1990). Cloninger's biosocial model of personality, which attempts to relate reinforcement sensitivity to major dimensions of human personality is thought to be conceptually similar to Gray's and thus his Tridimensional Personality Questionnaire (TPQ) is often used to provide measures of BIS/BAS sensitivity. Specifically, the TPQ Harm Avoidance scale is considered comparable to Gray's BIS. Whether Cloninger's Reward Dependence scale or Novelty Seeking is most comparable to Gray's conceptualization of BAS has been debated, with reports finding support for both (Corr, et al., 1995; Kim & Grant, 2001). Behavioural measures such as the go/no go task (Newman & Patterson, 1993) are also frequently used to examine individuals' ability to modulate responses in the present of various reinforcement contingencies.

Surprisingly, the BIS/BAS constructs and their relation to disinhibition have only been applied in a limited sense to the study of PG. In contrast, research in the areas of alcoholism, ADHD, and substance abuse has applied Gray's model as a means of further understanding these disorders.

Applying the Gray conceptualization of reward and punishment sensitivity to the study of problem gambling would be valuable for several reasons. First, it would more fully explore the motivational component of impulsivity in PG, and it would also provide a more concentrated and focalized examination of the type of impulsivity exhibited in PG. Second, a greater understanding of the mechanisms underlying PG could contribute to alternative ways of treating PG. Third, using the go/no-go (Newman et al., 1985) and the stop-signal tasks (Logan & Cowan, 1984) would allow for more general statements of the disinhibition exhibited by PG than has been previously possible in other studies. Finally, it might serve as a means of explaining the elevated rates of other disorders that share impulsivity as a common feature, such as alcoholism, substance abuse, and cluster B personality disorders in individuals with PG. Perhaps these disorders share a similar diathesis or deficit that manifests itself as impulsivity. Nigg (2000) argues that finding common causal mechanisms is something that should be strived for in the area of clinical research because it would be a basis for a superior taxonomy.

Hypotheses

A. Regarding problem gambling severity

1. Problem gambling severity, as measured by the PGSI, is predicted to be positively correlated with scores on measures of the Behavioural Activation System (i.e., CW-

Drive, CW-Reward Responsiveness, CW-Fun, GRAPES Reward Expectancy, TPQ Novelty Seeking and TPQ Reward Dependence).

2. Problem gambling severity, as measured by the PGSI, is predicted to be negatively correlated with scores on measures of the Behavioural Inhibition System (i.e., CW-BIS, GRAPES Punishment Expectancy, and TPQ Harm Avoidance).
3. Newman (Patterson &Newman, 1993) states that the disinhibitory problems of those with deficits in response modulation are manifested as passive avoidance errors (i.e., errors of commission), in situations where both reward and punishment are present. Accordingly, it was predicted that problem gambling severity would be correlated with errors of commission made in the reward and punishment condition. In addition, Newman's (Patterson &Newman, 1993) model would predict that the effects of disinhibition are not manifested in situations where only reward or only punishment are the contingencies, nor are they manifested by errors of omission. Hence it was predicted that there would be no correlation of problem gambling with commission errors in either of the other two conditions nor with errors of omission in any of the three contingency conditions.
4. Beyond using the CPGI as a continuous variable, it is also used as a set of four categories of gamblers, related ordinally in gambling severity (Ferris & Wynne, 2001). Based on Newman's (Patterson &Newman, 1993) response modulation hypothesis it was predicted that those classified as problem gamblers, based on the CPGI categorization system, would make the most errors of commission, in the reward and punishment condition. In addition, a strong formulation of the Newman (Patterson &Newman, 1993) theory carries an added stipulation that is not fully tested by Hypothesis 3. The stronger version would insist on both error type and response contingency moderating the effect of errors in predicting problem gambling severity, that is, it would insist on a 3 way interaction

between problem gambling, error type, and response contingency, and so we made this prediction. The highest PGSI group should have the most commission errors (relative to omission) in the mixed reward and punishment condition.

B. Regarding BIS and BAS within the entire sample

5. Newman (Patterson &Newman, 1993) theorizes that disinhibition is the result of deficient response modulation. Support for this theory would be reflected in the mixed reward/punishment condition of the go/no-go task, where it would be predicted that those with high scores on BAS measures (i.e., CW-Drive, CW-Reward Responsiveness, CW-Fun, GRAPES Reward Expectancy, TPQ Novelty Seeking and TPQ Reward Dependence), regardless of BIS scores, would make more errors of commission.
6. Gray hypothesizes that disinhibition is the result of low BIS functioning. Support for his theory would be reflected by low scores on measures of BIS (i.e., CW-BIS, GRAPES Punishment Expectancy, TPQ Harm Avoidance) predicting number of errors of commission made in the punishment only condition of the go/no-go task.

Exploratory Questions

As the application of BIS/BAS concepts to problem gambling is relatively new, there were some areas that we were less certain about and thus these questions were approached on a more exploratory basis.

1. Along the lines of the Newman (Patterson &Newman, 1993) response modulation hypothesis, we explored whether the difference between errors of commission and errors of omission made in the mixed reward and punishment condition differed on the basis of problem gambling category. This was done because after examining the passive avoidance data by gambling category, it became apparent that the moderate-risk group

committed the fewest errors, both of commission and omission, regardless of reinforcement condition. Moreover, the problem gambling group committed a high number of errors of commission and omission across reinforcement conditions. This led us to wonder if an unknown variable, such as learning ability or something else, was affecting participants' performance on the passive avoidance task and washing out the effect hypothesized by Newman (Patterson &Newman, 1993). He essentially suggests that disinhibited individuals are more likely to say "yes" in situations in which reward and punishment are present and do not benefit from punishment. Thus, calculating a difference score between types of errors was done to adjust for the effect of this third, "unknown" variable.

2. We also explored the relationship between problem gambling severity and Logan's stop signal paradigm and its related variables. We were not sufficiently confident to predict whether relations between gambling severity and these variables would be to stopping time or to reaction time or to both.
3. If problem gambling severity is associated with slower stop signal reaction times and sensitivity to reward and/or punishment, we were then interested in what best predicted problem gambling severity. Specifically, we tested whether stop signal reaction times from the Stop Signal Paradigm or self-reported BIS/BAS sensitivity (i.e., CW-Drive, CW-Reward Responsiveness, CW-Fun, GRAPES Reward Expectancy, TPQ Novelty Seeking, TPQ Reward Dependence CW-BIS, GRAPES Punishment Expectancy, TPQ Harm Avoidance) best predicted problem gambling severity.
4. Finally, we were interested in determining which variable, of all the variables that positively predicted or were correlated with problem gambling, best predicted problem

gambling severity. This was tested by regressing problem gambling severity on all variables that had shown a significant relationship to problem gambling severity.

CHAPTER II

METHOD

Participants

The final sample consisted of 88 individuals (55 females and 33 males) whose scores on the Problem Gambling Severity Index (PGSI) ranged between 0 and 26 (M = 7.22, SD = 7.14). Participants were recruited over a 4-year period with the majority of the sample (n = 70; 79.5%) coming from an undergraduate participant pool, while the remaining (n = 18; 20.5%) were recruited from community referral sources including clinics, the Salvation Army, and self-help support agencies. This study was part of a larger project consisting of 125 participants that investigated the mechanisms of disinhibition in populations in which impulsivity is a common feature (e.g. ADHD, Borderline Personality Disorder, Bulimia, and Cocaine Abuse). All individuals (except for controls described below) referred from the community were so referred because of their likelihood of having one or another of the disorders characterized by impulsivity (disinhibition). Some of these individuals were referred specifically because the referring agent thought they might have a problem with gambling. However, as might be expected when individuals are recruited on the basis of specific disorders with impulsivity as a common theme, some people referred for an impulsivity disorder other than problem gambling had at least moderately severe problem gambling; and vice versa, others referred for suspected problem gambling had other disorders in the impulsivity spectrum as well. A few (n = 14) individuals were admitted into the larger study not on the basis of impulsivity, but on the basis of having depression only, in order to provide a control

group with a non-impulsivity based disorder. None of these controls was included in the present study. Exclusion criteria were any prior history or diagnosis of schizophrenia or Bipolar I Mood Disorder.

During the first year only people from the community were recruited. Due to the low recruitment rate of individuals with any type of the impulsivity based disorders, the decision was made to recruit from the student participant pool individuals who might meet the criteria of one or more of the disorders being studied. The low recruitment rate of individuals from the community is most likely attributable to the compensation for participation, $60.00, being deemed insufficient by potential participants to warrant their coming to the University for the five-hour protocol. This is a widely known constraint when trying to recruit quasi-clinical samples into a study at a university setting that has no functioning clinic that serves the community. As a result, we began to recruit from the University participant pool. This was done by using the pre-screening mechanisms provided through the pool. Specifically, one or two questions targeting each of the impulsivity disorders relevant to the larger study were used in the pre-screening device that all subject pool applicants complete when signing up for initial participation. The same questions had been posted on fliers in the effort to recruit participants from the community, likely resulting in some (we can't know how many) self referrals from individuals who saw such posters in community clinics. In this way, although participants continued to be recruited in small numbers from the community, participants were also admitted into the larger study from the University participant pool. This means that for approximately the last 1.5 years of recruitment, participants were simultaneously recruited from two streams. The criteria for inclusion, however, were exactly the same in each stream. Since both streams allowed for pre-screening (i.e., referral contingent on a

suspected diagnosis for the community sample, referral contingent on responses to the actual pre-screening questionnaire in the participant pool stream), less than 10% of participants who made initial telephone contact with the lab were turned away from the study.

Successive entries into the larger study were considered for acceptance into this study, that is, all participants who met the above criteria were accepted into the larger study and their data were collected. The additional criteria for inclusion in the present study were engagement in gambling behaviour in the last 12-months and endorsement of at least one of the eight gambling-related questions based on Sullivan's (2001) brief screening questionnaire. Then, these same successive entries were considered for acceptance into this study. However, examination of the stop signal data revealed that nine participants had implausible data. Specifically, they were unable to inhibit a single response over the course of three blocks of trials even when the stop signal was presented almost immediately following the "go" signal. This pattern of failure to inhibit any responding at all strongly suggests some difficulty in understanding the nature of the task. Data from these participants were not used in relevant analyses. Other matters relating to more minor issues with missing data are discussed below.

To increase the variability within the sample, from the larger data set, the first eight successive individuals who did *not* endorse one of the eight gambling related interview questions, but who met the criterion of having gambled at least once in the last 12 months (and who met other criteria for the larger study, i.e., non-schizophrenic, non-Bipolar I, but who passed telephone screen on basis of likely symptoms of some other disinhibitory disorder) were also included in the sample. The admission of these eight individuals increased the likelihood of having at least some individuals in the study who

qualified as non-problem gamblers according to the PGSI (Ferris & Wynne, 2001). Admission of these individuals brought the sample to its final size, N = 88.

The mean age of the sample was 27.24 years (*SD* = 12.46). The majority were single or never married (79.8%), the status of the remainder of the sample was as follows: divorced or separated (9.5%); married or living with a partner (9.6%); widowed (1.2%). The majority endorsed completing some part (65.4%) or the entirety of (21.8%) post secondary school. 6.4% completed only high school, 3.8% completed a post graduate program, and 2.6% completed only some high school or less.

Measures

The *Canadian Problem Gambling Index* (CPGI; Ferris & Wynne, 2001) is a 31-item, researcher administered measure of problem gambling for use in general population surveys. Nine of these questions are used to classify respondents into one of five groups (i.e., non-gambling, non-problem gambling, low-risk gambling, moderate risk gambling, and problem gambling). These nine items constitute the Problem Gambling Severity Index (PGSI), which has good reliability and validity. The Cronbach alpha coefficients for this PGSI component of the index show good internal consistency at .84 (Wynne, 2003).The Pearson Product-Moment coefficients calculated to assess test-retest reliability was .78 (Wynne, 2003). The PGSI has also demonstrated good content validity and good concurrent validity with the DSM-IV criteria items and SOGS (Ferris & Wynne, 2001). Five of these questions address gambling behaviour (e.g., need to gamble to win larger amounts of money to get the same feeling of excitement) and the remaining four examine consequences of gambling (e.g., being criticized for one's betting or gambling). The remaining items in the larger index provide information about gambling involvement,

correlates of problem gambling, such as the social and environmental context of the gambler, and predispositions (i.e., some types of comorbidity).

Passive Avoidance Learning Task (computer-based task) is a successive go/no-go discrimination task based on the work of Newman et al. (1985). The task begins with participants seated in front of the computer monitor where they first verbally receive the task instructions, which are followed by the appearance of a written set of instructions on the screen. Participants begin the passive avoidance learning task with ten dimes.

Once participants indicate an understanding of the task, they are presented with eight random, 2-digit numbers, one at a time, on a computer screen and their task is to learn through trial and error which numbers are "good" numbers and which are "bad." Participants are told that they should respond to the "good" numbers by hitting the space bar. They are also told to withhold responding to the "bad" numbers. Thus, there is only one act that counts as a response, namely, hitting the space bar, and it always means "This is a good number." Failing to respond is equivalent to saying, "bad number", since all numbers are either good or bad.

Participants received feedback about their response (or non-response) after 2500 milliseconds when the word 'Correct!' or 'Incorrect!' appears on the screen. Participants are awarded one dime when they are correct (i.e., responded to a good number, refrained from responding to a 'bad' number) and they lose one dime when they are incorrect (i.e., not responded to a good number or responded to a 'bad' number).

In the second block, participants are presented with a new set of 8 numbers; however, this time, they are only rewarded for responding to a good number and not responding to a bad number – they receive no punishment and do not lose any dimes. In the last block, participants are again presented with a new set of numbers; however, now

they only receive punishment when they are wrong (i.e., fail to respond to a good number or respond to a bad number). Participants could keep their earnings. Theoretically, a participant who was a particularly poor learner or who for some reason was motivated to lose, might have "gone in the hole," but in fact, no participant even needed to have the initial supply of dimes replenished.

Although ideally, contingency type (reward and punishment, reward only, punishment only) would be counterbalanced across subjects, Newman (personal communication to R. Chopra, April 20, 2002) advises against this when the critical experimental condition is the mixed, reward and punishment condition. This is because one wants to observe opportunities for passive avoidance learning deficits (i.e., errors of commission) to emerge only when the ratio of punishments to rewards has been maintained as evenly as possible. The ideal would probably involve making the three contingencies a between-subject condition as well, but that would triple the number of subjects required.

Errors on the task are divided into two categories, errors of commission and errors of omission. Errors of commission occur when participants actively respond to "bad" numbers when they should have withheld a response and thus they are also considered passive avoidance errors. Errors of omission occur when participants failed to respond to a "good" number.

Informed readers will see that the passive avoidance task actually employs concepts from the perception literature. In both of these literatures, trials, items, or scales can be sorted into four groups, based upon two bivariate conditions. In signal detection theory from perception, a signal is either present or not present and the response is either "yes" or "no." In assessment, participants' scores on scales result in hits (true positives),

misses (false negatives), false positives (false alarms, errors of commission), or false negatives (errors of omission). From these four categories can be derived such statistics as *d* prime, beta, sensitivity, specificity, positive predictive power, negative predictive power, and so forth. Critical to Newman's use of these concepts is the notion of a threshold for response, or beta, drawn from signal detection theory. In fact, Newman's idea that disinhibited individuals will make many commission errors in the mixed contingency condition amounts to saying that for these individuals, when their reward (BAS) system becomes strongly activated, their threshold for responding (or their threshold for responding "yes") becomes very low, due to two facts. The first is that their BAS is overly sensitive and very highly activated at these times. The second is due to the fact that they have weaker BIS (punishment sensitive) systems, and so punishment for errors of commission is simply not effective with them at these times.

The *Stop Signal Task* is a choice reaction time task developed by Logan and Cowan (1984) that requires participants to distinguish between two letters and to respond as quickly as possible. Participants were instructed to press the "1" key if an "X" appeared on the screen or "3 if an "O" appeared. A loud, unpleasant noise, the stop-signal, was sounded on one quarter (25%) of the trials. Participants were instructed to withhold their response to the discrimination task if the stop signal occurred.

The entire task was presented on a computer. Participants completed the task while sitting in front of the monitor. Task instructions were conveyed verbally and then appeared in written form on the screen. Before commencing the task, a brief practice period was conducted to ensure that participants had an adequate understanding of the task.

The task consisted of three blocks of trials, separated by a 10-second rest period. To orient the participant, a small cross appeared in the middle of the screen for 500 milliseconds (ms) at the beginning of each block. Following onset of the cross the primary task stimulus (an X or an O) was presented for 5000 ms. After the offset of the primary task stimulus, there was a 2500 ms period prior to the onset of the next primary task stimulus (that is, prior to the next display of an X or an O).

Each block consisted of 144 trials. In 25% (36 trials) of these trials, the stop signal was sounded at some point after the onset of the primary task stimulus. The stop signal was randomly presented at one of six different intervals (with six presentations per block of each interval) after the onset of the primary task stimulus, defined by Logan (1994) as the stimulus-onset asynchrony (SOA) : 5 ms, 100 ms, 200 ms, 300 ms, 400 ms, and 500 ms. The variables of interest for this study are: (1) were participants able to inhibit their responding accordingly (i.e., when the stop signal was sounded); (2) participants' stop signal reaction time, or the amount of time needed to inhibit responding; (3) and participants' reaction time to the primary task (discrimination) stimuli.

Stop signal reaction times consist of the time between start and finish of the stopping process. The SOA marks the beginning of the stop process and the stop is calculated based on the response rate (RR) on the signal trials and the reaction time (RT) distribution on the non-signal trials. For each SOA, a probability of responding is obtained given a stop signal. This variable is known as the response rate and it has a value between 0 and 1. It is assumed that the "go" reaction times do not differ depending on the presence or absence of a stop signal and thus these times (i.e., RTs) are considered the "underlying distribution of go-processes" (Band, van der Molen, & Logan, 2003; p.

109). Overt responses are the result of the go processes finishing before the stop processes. As such, the upper limit of go responses corresponds to the finishing time of the stop processes. Another assumption is that stop SRT is constant, which means that the upper limit of go processes can be derived based on the response rate. Thus, at a given SOA (e.g., 500ms) if RR = x, then the stop processes must have been completed by at point x of the rank-ordered go RTs. For example, if stop signals at SOA = 300 result in a RR = .33, and the 33rd percentile RT of nonsignal trials is 435, then the observed SSRT is 435- 300 = 135 for this particular SOA. The decision was made to use only RR that were corresponded to the middle section of the inhibition function (i.e., $0.15 < RR < .85$) due to its close approximation of a straight line. The outer ends of this function, in contrast, are said to be "shallower" (Band et al., 2003, p. 114) as a result of floor and ceiling effects. For each of the three blocks of signals, an average SSRT was calculated based on the SSRTs resulting from the 6 SOAs (i.e., 5 ms, 100 ms, 200 ms, etc.). It was these values that were used in subsequent analyses.

The *Generalized Reward and Punishment Expectancy Scales* (GRAPES; Ball & Zuckerman, 1990) is a 30-item, true or false self report measure developed to assess respondents' expectations of reinforcement from different life events. The scale is composed of two factors, Reward Expectancy and Punishment Expectancy. The Reward Expectancy scales captures optimism about the occurrence of positive life events and expectations of success and satisfaction (e.g., "If I invested money in stocks I would probably make money"). The Punishment Expectancy scale, on the other hand, taps pessimistic views, mistrust of others and expectations of crime and punishment (e.g., "It is likely that most of us will have a serious car accident at some point in our lives"). The

scales have adequate levels of alpha coefficients of reliability: .63 for the Reward scale and 0.60 for the Punishment scale.

The *Behavioural Inhibition/Behavioural Activation Scales* (CW-BIS/BAS; Carver & White, 1994) is a 20-item, self-report measure that uses a 4-point Likert scale where 1 = strongly agree and 4 = strongly disagree. The scale consists of four subscales, one measuring BIS sensitivity and three tapping BAS reactivity (e.g., BAS Reward Responsiveness, BAS Drive, and BAS fun-seeking). The BIS scale is composed of items asking about potential negative future events and reactions to them (i.e., "I worry about making mistakes" and "Criticism or scolding hurts me quite a bit") and has been found to have an alpha reliability of .74 and a test-rest correlation of .66 (Carver & White, 1994). To reduce ambiguity between the overarching BIS and BAS constructs and Carver and White's subscales, the measure and BIS subscale will be referred to as CW-BIS/BAS or CW-BIS, with the "CW" representing Carver and White. The CW-BAS Reward Responsiveness consists of items about positive reactions to reward (e.g., "When I get something I want, I feel happy and excited" and "It would excite me to win a contest"). CW-BAS drive reflects individuals' appetitive motivation or approach (e.g., "When I want something, I usually go all-out to get"), while CW-BAS Fun-Seeking contains items regarding tendency to look for new and exciting experiences and do them at a moments notice (e.g., "I often act on the spur of the moment" and "I'm always willing to try something new if I think it will be fun"). The CW-BAS subscales alpha reliabilities and test-retest correlations are as follows, CW-BAS Reward Responsiveness, .73 and .59; CW-BAS Drive.76 and .66; and BAS Fun-Seeking, .66 and .69, respectively (Carver & White, 1994). Similar alphas have been subsequently reported (Jorm, et al., 1999).

The *Tridimensional Personality Questionnaire* (TPQ; Cloninger, 1987) is a 100-item self-administered, paper and pencil, true/false instruments. The instrument measures three personality dimensions, Novelty Seeking (TPQ-NS), Harm Avoidance (TPQ-HA), and Reward Dependence (TPQ-RD). Each dimension consists of four, lower-order dimensions. The scale has good reliabilities. Cronbach's alphas have been reported to range between .77 and .85 for TPQ-HA; 0.68 and .75 for TPQ-NS, and .61 and .69 for TPQ-RD (Cloninger, Przybeck, & Svrakic, 1991). As well, the measure is reported to have good temporal stability over six months, with test-retest correlations as follows: .70 for TPQ-RD; .76 for TPQ-NS; and .79 for TPQ-HA. The TPQ-HA scale appears to be a decent reflection of Gray's anxiety dimension (Caseras, et al., 2003), whereas there is debate as to whether TPQ-RD or TPQ-NS fits best with the impulsivity axis. Support exists for both scales (Corr et al., 1995).

Procedure

Participants from the University of Windsor who were specifically recruited on the basis of their gambling behaviour came from a sample of students enrolled in undergraduate psychology courses who responded positively to pre-screening questions on the Psychology Participant Pool survey taken at the beginning of each term (see Appendix C). Students thereby selected on the basis of the pre-screening responses were then contacted by email to inform them about the study (Appendix D). If they responded to this, they were again contacted by telephone. In the case of community participants, they were contacted by telephone following their leaving a telephone message at the lab. This initial telephone message was initiated by either an information

sheet about the study posted on the premises of a referring agency or in a few instances, were provided by a referring community agency employee.

Whether subject pool or community referred participant, during the initial telephone contact, the study was briefly described and participants were informed that they could withdraw from the study at any time without consequence. A brief telephone screening interview (see Appendix E) was conducted to further determine the individual's suitability for the study. The telephone interview was 10-15 minutes in length and assessed for mood disorders, Axis II conditions, substance use/dependence, ADHD, eating disorders, and gambling problems. Those who appeared to have at least one of the impulsivity related disorders, but not more than two additional co-morbid disorders were scheduled to come into the lab at the University and were assigned a number to identify their data while maintaining confidentiality. All other initial telephone interviewees were thanked for their time and informed that they would not be included in the study. In the case of university students, they received one bonus course credit.

Upon arrival to the lab, participants were greeted by a research assistant who provided them with an overview of the study, discussed confidentiality, and had them sign the consent forms (Appendix F). The protocol was divided into three sections, each consisting of an interview, computer tasks, and set of computer-based questionnaires, the order of which were counterbalanced. Upon completion of each section, participants were offered a 10-minute break. At the end of the study, participants were given a list of community mental health resources and the opportunity to request a copy of the final study results. In exchange for participation in the study, university students received course credit and $30 in gift certificates to a local mall or grocery chain. Community participants received $60 in gift certifications as compensation for their time.

Overview of Data Analyses

All analyses were performed using SPSS 10.0 for Windows. Although hypotheses and exploratory analyses were previously outlined, this section is a brief explanation of the statistical analyses used. Prior to testing Hypotheses One, a linear regression analysis was conducted as a means of controlling for the probability of a Type I error occurring as a result of using several different scales to measure BAS. A backward elimination method was chosen as initially all variables are included in the analysis. Compared to other entry options, such as forward selection, an advantage of backward elimination is that those variables which have an *F*-value greater than the removal value (an *F*-value greater than .10, in this study), are not included in subsequent analyses, reducing the number of correlations to ultimately be tested, as well as the probability of a Type I error from occurring (Pedhazur, 1997). Hypothesis One predicted that gambling severity would be positively correlated with measures of BAS. The hypothesis was tested by conducting bivariate correlations between PGSI, the measure of gambling severity, and CW-Drive, CW-Reward Responsiveness, CW-Fun, GRAPES Reward Expectancy, TPQ Novelty Seeking and TPQ Reward Dependence, the measures of BAS.

As with Hypothesis One, a linear regression using backward elimination was conducted to guard against the probability of a Type I error from occurring due to the multiple scales used to measure BIS. Hypothesis Two, predicted a negative correlation between gambling severity and BIS and was also tested using bivariate correlations. Variables included in this analysis were PGSI and CW-BIS, GRAPES Punishment Expectancy, and TPQ Harm Avoidance, the measures of BIS. A linear regression analysis was subsequently performed to test the ability of the BIS/BAS measures to

predict gambling severity. Hypothesis Three predicted a positive correlation between gambling severity and errors of commission made in the reward and punishment condition, and no significant correlation with the other five Newman paradigm error indicators. A bivariate correlation was conducted between PGSI and errors of commission in the reward and punishment condition to test the first step of this hypothesis, and five bivariate correlations were computed to test the second. Hypothesis Four predicted that those categorized as problem gamblers would make the greatest number of errors of commission in the go/no-go task. A three-way mixed ANOVA was conducted to test this hypothesis. The two within subject variables were reinforcement condition (i.e., reward and punishment, reward only, and punishment only) and error type (i.e., errors of commission, errors of omission). The between subjects variable was gambling category (i.e., problem gambler, moderate-risk, low-risk, non-problem gambler). Hypothesis Five predicted that measures of BAS would predict the number of errors of commission committed in the reward and punishment condition. This hypothesis was tested using a linear regression analysis in which the criterion variable was number of errors of commission made in the reward and punishment condition; the predictor variables were CW-Drive, CW-Reward Responsiveness, CW-Fun, GRAPES Reward Expectancy, TPQ Novelty Seeking and TPQ Reward Dependence. For Hypothesis Six, a linear regression analysis was conducted to determine if low BIS scores predicted number of errors of commission on the punishment only condition of the go-no task. The dependent variable was errors of commission made in the punishment only condition and the predictor variables were CW-BIS, GRAPES Punishment Expectancy, and TPQ Harm Avoidance.

Several exploratory questions were addressed. For Exploratory Analysis One, a one-way ANOVA was conducted to determine whether the difference between errors of commission and errors of omission made in the mixed reward and punishment condition (i.e., dependent variable) differed on the basis of problem gambling category (i.e., factor). In Exploratory Analysis Two, bivariate correlations were performed to examine whether stop signal reaction time (i.e., SSRT), a measure of general stopping ability was correlated with gambling severity (i.e., PGSI). Exploratory Analysis Three employed a linear regression analysis including both measures of BIS/BAS sensitivity (i.e., CW-BIS, GRAPES Punishment Expectancy, TPQ Harm Avoidance CW-Drive, CW-Reward Responsiveness, CW-Fun, GRAPES Reward Expectancy, TPQ Novelty Seeking and TPQ Reward Dependence) and stop signal reaction time (i.e., SSRT) to examine if gambling severity (PGSI) is better predicted by reward and/or punishment sensitivity or by slower "stopping" processes in the absence of explicit motivational cues. Finally, Exploratory Analysis Four used a linear regression analysis to determine which of the variables that were significantly related to gambling in this study, best predicted gambling severity. The alpha level was set to 0.05 for the multiple regression equation.

Missing data.

All data involved in the subsequent analyses were examined for normality, outliers, and missing data, etc. Variables of interest were normally distributed. Personality measures and PGSI were centered on their respective means to reduce the multicollinearity between the predictor variables. Examination of the data revealed missing questionnaires for several participants (6% or less of all participants). Specifically, three participants did not complete the TPQ or the Grapes questionnaires;

two did not complete the TPQ; and finally one person did not complete the CW-BIS/BAS. These participants' data were excluded in analyses involving these measures.

CHAPTER III

RESULTS

Demographic Variables

Participants' endorsement of gambling behaviours and consequences experienced due to gambling were consistent with the problem gambling category, mean Problem Gambling Severity Index = 7.22 (*SD* = 7.14). A breakdown of participants by gambling category indicated that 42% of the sample was categorized as problem gamblers, 22% were in the moderate risk category, 14% were in the low risk group, and 22% fell into the non-problem gambling category. According to the PGSI, the first category described is non-problem and includes individuals who did not gamble in the past 12 months. Given the inclusion criteria of engagement in gambling in the past 12 month, 0% of participants fell into this category. As such, subsequent use of the PGSI categories included only four of the five subgroups.

Since participants were recruited from several locations, preliminary analyses were conducted comparing participants from the university sample to participants recruited from the community on various demographic variables. Participants differed significantly in age, t (18.32) = -7.92, $p < .001$, with the undergraduate participants being significantly younger (M = 22.16, SD = 5.05) than the community participants (M = 47.00, SD = 13.07). Not surprisingly, based on the fact that participants who were recruited from the community were recruited from such places as a gambling treatment centre, participants also differed significantly in their gambling severity scores, t (20.53)

= -4.09, $p < .001$. Those from the community sample had mean gambling severity scores that placed them in the problem gambling category ($M = 14.22$, $SD = 8.70$), while the mean severity score for the undergraduate participants fell into the moderate risk category ($M = 5.41$, $SD = 5.44$).

The decision was made not to control for the effects of age. Age was correlated with only one self report measure in the study; Carver and White's BIS/BAS Reward scale, $r = .25$, $p < .05$. Neither the stop-signal nor the passive-avoidance learning variables have been reported to be related to age in adolescent or adult samples (J. Newman, personal communication to Reena Chopra, March 18th, 2006; Tannock, personal communication to Reena Chopra, April 24th, 2006).

Table 1

Raw Descriptive Data of Personality Questionnaires and Computer Tasks

Item	*Mean*	*Standard Deviation*	*Sample Size*
Age	27.24	12.46	88
PGSI score	7.22	7.14	88
CW- BIS	15.72	2.40	87
CW – Drive	9.48	1.63	87
CW – Reward	9.70	1.47	87
CW – Fun	8.83	1.71	87
TPQ – Novelty Seeking	18.81	5.20	83
TPQ – Reward Dependence	14.08	3.81	83
TPQ- Harm Avoidance	16.76	7.67	83
GRAPES – Reward Expectancy	6.86	3.41	84
GRAPES – Punishment Expectancy	8.05	3.15	84
Commission Errors – Reward and Punishment	11.44	5.53	88
Omission Errors – Reward and Punishment	8.13	6.30	88
Commission Errors – Reward Only	12.01	5.69	88
Omission Errors – Reward Only	5.83	4.99	88
Commission Errors - Punishment Only	11.50	5.93	88
Omission Errors Punishment Only	5.44	4.04	88

Item	*Mean*	*Standard Deviation*	*Sample Size*
SSRT (ms) Block 1	279.16	110.54	77
NSRT (ms) Block 1	614.13	186.77	85
NSRT (ms) Block 2	639.69	183.05	85
NSRT (ms) Block 3	647.96	184.11	85
NSRT Across Blocks	633.93	176.49	85

Note. SSRT = Stop Signal Reaction Time; NSRT = Non-Stop Signal Reaction Time.

Gambling Behaviour of Participants

Study participants reported spending an average of 16.50 hours a month engaging in gambling behaviour (*SD* = 26.89 hrs). In the past 12 months, they reported spending an average of $3499.15 (*SD* = $6843.58) on gambling. During that period, they reported winning an average of $2711.70 (*SD* = 5543.02) and losing an average of $3844.19 (*SD* = 9106.17. While independent *t*-tests did not reveal any significant differences between male and female participants on these variables, there were significant differences between participants based on recruitment location. Participants recruited from the community spent more money gambling, t (17.57) = 2.88, $p < .01$; won more, t (20.08) = 2.57, $p = .02$; and lost more money, t (17.17) = 3.45, $p < .01$, in the past 12 months compared to undergraduates in the sample.

The most popular forms of gambling that participants reported engaging in at least once in the past 12 months, as well as the percentage of participants who endorsed

engaging in them were as follows: slots or video lottery terminals in a casino (63%), lottery tickets (59%), scratch tickets (53%), cards or board games not in a casino (44%), casino games (44%), bingo (41%), sports pools (28%). Gambling activities endorsed by fewer than 23% of the sample included: betting on a casino out of province, day trading, gambling with a bookie, internet gambling, arcade or video games, games of skill such bowling or darts, sports select, slots or VLTs not in a casino, and horses. The only differences between male and female participants were that females engaged more frequently than male participants in bingo, t (85.99) = 2.93, $p < .01$, and slots or VLTs, t (78.76) = 2.11, $p < .04$, while males engaged more frequently than female participants in the following gambling activities: gambling on the internet, t (33.97) = -2.84, $p < .01$; arcade or video games (t = -2.62, $p < .02$); games of skill, t (37.38) = -2.48, $p < .01$; cards or board games, t (48.57) = -3.72, $p < .01$); sports pools, t (54.27) = -3.26, $p < .01$; sports select, t (40.35) = -3.10, $p < .01$; casino games, t (49.50) = -5.85, $p < .01$; and betting at casinos not in the Ontario, t (41.85) = -2.11, $p < .04$.

Correlation of BIS/BAS Measures

See Appendix G for all zero-order correlations between personality and computer measures, and gambling severity. To ensure that the subscales within and between measures were consistent with both theory and findings reported in the literature, the correlations between measured subscales and between theoretically similar scales of the three personality measures were examined. The correlations between the CW-BIS/BAS subscales were, for the most part, consistent with theory. Specifically, the CW-BAS scales were all positively and significantly correlated with one another ($p < .01$). The correlation coefficients ranged between .43 and .47. Surprisingly, BAS-Reward and CW-

BIS were significantly correlated, almost to the same degree as the CW-Reward subscale was associated with the other CW-BAS subscales ($r = .38, p < .01$). This finding, although contrary to theoretical expectation, has been reported previously (Smillie, et al., 2006). The correlations between the remaining CW-BAS subscales and the CW-BIS were not significant.

Although a negative correlation was theoretically expected between the GRAPES Reward and Punishment Expectancy scales the results revealed no significant correlation ($p = .69$). None of the TPQ subscales (HA, NS, RD) were significantly correlated with each other.

The correlations between each of the scales measuring BIS (i.e., CW-BIS, TPQ-HA, and GRAPES Punishment Expectancy) were significant and in the predicted direction. Again, because of the reverse scoring of the CW- BIS/BAS measure, the negative correlations between CW-BIS and TPQ-HA and GRAPES Punishment Expectancy were consistent with theory (i.e., a low score on CW-BIS and a high TPQ-HA score both reflect a high sensitivity to punishment). Examination of the correlation between each of the scales measuring BAS indicated that overall, the only one that was not significantly correlated in the expected direction with each of the other BAS scales was the TPQ-RD scale. A review of Appendix G indicates that TPQ-RD is most related to CW- BIS, ($r = -.18, p < .11$), suggesting that this subscale is measuring something different from the other BAS scales. The relation between GRAPES Reward Expectancy and TPQ NS was in the expected direction but was just shy of reaching customary levels of significance ($p = .06$).

Hypothesis 1 - Correlations between BAS measures and Gambling Severity

Hypothesis 1 predicted that PG severity would be positively correlated with scores on measures of the Behavioural Activation System (i.e., CW-FUN, CW-Drive, CW-Reward, and GRAPES Reward Expectancy, TPQ-RD, TPQ-NS). A linear regression using backward elimination was conducted to guard against the probability of a Type I error occurring due to the use of multiple scales to measure BAS. The dependent variable was gambling severity (PGSI) and the predictor variables were the previously described measures of BAS. The model was significant, $R^2 = .10$, $F(1, 79) = 9.01$, $p < .01$, adjusted $R^2 = .09$. Results indicated that the BAS measure that accounted for a significant proportion of the PGSI variance was TPQ-NS, $\beta = .32$, $t = 3.00$, $p < .01$. As a result, only the correlation between PGSI and TPQ-NS was examined, as TPQ-NS was the only variable to fulfill the entry criterion.

Overall, support for Hypothesis 1 was mixed (see Table 2). As might be expected based on the regression analysis, correlations between gambling severity and the BAS measures of the CW-BIS/BAS, GRAPES Reward Expectancy and TPQ-RD were not significant. In contrast, however, there was a positive and significant correlation between gambling severity and TPQ-NS, $r(81) = .32$, $p < .01$.

Table 2

Intercorrelations Between Gambling Severity and BAS measures (N = 81)

BAS Measures	1	2	3	4	5	6	7
1. PGSI	-						
2. CW-Drive	-.10	-					
3. CW-Reward	.01	.47**	-				
4. CW-FUN	-.20	.45**	.47**	-			
5. TPQ-NS	.32**	-.29**	-.17	-.39**	-		
6. TPQ-RD	-.10	-.02	-.11	.07	.08	-	
7. GRAPES-RE	.11	-.44**	-.27*	-.38**	.20	-.06	-

Note. PGSI = Problem Gambling Severity Index; CW – Drive = Carver and White BIS/BAS - Drive; CW Fun = Carver and White BIS/BAS Fun; TPQ-NS = Tridimensional Personality Questionnaire – Novelty Seeking; TPQ-RD = Tridimensional Personality Questionnaire; GRAPES RE = Generalized Reward and Punishment Expectancy Scale – Reward Expectancy.*p < .05. **p < .01.

Hypothesis 2 - Correlations between BIS measures and Gambling Severity

Given the significant "punishments" and losses associated with problem gambling, it was predicted that measures of the behavioural inhibition system (CW-BIS, GRAPES Punishment Expectancy, and TPQ – HA) would be negatively correlated with gambling severity, as measured by the PGSI. Again, a linear regression analysis using backward elimination was conducted to reduce the likelihood of a Type I error from occurring. The dependent variable was PGSI and the predictor variables were the aforementioned BIS scales. Although TPQ-HA *F*-value was below the .10 removal

criterion, the resulting model did not meet the minimum level necessary to be deemed significant, $R^2 = .04$, $F(1, 79) = 3.23$, $p < .08$, adjusted $R^2 = .03$. Accordingly, study results did not support Hypothesis Two as correlations between gambling severity and the BIS measures were not significant (see Table 3). Thus, lower BIS scores or decreased sensitivity to punishment, were not associated with greater gambling severity.

Table 3

Intercorrelations Between Gambling Severity and BIS measures (N = 81)

BIS Measures	1	2	3	4
1. PGSI	-			
2. CW-BIS	-.20	-		
3. TPQ-HA	.20	-.44**	-	
4. GRAPES-PE	.05	-.27*	.44**	-

Note. PGSI = Problem Gambling Severity Index; CW – BIS = Carver and White BIS/BAS BIS; TPQ-HA - Tridimensional Personality Questionnaire – Harm Avoidance; GRAPES PE = Generalized Reward and Punishment Expectancy Scale – Punishment Expectancy. *p < .05. **p < .01.

Predicting gambling severity from BIS/BAS. A post hoc linear regression analysis was conducted to evaluate how well the respective BIS/BAS measures predicted gambling severity. The predictors were the BIS/BAS measures (CW-BIS, CW-BAS-Reward, CW-BAS-Drive, CW-BAS-Fun, TPQ-HA, TPQ-NS, TPQ-RD, GRAPES Reward Expectancy, and GRAPES Punishment Expectancy). The criterion variable was the PGSI. The full model accounted for 28% of the variance in problem gambling severity, $R^2 = .28$, $F(9, 71) = 3.13$, $p = .003$, adjusted $R^2 = .19$. Results indicated that the BIS/BAS measures accounted for a significant proportion of the PGSI variance (see Table

4). TPQ-NS, $\beta = .27$, $t\ (70) = 2.29$, $p = .02$; TPQ-HA, $\beta = .57$, $t\ (70) = 3.61$, $p < .01$; and GRAPES Reward Expectancy, $\beta = -.11$, $t\ (70) = 2.10$, $p = .04$, made significant contributions to predicting PGSI severity.

Table 4

Summary of Regression Analysis for Variables Predicting Gambling Severity (N = 81)

Variable	*B*	*SE B*	*β*
CW - BIS	.26	.37	.09
CW – Drive	-.26	.56	-.06
CW – Reward	.74	.65	.16
CW - Fun	-.90	.56	-.22
TPQ - NS	.36	.16	.27*
TPQ - HA	.52	.14	.57**
TPQ - RD	-.19	.19	-.11
GRAPES RE	.57	.27	.28*
GRAPES PE	-.24	.28	-.11

Note. CW – BIS = Carver and White BIS/BAS BIS; CW – Drive = Carver and White BIS/BAS - Drive; CW Fun = Carver and White BIS/BAS Fun; TPQ-HA - Tridimensional Personality Questionnaire – Harm Avoidance; TPQ-NS = Tridimensional Personality Questionnaire – Novelty Seeking; TPQ-RD = Tridimensional Personality Questionnaire; GRAPES RE = Generalized Reward and Punishment Expectancy Scale – Reward Expectancy; GRAPES PE = Generalized Reward and Punishment Expectancy Scale – Punishment Expectancy. $R^2 = .28$ *$p < .05$, **$p < .01$.

Hypothesis 3 - Correlation between gambling severity and errors of commission made in the reward and punishment condition

To determine whether gambling severity is associated with a greater number of errors of commission in a situation in which both reward and punishment are present, in line with Newman's (Patterson & Newman, 1993) response modulation deficit hypothesis, the correlation between gambling severity and errors of commission made in the reward and punishment response consequence condition of the go/no-go task was examined. This Pearson correlation was significant, r (88) = .25, p = .03. As such, it provides support to the hypothesis that increased gambling severity is associated with passive avoidance deficits in situations in which rewards and punishments are present.

It is possible that gambling severity is correlated with errors of commission and/or errors of omission in other types of response consequence conditions as well. Thus, if we consider the full range of the 2 error types and 3 types of contingencies, there were 5 other possibilities, i.e., correlations of gambling severity with errors of omission in the reward and punishment condition, errors of commission made in the reward only condition, errors of omission in the reward only condition, errors of commission in the punishment only condition and errors of omission in the punishment only condition. All these correlations were examined. A review of Appendix G indicates that gambling severity was not significantly correlated with any other type of error committed in any of the other response consequence conditions. This finding further supports Newman's (Patterson & Newman, 1993) overall view that the basis of disinhibition is a deficit in passive avoidance learning, and that this deficit applies to problem gamblers. However, since the r values relating PGSI scores to errors *other than* commission errors in the

mixed reward and punishment condition do not differ significantly from the significant *r*, we cannot conclude that passive avoidance learning deficits are the *only* causes of PG.

Hypothesis 4 - Examining Go/No-Go data by Gambling Category

On the strong version of Newman's (Patterson & Newman, 1993) deficient response modulation hypothesis of disinhibition, one would expect the highest problem gambling group to make significantly more errors of commission than omission in a situation in which they are presented with the possibility of both reward and punishment, relative to less inhibited individuals, and relative to performance in the other contingency conditions. In other words, the strongest formulation of the theory predicts a 3-way interaction. To investigate this question, a 3-way mixed ANOVA was conducted. The within-subjects factors were response consequence with three levels (mixed reward and punishment, reward only, and punishment only) and error type with two levels (error of commission and error of omission). The between-subjects variable was gambling category, which consisted of four levels (non-problem, low risk, moderate risk, and problem gambler). The Greenhouse-Geisser epsilon was used to adjust the degrees of freedom used to test the significance of the *F*-test as Mauchly's test of sphericity was failed. The Greenhouse-Geisser epsilon was selected due to its conservative nature and appropriateness for small sample sizes. Results indicated that the 3-way interaction was not significant, $F(5.49, 153.66) = 1.47$, $p = .22$. This means that the number of errors made did not differ significantly based on the interaction of error type, response consequence, and gambling type.

There was a significant 2-way interaction between response consequence and error type, F (1.81, 154.08) = 5.71, p = .005, η^2 = .06. Because we were only interested in results involving the between subjects factor (problem gambling severity group), these results are peripheral to the study. Three paired samples t-tests were conducted to follow-up the significant interaction. The differences in mean number of errors made between the two error types were significant between the reward and punishment response consequence and reward only condition, t (87) = 2.74, p = .008 and between reward and punishment response consequence and punishment only, t (87) = 2.69, p = .009. The difference in mean number of errors between the two error types was not significant between the reward only and punishment only consequences. Table 5 shows that although the number of errors of commission remained constant across response consequence condition, more errors of omission were made in the reward and punishment response consequence condition than in either the reward or the punishment only conditions. No other two-way interaction was significant. Thus, errors made did not differ based on the combination of a particular level of gambling category and error type, F (3, 84) = 30.84, p = .49, or gambling category and response consequence, F (5.29, 148.20) = .72, p = .62.

Results indicated that there was a significant main effect of response consequence, F (1.78, 154.40) = 4.64, p = .02, η^2 = .05. The significant main effect for response consequence was analyzed by single degree of freedom "simple" contrasts. Effect sizes were computed as partial η^2 squared values. The contrasts indicated that there were more errors made in the mixed reward and punishment condition (M = 9.78, SE = .44) compared to the number of errors made in the punishment only condition (M = 8.40, SE = .43), F (1, 87) = 7.71, p = .007, η^2 = .08. The difference in number of errors made in the

mixed reward and punishment condition ($M = 9.78$, $SE = .44$) and reward only condition was marginally significant ($M = 8.92$, $SE = .44$), $F(1, 87) = 3.27$, $p < .08$. The main effect for error type was also significant, $F(1, 87) = 95.33$, $p < .001$, $\eta^2 = .52$, with more errors of commission ($M = 11.65$, $SE = .48$) made than errors of omission ($M = 6.47$, $SE = .41$).

Finally, the main effect of the between-subjects variable, problem gambling category, was also significant, $F(3, 84) = 3.21$, $p = .03$, $\eta^2 = .10$. The follow-up tests consisted of all pair wise comparisons among the four types of gamblers. The Tukey HSD procedure was used to control for the Type I error across the pair wise comparisons. The alpha was set at .05. The results of this analysis indicate that overall, problem gamblers made more errors than moderate-risk gamblers (mean difference = 2.41, $SE = .91$, $p < .05$), while low risk gamblers made more errors than moderate risk gamblers (mean difference = 3.27, $SE = 1.19$, $p < .04$).

Table 5

Means and Standard Deviations of Errors Committed by Response Consequence, Error Type and Problem Gambling Category in the Go/No-go Task

	Response Consequence			
	Reward & Punishment	Reward only	Punishment Only	Sample Size
		Errors of Commission		
Problem Gambler	13.11 (6.13)	12.16 (5.84)	12.30 (5.73)	37
Moderate Risk	9.00 (4.57)	10.47 (5.24)	8.89 (4.09)	19
Low Risk	12.50 (3.87)	14.08 (6.71)	13.83 (8.40)	12
Non-Problem	10.05 (5.12)	11.95 (5.12)	11.10 (5.51)	20
		Errors of Omission		
Problem Gambler	7.81 (6.59)	6.05 (5.85)	6.11 (4.11)	37
Moderate Risk	5.63 (4.35)	5.21 (4.72)	3.84 (3.15)	19
Low Risk	11.08 (6.92)	6.50 (4.42)	4.67 (4.19)	12
Non-Problem	9.30 (6.35)	5.60 (3.97)	6.20 (4.32)	20

Note. Numbers in parenthesis indicate standard deviation.

B. BIS/BAS Related Hypotheses

Hypothesis 5 - Testing Newman's Deficient Response Modulation Hypothesis in a Gambling Population

According to Newman's (Patterson & Newman, 1993) deficient response modulation hypothesis of disinhibition, impulsivity is associated with deficits in passive avoidance learning in situations in which both rewards and punishments are present due to heightened reward sensitivity. A linear regression analysis was conducted to evaluate whether high BAS scores predicted number of errors of commission made in the reward and punishment response consequence condition of the go/no-go task. The predictor variables were the self-report BAS measures (CW-BAS-Fun, CW-BAS-Drive, CW-BAS-Reward, GRAPES Reward Expectancy, TPQ-NS and TPQ-RD) and were entered as a block. Errors of commission in the reward and punishment response consequence condition of the go/ no-go task served as the dependent variable. Contrary to prediction, the six BAS scores were not significantly related to the number of errors of commission made in the reward and punishment response consequence condition, $R^2 = .02$, adjusted $R^2 = -.06$, $F(6, 74) = .26$, $p = .95$. None of the BAS measures were significant predictors of the number of errors of commission made in the reward and punishment response consequence condition (see Table 6).

Table 6

Summary of Regression Analysis for Variables Predicting Errors of Commission made in Reward and Punishment Condition (N = 80)

Variable	*B*	*SE B*	*β*	*p*
CW-Drive	-.29	.49	-.09	.55
CW-Reward	-.38	.52	-.10	.47
CW-Fun	.15	.48	.05	.76
TPQ-NS	.04	.14	.03	.80
TPQ-RD	-.05	.17	-.03	.79
GRAPES-RE	-.04	.22	-.03	.84

Note. CW – Drive = Carver and White BIS/BAS - Drive; CW Fun = Carver and White BIS/BAS Fun; TPQTPQ-NS = Tridimensional Personality Questionnaire – Novelty Seeking; TPQ-RD = Tridimensional Personality Questionnaire; GRAPES RE = Generalized Reward and Punishment Expectancy Scale – Reward Expectancy. $R^2 = .02$

Post hoc linear regression analyses were conducted to explore if the BAS scales predicted error type in any of the other response consequence conditions. As above, the number of commission errors or omission errors served as the criterion variable. The predictor variables were the BAS personality measures (CW-BAS-Fun, CW-BAS-Drive, CW-BAS-Reward, GRAPES Reward Expectancy, TPQ-NS and TPQ-RD) and they were entered simultaneously in the equation. Results indicated that none of the BAS measures were significant predictors of either type of error in any of the response consequence

conditions (see Table 7). In other words, none of the BAS nor BIS scales predicted either type of error in any condition in the Newman go/no-go paradigm.

Table 7

Summary of Regression Models Predicting Errors committed in Response Consequence Conditions (N = 80)

Criterion Variable	R^2	Adjusted R^2	F	p
EO – R & P	.08	.01	1.12	.36
EC – R only	.05	-.03	.59	.74
EO – R only	.05	-.03	.59	.74
EC – P only	.04	-.04	.54	.78
EO – P only	.04	-.03	.51	.80

Note. EO – R & P = Errors of Omission made in Reward and Punishment Response Consequence Condition; EC – R only = Errors of Commission made in Reward only Response Consequence Condition; EO – R only = Errors of Omission made in Reward only Response Consequence Condition; EC – P only = Errors of Commission made in Punishment only Response Consequence Condition; EO – P only = Errors of Omission made in Punishment only Response Consequence Condition. Predictor variables were the 6 computed BAS-BIS difference scores. Degrees of freedom for all F tests were 6, 74.

Hypothesis 6 - Testing Gray's hypothesis in a gambling population

It was predicted that those low in BIS functioning would make more errors of commission in the punishment only response consequence condition of the go/no-go task. This hypothesis was evaluated using a linear regression analysis. Predictor variables were BIS scores (i.e., CW-BIS, TPQ – HA, GRAPES-PE). The criterion variable was number

of errors of commission made in the punishment only response consequence condition of the go/no-go task. The results of the regression analysis did not support Gray's hypothesis, adjusted R^2 = -.03, F (3, 77) =.92, p = .44 (see Table 8). Thus, in this study, CW-BIS, GRAPES Punishment Expectancy and TPQ-Harm Avoidance did not significantly predict the number of errors of commission produced in the punishment only condition.

Table 8

Summary of Regression Analysis of Variables Predicting Gambling Severity (N = 81)

Variable	*B*	*SE B*	*β*
CW - BIS	.26	.31	.11
TPQ - HA	.004	.11	.05
GRAPES PE	.32	.24	.17

Note. CW – BIS = Carver and White BIS/BAS BIS; TPQ-HA - Tridimensional Personality Questionnaire – Harm Avoidance; GRAPES PE = Generalized Reward and Punishment Expectancy Scale – Punishment Expectancy.

Five additional post hoc linear regression analyses were conducted to examine if BIS scores predicted number and type of errors made in any of the response consequence conditions. Again, the self-report BIS measures (CW-BIS, GRAPES Punishment Expectancy and TPQ-Harm Avoidance) served as the predictor variables and were entered simultaneously into the equation. The criterion variable was the type of error made in a particular response consequence condition (e.g., errors of commission made in the reward only response consequence condition, errors of omission made in the reward

only response consequence). Results indicated that none of the models were significant, indicating that the BIS measures did not significantly predict the number of errors (see Table 9).

Table 9

Summary of Regression Models Predicting Errors committed in Response Consequence Conditions (N = 80)

Criterion Variable	R^2	Adjusted R^2	F	p
EC – R & P	.04	.003	1.09	.36
EO – R & P	.07	.04	1.99	.12
EC – R only	.03	-.01	.86	.46
EO – R only	.03	-.01	.69	.56
EO – P only	.04	-.001	.98	.41

Note. EO – R & P = Errors of Omission made in Reward and Punishment Response Consequence Condition; EC – R only = Errors of Commission made in Reward only Response Consequence Condition; EO – R only = Errors of Omission made in Reward only Response Consequence Condition; EC – P only = Errors of Commission made in Punishment only Response Consequence Condition; EO – P only = Errors of Omission made in Punishment only Response Consequence Condition. Predictor variables were the 3 BIS measures (Carver and White BIS/BAS BIS; Tridimensional Personality Questionnaire – Harm Avoidance; Generalized Reward and Punishment Expectancy Scale – Punishment Expectancy). Degrees of freedom for all F tests were 6, 74.

Exploratory Analysis 1 - Further Examination of Newman's Hypothesis across Gambling Categories

According to Newman's (Patterson &Newman, 1993) deficient response modulation hypothesis of disinhibition, one might expect that those who are more disinhibited, in this case, those in the problem gambling category, to make more errors of commission than omission in a situation in which they are presented with the possibility of both reward and punishment, but not necessarily in other situations. All else being equal, those individuals characterized by disinhibition are thought to set their threshold for saying "good number" very low once they are rewarded because they want more rewards and they do not benefit from punishment. This would suggest that their errors of commission (false positives) would be high, but the condition should not increase their false negatives (errors of omission). In point of fact, however, once we examined the data in Table 5, it became evident that more than one factor must be having a differential effect on the different PGSI groups error scores, regardless of type of error. Preliminary correlational analyses confirmed that collapsing across groups and collapsing across contingencies, omission errors predicted commission errors, $r = .29$, $p = .003$. This is apparent because the problem gambling group is relatively high on both commission and omission errors. If the high level of commission errors in the problem gambling group were due solely to a very low threshold for "good" number, the same group's errors of omission would not be affected, and if anything, it would be smaller.

Even more striking are the results for the "moderate risk" group. This group has the fewest errors *of both types of errors* and *in every response contingency condition.* Further exploration showed that the moderate risk group in fact had both the fastest

stopping time and the fastest reaction time of all the groups in the stop signal paradigm. Added together, these considerations suggest both that there are one or more determinants of problem gambling severity that are non linear, and that the third, moderate risk group seems to have one or more of these traits to a degree that makes them more healthy. We therefore began to think of whether there may be a different means of testing the Newman (Patterson & Newman, 1993) hypothesis that would adjust for some of these traits. Because the original Hypothesis 4 cast error as a within subject variable, the most straightforward way of making this adjustment would be to use a difference score. That is, a test of whether passive avoidance learning deficits were implicated in problem gambling severity generally could be made by subtracting omission error frequency from commission error frequency, and testing for a linear trend across the groups. This would be the same as testing for linearity in the interaction between error type and problem gambling severity, within the mixed condition.

The hypothesis that PGSI groups could be ordered by this error difference was tested. The independent variable was gambling category, which consisted of four levels: non-problem gambler, low-risk problem gambler, moderate risk problem gambler and problem gambler. These were given contrast weights of -1.5, -.5, .5, and 1.5, respectively, that is, weights for a perfectly linear contrast with monotonically increasing means. The dependent variable was the difference between the number of errors of commission and the number of errors of omission made in the reward and punishment response consequence condition. The ANOVA indicated a significant linear effect $F(1, 84) = 4.34$, $p = .04$, with no support for deviation from linearity. Examination of the errors made by group indicated that the difference between errors of commission and

errors of omission increased monotonically across groups with problem gambling category (see Table 10).

Two additional linear contrasts were conducted, with the independent variable in each being gambling category, and with the same weighting. In each of these contrasts the dependent variable was the difference in the mean number of errors of commission minus errors of omission, but in the reward only response consequence condition for the first, and the punishment only response consequence condition for the second. Neither resulted in a significant linear contrast: reward only condition contrasts, $F(1, 84) = .04, p = .84$; punishment only contrasts, $F(1, 84) = .12, p = .73$. Taken together, these three one-way ANOVAs provide support for Newman's (Patterson & Newman, 1993) deficit response modulation theory of disinhibition in a sample of gamblers. Moreover, this finding also supports the demarcation of gambling categories suggested by the PGSI.

Table 10

Difference Between Commission and Omission Errors by Gambling Category and Response Consequence

Gambling Category	Reward & Punishment Mean	Reward Only Mean	Punishment Only Mean	Sample Size
Non-Problem Gambler	0.75 (9.16)	6.35 (5.99)	4.90 (5.37)	20
Low-Risk Gambler	1.42 (8.23)	7.58 (5.42)	9.17 (9.24)	12
Moderate-Risk Gambler	3.37 (6.85)	5.26 (4.13)	5.05 (4.02)	19
Problem Gambler	5. 30 (8.92)	6.12 (8.44)	6.19 (5.50)	37

Exploratory Analysis 2 - Correlations of Stop-Signal with PGSI score

Correlation coefficients were computed among PGSI score and stop signal and non-signal reaction times. A review of Appendix G indicates that the only correlation to reach significance was between gambling severity and stop signal reaction times in Block 1, r (77) = .31, p = .007. No other correlations with gambling severity were significant.

Exploratory Analysis 3 Regression Analysis - Testing the ability of Stop Signal Reaction Time and BIS/BAS functioning to predict Gambling Severity

Given that there was no overall relation between stop signal reaction time and either PGSI or gambling category, combined with the previously reported associations between PGSI and some of the BIS/BAS measures, the question of whether slower inhibition processes or BIS/BAS functioning are a better predictor of problem gambling severity already appeared to be answered. As such, the planned regression analysis that was to include the following predictor variables: mean stop-signal reaction times for each block (1, 2, and 3), measures of the BAS (i.e., CW-Fun, CW-Drive, CW-Reward, GRAPES Reward Expectancy, TPQ Novelty Seeking, and TPQ-Reward Dependence), measures of BIS (i.e., CW-BIS, GRAPES Punishment Expectancy, and TPQ-Harm Avoidance) was considered unnecessary and redundant and thus was not conducted.

Exploratory Analysis 4- Determining what Best Predicts Gambling Severity.

A final, post hoc, multiple regression analysis was performed to determine which variables were the "best of the best" predictors of problem gambling severity, based on their ability in previous analyses in this study to predict gambling severity or to differentiate between gambling categories. Predictor variables entered into the model included: TPQ - NS, TPQ – HA, GRAPES – Reward Expectancy and the difference score

between errors of commission and errors of omission in the mixed reward and punishment response consequence condition of the go/no-go task. The dependent variable was gambling severity. The full model accounted for 17% of the variance in gambling severity. These predictor variables accounted for a significant proportion of the gambling severity variance, $F(4, 77) = 5.20, p = .001$ (see Table 11). This model indicated that TPQ-NS ($\beta = .32, t = 3.07, p = .003$) and TPQ-HA ($\beta = .24, t = 3.00, p = .004$) made significant contributions to predicting PGSI severity. GRAPES Reward Expectancy ($\beta = .24, t = 1.95, p = .055$) was just shy of customary levels of significance. These findings indicate that both TPQ-NS and TPQ-HA are predictive of gambling severity.

Table 11

Summary of Regression Analysis of Variables Predicting Gambling Severity (N = 81)

Variable	*B*	*SE B*	*β*
TPQ-NS	.43	.14	.32**
TPQ-HA	.34	.11	.38**
GRAPES-RE	.49	.25	.24
EC-EO in R & P	.07	.08	.08

Note: TPQ-NS = Tridimensional Personality Questionnaire – Novelty Seeking; TPQ-HA - Tridimensional Personality Questionnaire – Harm Avoidance; GRAPES RE = Generalized Reward and Punishment Expectancy Scale – Reward Expectancy; EC-EO in R & P = errors of commission minus errors of omission in reward and punishment response consequence condition. $R^2 = .17$. ** $p < .01$.

CHAPTER IV

DISCUSSION

This study explored how disinhibition, via the concepts of the behavioural activation and behavioural inhibition systems, could be applied to the area of problem gambling. The study sample consisted of 88 individuals who reported gambling in the past 12-months. Using both self-report measures that have been associated with BIS and BAS activity (e.g., BIS/BAS, GRAPES, and TPQ) and two computer-based tasks frequently used to study disinhibition (e.g., go/no-go and stop-signal), this study examined whether gamblers can be best understood as (a) having an overactive reward approach system (overactive BAS) that results in an inability to stop a potentially rewarding behaviour in the face of continued loss or punishment; (b) having a weakness in their ability to learn from punishment (underactive BIS); (c) having a deficit in the modulation of their responses when presented with the possibility of reward or punishment, or (d) having slower "stopping" processes regardless of reinforcers.

Support for these hypotheses was mixed. Overall, results of the study provided support for the idea that greater problem gambling severity is associated with greater reward sensitivity as TPQ-NS was significantly correlated with problem gambling severity. An interesting and unexpected finding was that sensitivity to punishment, as operationalized by TPQ-HA, was also predictive of problem gambling severity. In addition, study results provided preliminary support for the classification of problem gambling as a syndrome disinhibition. Consistent with Newman's (Patterson & Newman, 1993) response modulation hypothesis, there was a significant correlation between problem gambling severity and errors of commission in *only* the reward and punishment

condition. Moreover, there was a linear relationship between gambling category and the difference between errors of commission and omission made in the reward and punishment response consequence condition of the go/no-go task. As the group who was expected to be the most disinhibited, those categorized as problem gamblers exhibited the greatest difference between the number of errors of commission and errors of omission committed. The study did not support Gray's (1970) hypothesis that disinhibition is the result of low BIS in a sample of gamblers, nor was there evidence to support the idea that gamblers are better characterized by a general deficit in their ability to stop a prepotent behaviour. Issues pertaining to each finding will be discussed in turn.

Hypothesis 1 – Relation of Gambling Severity with BAS self-report measures

According to Gray, impulsivity is a reflection of BAS sensitivity. He proposed that individuals with highly reactive BASs will have high scores on impulsivity inventories designed to quantify this construct. Given gambling's classification as a disorder of impulse control and its reported association with impulsivity (Raylu & Oei, 2002; Sharpe, 2002), it seemed reasonable to predict that gambling severity would be positively associated with measures of the behavioural activation system. Results of the correlation analyses were mixed. Of the seven BAS scales used in this study, only TPQ-NS was positively and significantly correlated with gambling severity.

Hypothesis 2 – Relation of Gambling Severity with BIS self-report measures

Given that the BIS is theoretically activated in response to punishment, one might expect problem gamblers to be less sensitive to punishment. Thus, it was predicted that measures of BIS (i.e., CW-BIS, GRAPES Punishment Expectancy, and TPQ-HA) would

be negatively related to gambling severity. Results did not support this hypothesis. None of the BIS measures were significantly related, positively or negatively, to gambling severity.

Predicting Gambling Severity based on BIS/BAS measure. As the BIS/BAS components of the three self-report measures tended to hang together in the anticipated manner, that is the BIS components of the CW- BIS/BAS, GRAPES, and TPQ, correlated with one another and the BAS components of the self-report measures correlated with one another, post-hoc analyses were conducted to examine how well these scales predicted problem gambling severity. Surprisingly, given that only TPQ-NS was significantly correlated with gambling severity, three of the nine subscales accounted for a significant portion of the gambling severity variance. Specifically, TPQ-NS, as well as TPQ-HA and GRAPES Reward Expectancy were significant predictors of gambling severity. The ability of TPQ-HA and GRAPES Reward Expectancy to predict gambling severity when included with other scales may have been due to a reduction in the signal noise between TPQ-HA and gambling severity and between GRAPES RE and gambling severity as a result of the redistribution of the variance amongst all the predictor variables.

The ability of TPQ-NS and GRAPES Reward Expectancy to predict gambling severity is not terribly surprising. In a revised version of the Reinforcement Sensitivity Theory, Pickering and Gray (1999) argue that impulsivity is a narrow trait that tends to covary with other traits including novelty seeking and sensation seeking, both of which have been previously associated with problem gambling (Kim & Grant, 2001; Langewisch & Frisch, 1998). As such, these constructs were subsumed under the broad umbrella of Impulsive Sensation Seeking, under the Reinforcement Sensitivity Theory (Pickering & Gray, 1999). These findings lend support to the idea that those with greater

gambling problems have strong responses to rewarding stimuli that activate the BAS. Thus, for gamblers with a more responsive BAS, it might be that the potential riches that could be won in a game of poker or any other gambling activity might be more salient than the losses they have already incurred. As a result, such individuals would be expected to continue playing in the hopes of winning compared to those with a less reactive BAS.

What is surprising in light of the above argument; however, is the ability of TPQ-HA, a measure of BIS reactivity and sensitivity to punishment, to predict gambling severity. As suggested earlier, one might expect that those who are more sensitive to punishment would be less likely to develop a problem gambling disorder because the loss of money would be experienced as more punishing, resulting in a decreased likelihood of continuing the activity. In the present sample, this finding suggests this may not be the case.

One potential explanation for this finding involves further examination of the Harm Avoidance scale. In addition to being associated with a "heritable tendency to respond intensely to signals of aversive stimuli, thereby learning to inhibit behaviour to avoid punishment," (Cloninger, 1987, p. 575) elevations on the Harm Avoidance scale have also been associated with depressive symptomatology (Hansenne, Pitchot, Gonzalez Moreno, Machurot, & Ansseau, 1998). Elevations on other measures of BIS have also been reported in individuals with a lifetime history of depression (Johnson, Turner, & Iwata, 2003). Moreover, Black and Moyer (1998) found that 60% of their samples of individuals categorized as problem gamblers reported a lifetime history of a mood disorder. Recall as well that some have considered PG to be an affective disorder. Taken together, it is possible the elevated levels of Harm Avoidance were a reflection of

depressive levels. Given the significant losses that often result from excessive gambling such as increased rates of bankruptcy claims (Ison, 1995), job loss (Ladouceur et al., 1994), and interpersonal problems (Dickerson, Baron, Hong, & Cottrell, 1996), it would not be unreasonable for those with greater severity of problem gambling to be more depressed.

Although Cloninger (1987) proposed that Harm Avoidance, like Novelty Seeking and Reward Dependence, is a fundamental trait that, by definition, should be fairly stable across an individual's lifetime, subsequent studies have suggested there might be a state-like component to Harm Avoidance. For instance, Chien and Dunner (1996) found that Harm Avoidance decreased significantly in patients whose depression improved, a finding that has been reported elsewhere (Brody et al., 2000). Given the cross-sectional design of the present study, it is impossible to know whether these elevated levels of Harm Avoidance predated individuals' gambling behaviour or were a result of it.

The ability of both a measure of impulsivity (i.e., BAS) and one that has been associated with depression to both predict problem gambling severity might also be understood through the results reported by Clarke (2006). He reported finding in sample of 159 university students that impulsivity was a mediator between depression and problem gambling. One wonders if participants in the Clarke (2006) study had completed the TPQ, if they too, would have had Harm Avoidance and Novelty Seeking scores that were associated with gambling severity. Additional research is required to better understand the relation between gambling, depression, and impulsivity to allow for the development of treatment options that can address these various areas that appear to be related to gambling.

Hypothesis 3 - Correlation between gambling severity and errors of commission made in the reward and punishment condition

Newman (Patterson & Newman, 1993) argued that disinhibited behaviour is the result of deficits in response modulation (i.e., when disinhibited individuals are confronted with situations that involve the possibility of both reward and punishment they tend to focus on the potential reward to the exclusion of punishment cues and continue to respond when it might no longer be in their best interest). Deficits in passive avoidance learning, particularly in those situations that involve both the possibility for reward and punishment, is one way of operationalizing this difficulty. It has been successfully applied to the description of the behaviour of such groups of individuals as psychopaths (Newman, et al., 1992) and extraverts (Newman, et al., 1986). The finding that problem gambling severity was positively correlated with the number of errors of commission (passive avoidance errors) in the mixed reward and punishment condition *only* was consistent with Newman's (Patterson & Newman, 1993) model of disinhibition. As such, it was the first piece of evidence for making the case for the inclusion of problem gambling as a syndrome of disinhibition.

Hypothesis 4 - Examining Go/No-Go data by Gambling Category

To further explore Newman's (Patterson & Newman, 1993) response modulation hypothesis, the number of errors of commission made in the reward and punishment condition were examined by problem gambling category. Here, the expectation was that errors of commission would differ in a linear fashion based on gambling category, with those falling into the problem gambling category expected to make the most errors of commission in comparison to those in a less severe gambling category (i.e., moderate-

risk, low-risk, etc.). A three-way interaction between response consequence condition (i.e., reward and punishment, reward only, and punishment only), error type (i.e., commission and omission) and problem gambling category would have provided support for their hypothesis. This interaction, however, was not significant. In fact, gambling category did not significantly interact with either response consequence condition or error type. One finding of note that resulted from this analysis was that although errors of commission remained relatively constant across response consequence conditions, more errors of omission were made in the reward and punishment condition than in either of the other conditions. This finding might represent an order effect because this condition was presented first as suggested by Newman (Patterson & Newman, 1993). What was more important, however, was that the observation of this finding led to our discovery that commission errors ought to be considered relative to omission errors.

Hypothesis 5 - Testing Newman's Deficient Response Modulation Hypothesis in a Gambling Population

As the application of the BIS/BAS constructs, and by extension Newman's (Patterson & Newman, 1993) response modulation hypothesis, to the study of problem gambling is relatively new, we were interested in whether measures of BAS could predict errors of commission in the reward and punishment response consequence condition. Contrary to prediction, when the CW-BIS/BAS, GRAPES, and TPQ respective BAS scales were regressed onto errors of commission made in the mixed response consequence condition of the go/no-go task, there was no support for this hypothesis. Thus, it did not appear that errors of commission were contingent on the reactivity of individuals' behavioural activation systems.

Hypothesis 6 - Testing Gray's hypothesis in a gambling population

In contrast to Newman (Patterson & Newman, 1993), Gray (1981) proposed that disinhibition and difficulties in passive avoidance learning might be the result of a weak behavioural inhibition system. According to this view, impulsive behaviour is due to the "stop" processes being less sensitive to punishing stimuli, or at the very least, less able to put the brakes on approach behaviour. Given that many gamblers continue to bet despite incurring severe financial loss, not to mention other negative consequences that are commonly associated with gambling such as, loss of jobs, and relationships, it would be reasonable to postulate that gamblers might be less sensitive to cues of punishment. The current study investigated this question by predicting that people with an underactive BIS would make more errors of commission (i.e., passive avoidance errors) in aversive situations. This hypothesis was tested in a group of gamblers by predicting that errors of commission in the punishment only condition of the go/no-go condition would be predicted by low BIS scores. Results did not support this hypothesis. In addition, the relation between number of errors of commission committed in the punishment only condition and gambling category was not significant. Reasons to explain inability of self-report measures to predict behavioural measures will be addressed more fully shortly.

Finding that gambling category was not related to the difference between errors of commissions and errors of omission committed in the punishment only condition is consistent with Newman's (Patterson & Newman, 1993) response modulation hypothesis. According to this view of disinhibition, it is *only* when individuals are presented with both reward and punishment that difficulties occur. Similar findings of disinhibited behaviour occurring only in the context of situations in which both rewards and

punishments are present and not in reward only or punishment only situations have been reported in samples of extraverts (Newman et al., 1985).

Exploratory Analysis 1 - Further Examination of Newman Hypothesis across Gambling Categories

As previously discussed, when the errors of commission and omission were examined by gambling category and across response consequence conditions, two things became apparent. First, and perhaps unexpectedly, those in the moderate-risk group appeared to be the best learners (and perhaps best gamblers) because they made the fewest number of errors (both of commission and omission) in each of the response consequence conditions. In comparison, those in the problem gambling group made the greatest number of errors, both of commission and omission, in the reward and punishment condition only. This suggested to us that some other "unknown variable" was at play, driving these results, and perhaps was masking support for the response modulation hypothesis. In an attempt to adjust for this "unknown" variable, which might have been learning ability, a difference score was calculated between errors of commission and errors of omission.

It was interesting to note that when the difference between the number of errors of commission and errors of omission in the reward and punishment condition was compared across gambling groups, the results were consistent with Newman's (Patterson & Newman, 1993) response modulation deficit model hypothesis. Specifically, participants classified as problem gamblers had greater difference scores between their errors of commission and errors of omission than those in the less pathological categories. This finding, together with finding that problem gambling severity is positively correlated

with errors of commission in the reward and punishment condition, is important for several reasons. First, it suggests a mechanism to explain the disinhibited behaviour exhibited by gamblers beyond the mere assignment of a label. Deficits in response modulation in problem gamblers would mean that they have difficulties stopping appetitively motivated activity (e.g., placing a bet) to evaluate the appropriateness of their behaviour when it may no longer be appropriate (i.e., continuing to place bets in the face of mounting losses). Finding that adult gamblers exhibit deficits in response modulation is consistent with results from a prospective-longitudinal study with adolescents that reported that disinhibited individuals with response modulation deficits were at increased risk for developing problem gambling (Vitaro, et al., 1999). In addition, Breen and Zuckerman, (1999), using a card-sorting task used by Newman et al. (1987) to investigate their response modulation deficit proposal reported that participants who were classified as "chasers" meaning those who continued to play despite continued losing, had significantly higher values on an impulsivity measure. Interestingly, SOGS score was not related to chasing behaviour or even to the decision to gamble in the first place. Taken together, these findings begin to make the case for adding problem gambling to the list of syndromes of disinhibition.

Another reason that finding that difference scores between errors of commission and errors of omission in the reward and punishment condition differed on the basis of which PGSI gambling category individuals fell into is important is because it lends support for the present demarcation of group membership suggested by the CPGI. In particular, it suggests that there is something that differentiates individuals who fall into these groups in a way that extends beyond what is captured in an interview or self-report measure. In this case, it appeared that the ability to withhold a response that was

previously punished in a situation where the possibility for reward also existed differed based on gambling category. For some reason, those classified as problem gamblers by the PGSI had more difficulty in this condition than in those in which only reward or punishment was possible. This "reason" might have been that individuals who were classified as problem gamblers were deficient in their ability to modulate responses, as would be expected by those who have a characteristics of one of the syndromes of disinhibition (psychopathy, extraversion, etc), and were originally described by Gorenstein and Newman (1980).

Findings in the present study make a case for the continued and more widespread use of the CPGI. The CPGI and its measure of gambling severity, the PGSI, is a relatively infrequently used measure that was developed in a Canadian sample for use in general populations, as well as to "reflect a more holistic view of gambling" (Ferris & Wynne, 2001, p. 1). This was done by inquiring about factors related to the social context of gambling, such as the financial impact of gambling on respondents' household and the receipt of criticism for gambling. One of the primary goals of the CPGI's developers was for the measure to be more inclusive than pre-existing measures, such as the SOGS, which was originally developed for use in clinical populations (Lesieur & Blume, 1987). This was attempted by including items that might capture "non-traditional" gamblers, such as women, ethnic minorities and those at the lower end of the socio-economic spectrum. As very few studies have used the PGSI, support for the categorization of gamblers suggests that it is a viable alternative to the SOGS as a means of classifying problem gamblers. An additional benefit of this measure is the wealth of information that it provides about gamblers, above and beyond diagnostic criteria or the consequences resulting from gambling behaviour. In addition, it collects information regarding

gamblers' gambling preferences, their substance use history, as well as motivations driving their behaviours, such as why they go to casinos in the first place.

Exploratory Analysis 2 - Correlations of Stop-Signal with PGSI score

Thus far, the hypotheses discussed have involved a motivational component in the form of either reward or punishment. It might be, however, that disinhibition in problem gamblers is not related to reward or punishment sensitivities, but rather to a more general deficit in their ability to stop an ongoing behaviour. Previously a link between problem gambling and ADHD was suggested by Rugle and Melamed (1993), who reported that gamblers endorsed a greater number and intensity of childhood behaviours associated with ADHD than a control group. Moreover, studies employing neuropsychological and physiological tests have reported that problem gamblers exhibit deficits similar to those diagnosed with ADHD (Carlton et al., 1987; Goldstein et al., 1985). As slower stop signal reaction times have been frequently observed in people diagnosed with ADHD, this study examined whether problem gamblers too, exhibited slower stop signal reaction times and whether this type of disinhibition was more associated with problem gambling severity than measures involving a motivational component.

The preliminary examination of the stop signal data indicated that while stop signal reaction time was positively correlated with gambling severity in the first block, the association dropped away with time. Ultimately, when the stop signal reaction times were averaged over the three blocks of trials, there was no correlation between gambling severity and the time it took for participants to stop their prepotent behaviour. This finding might be explained two ways. One, the association between stop signal reaction time and gambling severity might have been an artefact. Consistent with this explanation,

is a study that suggested that the first block of trials is often unreliable (Nigg, 1999). The second possible explanation is that over time (blocks), a fatigue component set in whereby those with lower gambling severity scores slowed down to the level exhibited by participants with higher scores. This explanation is less likely since one would not expect fatigue to differentially affect participants on the basis of gambling severity. These findings suggest that gambling severity is likely not associated with a deficit in more global stopping processes, as captured by stop signal reaction time.

Based on the reported similarities between gamblers and people with ADHD, these findings might appear somewhat curious. A review of the studies that have reported such differences reveal two potential reasons why similar results in this study were not obtained. First, several of the studies that have reported characteristics of ADHD in gamblers relied on retrospective self-report measures to assess childhood behaviours. Such measures are open to a whole host of biases (i.e., biases based on current state, inaccuracies, etc.). Moreover, the sample sizes that reported these differences tended to be rather small. For instance, Carlton and colleagues (1987) compared only 14 pathological gamblers to a control group of 16 individuals, while Goldstein and colleagues (1985) compared 8 individuals with a history of problem gambling to a sample of 8 matched controls. Thus, it is possible that the findings reported in these studies might have been subject to a Type I error and would not be replicable if repeated. Alternatively, the finding that stop signal reaction times were not associated with gambling category nor with gambling severity, might reflect the reality that indeed, gamblers do not have a general deficit in their ability to "stop" in the absence of explicit reinforcers.

Exploratory Analysis 3 Regression Analysis - Testing the ability of Stop Signal Reaction Time and BIS/BAS functioning to predict Gambling Severity

As mentioned in the results section, this analysis originally sought to explore whether a more general deficit in the ability to stop an ongoing behaviour in the absence of explicit reward and punishments or sensitivity to rewards and/or punishment predicted problem gambling severity was not conducted due to the previously reported lack of correlation between stop signal reaction time and problem gambling severity.

To date, very few studies have applied the concepts of behavioural activation and inhibition to the area of problem gambling. This may in part be due to the continued debate regarding how best to operationalize and quantify these constructs that were initially tested in animal models. As such, the fact that the hypotheses employing self-report measures to predict behavioural measures were largely unsupported is not entirely surprising nor necessarily a complete refutation of the hypotheses. Rather, what these findings speak to is the difficulty involved when using measures of a particular construct in one modality to predict a seemingly similar construct in another modality. This has been a problem previously expressed (Reynolds, Ortengren, Richards, & deWit, 2006).

This difficulty can be better understood by examining some of the common differences that often exist between self report and behavioural measures. First, self report measures require respondents to reflect on their own behaviour and then determine how to best capture it within the survey's rating system, which often tend to offer little anchoring other than by way of labels such as "not at all," "somewhat," "a lot." Error and variance are introduced at each of these steps as people vary in their level of self-awareness and judgment regarding what constitutes "a lot" or "a little" of something

depending on their environment, learning history, etc. In contrast, the behavioural measures might be considered more "objective" because they avoid the bias of self-perception.

Discrepancies between self-report and behavioural measures might also stem from the breadth of the concept measured. Often times, behavioural measures capture one specific dimension of behaviour (i.e., number of errors made in a response to different response consequences); whereas self-report measures typically focus on a wider scope. If this were the case in this study, then it is not surprising that the self-report measures did not predict behaviour on the computer tasks.

Exploratory Analysis 4- Determining what Best Predicts Gambling Severity

The final exploratory analysis was interested in determining which of all the variables that either predicted problem gambling severity or differed based on gambling category, was the best at predicting gambling severity. The best predictors of gambling severity were TPQ Novelty Seeking and Harm Avoidance, where high scores on each were associated with greater gambling severity. This is an interesting state of affairs since Novelty Seeking is considered a measure of BAS sensitivity and Harm Avoidance is a measure of BIS sensitivity. Reasons why this might have occurred were previously discussed.

Summary

One of the primary questions addressed in this study was: Can problem gambling be added to the list of syndromes of disinhibition that already includes psychopathy, hysteria, hyperactive children, and non-pathological impulsive personalities (Patterson & Newman, 1993). The results of this study provide preliminary evidence for its inclusion

to this list. First, consistent with a previous study (Vitaro, et al., 1999), there was evidence to suggest that individuals categorized as problem gamblers have a deficiency in their ability to modulate responses in situations in which both cues of punishment and reward are present. Similar findings have been reported in other samples of individuals characterized by disinhibition including, extraverts, psychopaths, and children with ADHD (Patterson & Newman, 1993). This finding opens the door to such questions as; do problem gamblers react more quickly following punishing stimuli, as other disinhibited individuals have been found to do? If this is the case, it might lead to implications for treatment. For instance, it has been reported that the response modulation differences between extraverts and introverts disappear once the delay between punishment and a subsequent response is increased (Newman et al., 1985). Applying this to the treatment of problem gamblers might involve encouraging gamblers to sit out a round after losing a hand to allow time to process the negative consequence of their behaviour.

Another important finding resulting from this study was support for the PGSI's present demarcation of problem gambling categories. Finding a linear effect for the difference between errors of commission and errors of omission by problem gambling category in only the reward and punishment category provides objective evidence that problem gamblers differ in some fundamental way from those with less severe forms of gambling. Specifically, it means that there might be a biologically based reason that differentiates those people who become categorized as problem gamblers from those who fall into a less severe category.

Study Limitations

Limitations that curtail the conclusions that can be drawn from this study include: participant recruitment and associated issues, as well as failure to ensure that the motivation manipulation in the go/no-go task was in fact experienced as rewarding. First, difficulties recruiting participants from the community within a reasonable amount of time necessitated recruiting participants from a university sample. While a greater sample size was achieved, group differences were introduced such as levels of education and age. In an effort to further increase the sample size of the study, participants who were recruited into the larger study on the basis of having another disorder characterized by impulsivity, but who also endorsed gambling in the last 12 months, were included. Inclusion of these individuals might have "muddied the waters," so to speak, and introduced greater heterogeneity into the sample. While this might have compromised the internal validity of the findings, it had the opposite effect on the external validity of the study and increased the study's generalizability, since problem gambling rarely occurs in the absence of other difficulties. One of the most salient differences that was introduced by recruiting from both a university and community sample occurred in the form of background variables. Those who were recruited from the university population likely brought with them a set of background variables that differed appreciably from the community sample, who could have been considered a clinical sample.

Although every effort was made to recruit participants to increase the sample size, a larger sample would have been preferred to increase the power of the statistical analyses. Had the study had more statistical power, results that were just shy of reaching

customary levels of significance, such as the correlations between gambling severity and Harm Avoidance and CW-Fun, might have become significant.

The final limitation of the study involved the go/no-go task. Recently, Corr (2001) suggested that studies employing Gray's model to examine reactions to appetitive and aversive situations need to assess levels of subjective reward to ensure that manipulations of motivation (in particular appetitive; see Corr, 2002b) are effective. A potential shortcoming of this study is that participants were not asked how rewarding they found the motivation manipulation in the go-no-go task to ensure that participants did indeed experience the reinforcement manipulation as motivating (Corr, 2001) This may have been less of an issue because for many, it seemed as though the manipulation was effective based on participants' behavioural reactions to whether their responses were rewarded or punished.

Future Directions

As with most research, answering one question tends to lead to many more. First, there was evidence to support the notion that problem gambling could be predicted on the basis of BAS sensitivity. In contrast to expectation, BIS was also able to predict gambling severity, with higher, rather than lower BIS scores on TPQ harm avoidance scale associated with gambling severity. Future work should seek to replicate this finding to ensure that it was not an artefact of this study. In addition, future studies might want to tease apart the contribution of depressive tendencies from true sensitivity to punishment to further investigate the ability of TPQ-HA to predict gambling severity. To this end, it might also be important to gather information about the chronology of gamblers'

depressive experiences (i.e., did they predate their difficulties with gambling or were they a result of it).

Finally, there was support for a response modulation deficit in those categorized as problem gamblers and not a more general deficit in stopping processes. Given the substantial economic and personal burden of problem gambling, further research into factors that speak to the mechanisms that drive people to gamble to the point where it becomes problematic are essential to the development of treatments that work. Learning more about the situations and contexts in which problem gambling develops should in turn lead to treatments that specifically target these issues, and as a result, be more effective. For instance, future work should investigate whether the deficits in passive avoidance learning in the mixed reward and punishment condition exhibited by those in the problem gambling category would disappear if study participants were forced to wait before responding to the next trial, a finding that has been reported in a group of extraverts (Patterson, et al., 1987). Were this the case, one could imagine customizing treatment of gamblers based on this finding. For instance, the treatment of those who exhibit deficits in response modulation could include instituting some sort of break after a loss was incurred. As the treatment of pathological gamblers is still in the very early stages of development (Ladouceur & Shaffer, 2002), the more that it is learned about the mechanisms that lead to the development of problem gambling, the better able clinicians will to be to "ante up" and provide treatments that work.

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APPENDIX A

Participant Recruitment Information Sheet
(On University of Windsor Letterhead)
PSYCHOLOGICAL DISINHIBITION MECHANISMS
Principal Investigator: Stephen Hibbard, Ph.D. Department of Psychology,
University of Windsor: 519 253-3000 ext. 2248

Disinhibition mechanisms are psychological or brain processes that lead people to do things that normally they would not do or that may be harmful to themselves or to others. In recent years, researchers have identified some good methods of studying these processes. It is believed that problems in these areas are partly responsible for some kinds of emotional problems or difficulties in living that some people have. Often, these people are given quite different psychiatric "labels". Therefore, we are asking various individuals to come to our lab to participate in a study of disinhibition mechanisms. Disinhibition refers to the fact that some people have a hard time stopping themselves from doing things they don't want to do or that they later regret. People with different emotional make-ups are being solicited for the study.

The study is being conducted at the University of Windsor. Various referral sources, including the person who gave you this sheet, have volunteered to help us find people who might be suitable for this study. People are coming from different clinics, from the University, and from the general population. If you participate, you would be asked to contribute 5 hours of your time on one occasion at our lab in Chrysler Hall on the Windsor campus. You will be compensated $60.00 in either gift certificates for the mall, or grocery store. You will do tasks that study your reaction time and your decision processes. You will also be administered a diagnostic interview. No medicines are administered. No wires are attached to you, nor are any physical procedures involved. You will also fill out questions regarding personality and emotions, which you may or may not have. People of various backgrounds are participating in this study. The results will be entirely confidential within ethical and legal limits. No one at the University (except the researchers) will have any idea how you were referred to the study or why you are there except to participate in some research. By the same token, no one who may have referred you to the study will get feedback or information about you that you have told to the researchers (unless you tell the researchers something they are legally required to follow up on, such as child abuse or the intention to commit suicide). They will not know whether or not you have participated in the study.

If you would like further information about participating please call the research team at 519 253-3000 ext. 2250. If your call is not answered immediately, please leave a number and a convenient time to reach you. Your call will be treated completely confidentially. There is a telephone screening process that will take 10 to 15 minutes. After that call, if you are still interested and if you meet the needs of the study, you will be asked to come to the University for the 5 hour period. If you are interested, just call the following number: 519 253-3000, ext. 2250. Please realize some people who call will not be able to participate because they may not fit the exact needs of the research.

APPENDIX B

Mechanisms of Impulsivity Recruitment Poster for Problem Gamblers

UNIVERSITY of WINDSOR
ONTARIO ♦ CANADA

Primary Investigator: Dr. Stephen Hibbard, Department of Psychology

Interested in Research?

Have you ever:

- **Felt depressed or anxious after you gamble?**
- **Felt guilty about gambling?**
- **Had problems because of your gambling?**
- **Hidden your gambling from family/friends?**
- **Been criticized about your gambling?**
- **Gambled to win back past losses?**
- **Gambled to pay of your debts?**
- **Only stopped gambling because you ran out of money?**

If you said yes to most or all of these questions and are interested in being a research participant, please call 253-3000, ext. 2250

compensation for your time is provided □

APPENDIX C
Student Recruitment Questions

In the past 12 months, have you engaged in any form of gambling (e.g., going to the casino, buying lottery tickets, playing bingo, etc)? Y/N

In the past 12 months, have you thought that you might have a problem with gambling or been told by others that you might have a problem with gambling or gaming? Y/N

APPENDIX D

Email to Undergraduates

Bonus Points and Cash Opportunity

Hi! Your name was generated from a list of people who registered for the Psychology Research Participant Pool. We are the Impulsivity Research Group, lead by Dr. Stephen Hibbard, and we are conducting a study looking at different mechanisms of disinhibition, which in other words, means the ways in which people have trouble stopping themselves from doing things they do not really want to do, or at least before they are ready.

What do I have to do?

(a) Complete a 10-15 minute phone interview.
(b) If you're a good match for the study, you'll come into the research lab, 283-3 in Chrysler Hall South, where you will spend about 4.5 - 5 hours doing the following:

i. Complete some interview questions about emotional and diagnostic issues that you may or may not have.
ii. Complete 3 computer tasks, on one of which you could win a small amount of cash (less than $10).
iii. Complete personality and emotional problems questionnaires.

What do I get out of this? If, after the telephone interview, we don't think you'd be a good match for the study, you'll get one bonus point. If you are a good match, you will receive 3 bonus points and $30 in Devonshire mall gift certificates, in addition to any money you win on the computer task. During the two breaks when you come into the lab, we supply snacks and juice.

Potential Risks: Nothing is done to people physically in this study. Some of the questions that are asked might bring up feelings that are scary, sad, or otherwise uncomfortable for you if they remind you of any emotional difficulties you might have.

Potential Benefits: The compensation you receive (3 bonus points and $30 in gift certificates); potential interest in taking part in a research study; taking part in a study that will likely be of benefit to researchers who try to understand the relationship of disinhibition to emotional problems.

Ok, I'm interested, what do I do now? Respond to this email in the next few days, stating what day and time of day is best to reach you to do the telephone interview and we'll do our best to accommodate it. You can also leave a voice message at 253-3000, ext. 2250 stating your name and the day and time that it is best to reach you.

APPENDIX E

Mechanisms of Impulsivity Telephone Screen

Participant ID number (to be assigned at lab appt): _____

Rapport: (This element can be phrased in any way the research assistant finds comfortable and accommodating to the needs of the caller and researcher.) Thanks very much for calling. I hope you didn't have a hard time reaching us. I'm so glad we could have a chance to talk. You know that we are going to be collecting some data on processes of disinhibition. Can I ask how you were referred to the study?

REFERRAL SOURCE: _____
(if from poster, where did they see/get it? _____)

Most likely group into which this person will be recruited: _____

Consent to diagnostic aspects of phone interview: I am going to need to ask you some specific questions about problems you may or may not have had in the past, or may currently have. Many of these questions are about people with various kinds of emotional problems and so they may make you feel uncomfortable to a certain extent. Of course, you don't have to answer these questions, but in order to determine whether you are suitable for our study, I need to ask them. If you don't want to proceed, this will in no way jeopardize any treatment you might be getting from the people who referred you. It's just that I need to ask you the questions and some people get uncomfortable about being asked questions about their emotional life. Is that going to be alright with you? (If the person indicates that it is alright to proceed then do so. The interviewer is at liberty to field further questions from the potential participant at this point about whether there are any penalties for not participating, how long it will take, etc.)

Indicate: YES, the interviewee consents _____
NO, the interviewee declines further participation _____

Any notes relevant to informed consent:

_____

_____

Let's get started. Remember, if by any chance you become so uncomfortable that you need to talk about it, just let me know. If you feel it's necessary to do so, we can call the whole thing off at any time and there will be no penalty to you.

Diagnostic Portion of the Interview: (Based on Structured Clinical Interview for DSM – IV Axis I Disorders, Clinician Version; First, Spitzer, Gibbon, & Williams, 1997)

Circle '1' if there is no indication of a problem
Circle '2' if unsure or if there is some indication of a problem
Circle '3' if it is likely that there is a problem or definitely a problem

1. a) How old are you? _____ What is your date of birth? _____

1. b) Have you ever been diagnosed by a physician, therapist, psychiatrist or psychologist? _____

1. c) Have you been given any other diagnoses? _____

1. d) Have you ever had a head injury? _____

Depression

2. a) Has there ever been a time in your life when you were feeling depressed or down most of the day, nearly every day? 1 2 3

2. b) IF YES: What was that like? _____
(check if they mention any of the following symptoms)
_____ subjective report (i.e., feeling sad or empty)
_____ objective report (i.e., others say I appear tearful)
_____ low energy
_____ hyper/insomnia
_____ excessive guilt/worthlessness

2. c) How long did that last? _____ _____ check if at least 2 weeks

2. d) Has there ever been a period of time in your life when you lost interest or pleasure in things you usually enjoyed? 1 2 3
_____

2. e) How long did it last? _____ _____ check if at least 2 weeks

2. f) If there is indication of depressive episode:
How many separate times in your life have you been depressed (USE OWN WORDS) nearly everyday for at least two weeks?
_____ number of episodes

2. g) In the last month have been feeling depressed? _____
Are you currently depressed? _____

Bipolar Disorder

3. a) Has there ever been a time in your life when you were feeling so good, high, excited, or hyper that other people thought you

were not your normal self or you were so hyper you got into trouble? (did anyone say you were manic?) (was that more than just feeling good?) _____ 1 2 3

3. b) IF NO: What about a period of time when you were feeling so irritable that you found yourself shouting at people or starting fights or arguments? 1 2 3

3. c) IF YES (to either 3a or 3b): How long did (USE OWN WORDS) last?

_____

If at least 1 week check here _____
Did you have to go into hospital? _____

Substance Abuse

4. a) Are you taking an medications or vitamins? _____
4. b) IF YES: What medications? (get specific names)
How often do you take them>
What dosages? (if unable to remember dosage, ask to write down to bring in on day of testing)

_____
_____
_____

4. c) Has there been any time in your life when you had five or more drinks (beer, wine, or liquor) on one occasion? 1 2 3

4. d) Have you ever been told that you have a drinking problem? _____

4. e) IF YES: By whom? _____
How long have you been drinking? _____
What do you usually drink? _____
How much do you usually drink during one session? _____
Do you ever drink more than you planned? _____

4. f) Have you ever used street drugs? 1 2 3

4. g) Have you ever been told that you have drug problem? _____

4. h) IF YES: By whom? _____
How long have you been doing drugs? _____
What drugs do/did you usually take? _____

4. i) Have you ever gotten 'hooked' on a prescribed medicine or taken a lot more of it than you were supposed to? 1 2 3

4. j) IF YES: What drugs do/did you usually take? _____

How much do/did you usually take? _____

Anxiety

5. a) Have you ever had a panic attack, when you suddenly felt frightened or anxious or suddenly developed a lot of physical symptoms? 1 2 3

5. b) Were you ever afraid of going out of the house alone, being in crowds, standing in a line, or traveling on buses or trains? 1 2 3

5. c) Is there anything that you have been afraid to do like speaking, eating or writing? 1 2 3

5. d) Have you ever been bothered by thoughts that didn't make any sense and kept coming back to you even when you tried not to have them? 1 2 3

5. e) IF YES: What were they? _____
When you had these thoughts, did you try hard to get them out of your head? _____
What did you do to try and stop them? _____
_____

5. f) Was there ever anything that you had to do over and over again and couldn't resist doing, like washing your hands again and again, counting up to a certain number, or checking something several times to make sure that you'd done it right? 1 2 3

5. g) IF YES: What did you do? _____
Why did you have to do it? _____
What would happen if you didn't do it? _____

5. h) In the last six months, have you been particularly nervous or anxious? 1 2 3

Eating Disorders

6. a) Have you ever had a time when you weighed much less than other people thought you ought to weigh? 1 2 3

6. b) IF YES: How much did you weigh? _____
How old/tall were you? _____
Were you trying to lose weight because you thought you were fat?
_____

6. c) Have you often had times when your eating was out of control? 1 2 3

6. d) IF YES: During these times, do you often eat within a two hour time period what most people would regard as an unusual amount? (Tell me about it) _____

_____

_____

Did you do anything to counteract the effects of eating that much? What was it? _____

_____

_____

Eight Gambling Screen

7. a) Have you ever felt depressed or anxious after a session of gambling? NO YES

7. b) Have you ever felt guilty about the way you gamble? NO YES

7. c) Has gambling ever caused you problems? NO YES

7. d) Have you found it better to not tell others, especially your family about the amount of time or money you spend gambling? NO YES

7. e) Have you often found that when you stop gambling it is because you ran out of money? NO YES

7. f) Do you ever get the urge to return to gambling to win back losses from a past session? NO YES

7. g) Have you ever received criticism about your gambling in the past? NO YES

7. h) Have you tried to win money to pay debts? NO YES

Borderline Personality Disorder (Based on Structured Clinical Interview for DSM – IV Axis II Personality Disorders, Clinician Version; First, Gibbon, Spitzer, Williams, & Benjamin, 1997)

8. a) Have you often become frantic when you thought that someone you really cared about was going to leave you? NO YES

8. b) Do your relationships with people you really care about have extreme ups and downs? NO YES

8. c) Have you all of a sudden changed your sense of who you are and where you are headed? NO YES

8. d) Does your sense of who you are often change dramatically? NO YES

8. e) Are you different with different people or in different situations so that you sometimes don't know who you really are? NO YES

8. f) Have there been lots of sudden changes in your goals, career plans, religious beliefs, and so on? NO YES

8. g) Have you often done things impulsively? NO YES

8. h) Have you tried to hurt or kill yourself or threatened to do so? NO YES

8. i) Have you ever cut, burned, or scratched yourself on purpose? NO YES

8. j) Do you have a lot of sudden mood changes? NO YES

8. k) Do you often feel empty inside? NO YES

8. l) Do you often have temper outbursts or get so angry that you lose control? NO YES

8. m) Do you hit people or throw things when you get angry? NO YES

8. n) Do even little things get you very angry? NO YES

8. o) When you are under a lot of stress, do you get suspicious of other people or feel especially spaced out? NO YES

AD/HD

1. Do you find that, more than most people, you tend to be forgetful and disorganized, you have trouble keeping track of things (like paperwork, bills, chores/tasks) and/or you are easily distracted and have trouble staying focused (i.e., on what someone is saying or on a task or job)? (as for example/typical problems) NO YES

2. Do you find that, more than most people, you are overactive or restless when you are required to sit still or be quiet, you have trouble waiting your turn (i.e., in traffic, in line, in conversation), and/or you tend to be impatient with or interrupt others? (ask for example/typical problems) NO YES

3. Do these tendencies interfere with your ability to (a) do your job well and on time? (b) do your schoolwork well and on time? (c) perform household duties well and on time (i.e., pay bills, do chores, organize schedules/appointments, for self/family)? NO YES

4. When did you first experience these tendencies? (i.e., any event you can remember that triggered these – substance use, physical

or psychological trauma or illness, sleep problems – or have you always been this way?) NO YES

That's really all the questions I had to ask. It looks like:

1. *Patient is included in the study:* _____ you'd be a real good person to have in the study.

2. *Patient is excluded from the study:* _____ unfortunately, you're not the kind of person we need in the study.

3. *Uncertainty, call back:* _____ I'm not quite sure if you're exactly the fit we need for the study. I'll confer with my supervisor and call you back within a day or so.

APPERENDIX F

Consent Form
(on University of Windsor Letterhead)

CONSENT TO PARTICIPATE IN A RESEARCH PROJECT
PSYCHOLOGICAL DISINIHIBITION MECHANISMS

PRINCIPAL INVESTIGATOR: STEPHEN HIBBARD, PH.D.
DEPARTMENT OF PSYCHOLOGY
UNIVERSITY OF WINDSOR
(519) 253 -3000 ext. 2248

Purpose of the study. In this study, we are trying to look at different "mechanisms of disinhibition" in various people. Psychologists tend to study many of these "mechanisms" from different points of view. "Mechanisms of inhibition" just means how people stop themselves from doing things they don't want to do. Mechanisms of *dis*inhibition means the ways in which some people have trouble stopping themselves. People who are disinhibited often have trouble in stopping themselves from doing things they might not really want to do or at least before they are ready. This study uses different lab assessment tasks to look into this in various people.

Procedures of the study. A) Tasks. You will be asked to do various lab tasks in this study. In two of these you will be asked to press a key on the computer keyboard when a certain signal comes up. In a third, you will learn which of different numbers are the ones that will give you a small monetary reward. In two others, you will judge whether certain figures on pieces of paper are the same (or similar) or not. You have a chance of winning a small amount of cash (less than $10.00). You have no risk of losing any money. B) Interview. There will also be some interview questions that the researchers will ask you. These questions are about emotional problems and diagnostic issues that you may or may not have. C) There will also be some personality and emotional problem questionnaires that you will answer. These are answered on computer.

Potential risks. There is nothing done to people physically in this study. There are no wires attached and nothing is put into anyone. No drugs will be administered. Some of the questions that are asked about emotional problems may bring up feelings in you that are scary, sad or otherwise uncomfortable for you if they remind you of your emotional difficulties.

Potential benefits. This is not a treatment study. Nobody is offering treatment in this study and no one is collecting information that might be used to help you later. So there is no direct benefit to you other than the compensation you will receive. Your participation in the lab tasks might be interesting to you because they are sort of like games. This study will likely be of benefit to researchers who try to understand the relationship of disinhibition to emotional problems.

Payment. You will be remunerated $60.00 in either mall or grocery gift certificates for your participation. Your parking fees will also be paid to you and you may keep any money you earn in the lab tasks.

Confidentiality. The researchers who collect your data will keep your identity completely confidential, except in rare cases when they are ethically required to do otherwise. Data collected from you will be coded to an identification number that is not linked to your name in any way. Once you sign this form you are assigned this number and your name will never be connected to the data you give. The only place we will collect your name after you start the study is your signature on the receipt for compensation. This will never be linked with any data collected from you. There are a few situations in which researchers might be ethically required to break confidentiality. These include a credible indication of current suicidal or homicidal intent or the disclosure of child abuse. If you participate in the study, you give your consent for the researchers to break confidentiality in these instances.

Withdrawal from the study. You may withdraw from the study at any time with no further obligation. You will be paid on a pro rated basis for the amount of time you spent in the lab. That is, you will be paid for the fraction of the full 5 hour study time that you actually participated: time you spent in study/5 hours x $60.

You may withdraw your consent at any time and discontinue without penalty. This study has been reviewed and received ethics clearance through the University of Windsor Research Ethics Board. If you have problems regarding your rights as a research subject, contact:

Madeleine Mekis
Research Ethics Co-ordinator
University of Windsor
Windsor, Ontario
N9B3P4

Telephone: 519-253-3000, ext. 3916
E-mail: ethics@uwindsor.ca

I hereby acknowledge that I have read both sides of this consent form and I freely agree to participate in the study.

_____
Printed name

_____ _____
Signature Date

Copy of the consent: I have received a copy of this consent form to take with me.

APPENDIX G

Intercorrelations Between Personality and Computer Variables (N = 81)

Variable	1	2	3	4	5	6	7	8	9	10
1. Age[a]	-									
2. PGSI [b]	.40**	-								
3. CW-BIS[b]	.18	-.02	-							
4. CW-Drive[b]	.15	-.11	.10	-						
5. CW-Reward[b]	.25*	.02	.38**	.48**	-					
6. CW-Fun[b]	.19	-.19	-.06	.43**	.46**	-				
7. TPQ –NS[b]	-.08	.27*	-.05	-.26*	-.17	.40**	-			
8. TPQ –RD[b]	-.08	-.10	-.18	-.03	-.11	.08	.08	-		
9. TPQ- HA[b]	.14	.20	.44**	.28*	.08	.35**	-.15	.09	-	
10. GRAPES – RE.[b]	-.20	.10	.09	-.43**	-.27*	-.38**	.21	-.04	-.56**	-
11. GRAPES – PE.[b]	-.09	.08	-.29**	-.16	-.19	-.10	-.15	.10	.44**	-.08
12. EC - R & P	.20	.25*	-.15	-.06	-.08	-.07	.07	-.01	.15	.06
13. EO - R & P	.13	-.004	.25*	.04	.20	.05	.01	.02	-.15	.12
14. EC -RO	.19	.07	-.04	.02	-.12	-.03	-.02	.13	.08	.10
15. EO -RO	.17	.07	.07	.01	-.10	-.06	.14	.09	.06	.11
16. EC - PO	.34**	.04	.03	.15	.02	-.01	-.08	-.02	.07	.07
17. EO-PO	.19	.12	.05	.06	-.04	.04	-.01	.05	.10	.14
18. SSRT -1	.39**	.31**	.004	-.03	.04	-.004	.02	.06	.05	-.02
19. SSRT- 2	.09	.13	.03	-.05	-.00	-.04	.03	.08	-.07	.10
20. SSRT-3	-.03	.09	.04	.11	-.10	-.003	.17	.02	.08	-.13
21. SSRT-m	.19	.21	.08	.02	.004	.10	.07	.11	.04	-.06
22. NSRT-1	.22*	-.01	.10	.07	.24*	.03	-.06	-.02	-.04	.07
23. NSRT-2	.21	.02	.08	.02	.26*	-.001	-.00	.02	.02	.02
24. NSRT-3	.20	.05	.05	.06	.20	-.01	.10	.01	.08	-.01
25. NSRT-m	.22*	.02	.08	.05	.24*	.01	.01	.003	.004	.03

Note. [a] denotes log transformed variable. [b] denotes centered variable. *p <.05. **p <.01.
PGSI = Problem Gambling Severity Index; CW – Carver and White measure subscale TPQ-HA - TPQ – Harm Avoidance; TPQ-NS = TPQ – Novelty Seeking; TPQ-RD = TPQ; GRAPES RE = Generalized Reward and Punishment Expectancy Scale – Reward Expectancy; GRAPES PE = Generalized Reward and Punishment Expectancy Scale – Punishment Expectancy. EO – R & P = Errors of Omission, Reward and Punishment Condition; EC – R only = Errors of Commission made in Reward only Condition; EO – R only = Errors of Omission made in Reward only Condition; EC – P only = Errors of Commission made in Punishment only Condition; EO – P only = Errors of Omission made in Punishment only Condition. SSRT-1 = Stop Signal Reaction Time Block 1; SSRT-2 = SSRT Block 2; SSRT-3 = SSRT, Block 3; SSRT-m= Mean SSRT Across Blocks; NSRT-1= Non-signal Reaction Time Block 1; NSRT-1= NSRT, Block 1; NSRT-2= NSRT, Block 2 NSRT-3= NSRT Block 3; NSRT-m= Mean NSRT Across Blocks.

	11	12	13	14	15	16	17	18	19	20
1. Age[a]										
2. PGSI [b]										
3. CW-BIS[b]										
4. CW-Drive[b]										
5.CW-Reward[b]										
6. CW-Fun[b]										
7. TPQ –NS[b]										
8. TPQ –RD[b]										
9. TPQ- HA[b]										
10. GRAPES – RE.[b]										
11. GRAPES – PE.[b]	-									
12. EC - R & P	.14	-								
13. EO - R & P	-.08	-.04	-							
14. EC -RO	.18	.30**	.15	-						
15. EO -RO	.09	.12	.32**	.21*	-					
16. EC - PO	.16	.35**	.16	.62*	.33**	-				
17. EO-PO	.04	.13	.27*	.27*	.44**	.30**	-			
18. SSRT -1	-.05	.30**	.11	.09	.15	.16	.11	-		
19. SSRT- 2	.12	.22	.18	.13	.15	.13	.32**	.41**	-	
20. SSRT-3	-.03	.02	.08	.09	.06	.12	.18	.28*	.33**	-
21. SSRT-m	-.07	.17	.16	.09	.10	.11	.17	.80**	.72**	.80**
22. NSRT-1	.16	.11	.25*	.20	.11	.16	.05	.43**	.22	.21
23. NSRT-2	.17	.10	.27*	.12	.10	.13	.07	.31**	.31**	.18
24. NSRT-3	.17	.07	.31**	.09	.20	.15	.13	.33**	.22*	.41**
25. NSRT-m	.17	.17	.29**	.14	.14	.14	.09	.40**	.26*	.25*

Note. [a] denotes log transformed variable. [b] denotes centered variable. *p <.05. **p <.01.

PGSI = Problem Gambling Severity Index; CW – Carver and White measure subscale TPQ-HA - TPQ – Harm Avoidance; TPQ-NS = TPQ – Novelty Seeking; TPQ-RD = TPQ; GRAPES RE = Generalized Reward and Punishment Expectancy Scale – Reward Expectancy; GRAPES PE = Generalized Reward and Punishment Expectancy Scale – Punishment Expectancy. EO – R & P = Errors of Omission, Reward and Punishment Condition; EC – R only = Errors of Commission made in Reward only Condition; EO – R only = Errors of Omission made in Reward only Condition; EC – P only = Errors of Commission made in Punishment only Condition; EO – P only = Errors of Omission made in Punishment only Condition. SSRT-1 = Stop Signal Reaction Time Block 1; SSRT-2 = SSRT Block 2; SSRT-3 = SSRT, Block 3; SSRT-m= Mean SSRT Across Blocks; NSRT-1= Non-signal Reaction Time Block 1; NSRT-1= NSRT, Block 1; NSRT-2= NSRT, Block 2 NSRT-3= NSRT Block 3; NSRT-m= Mean NSRT Across Blocks.

	21	22	23	24	25
1. Age[a]					
2. PGSI [b]					
3. CW-BIS[b]					
4. CW-Drive[b]					
5.CW-Reward[b]					
6. CW-Fun[b]					
7. TPQ –NS[b]					
8. TPQ –RD[b]					
9.TPQ- HA[b]					
10. GRAPES – RE.[b]					
11. GRAPES – PE.[b]					
12. EC - R & P					
13. EO - R & P					
14. EC -RO					
15. EO -RO					
16. EC - PO					
17. EO - PO					
18. SSRT -1[a]					
19. SSRT- 2[a]					
20. SSRT-3[a]					
21. SSRT-m[a]	*				
22. NSRT-1	.21	*			
23. NSRT-2	.18	.90**	*		
24. NSRT-3	.41	.82**	.89**	*	
25. NSRT-m	.32**	.93**	.97**	.95**	*

Note. [a] denotes log transformed variable. [b] denotes centered variable. *p < .05. **p < .01.

VITA AUCTORIS

Nicola E. Fitzgerald was born in 1976 in Etobicoke, Ontario. She graduated from Thomas L. Kennedy Secondary School in 1995. From there, she went to the University of Toronto where she obtained a B. Sc. (hons) in Psychology in 1999. In 2002 she earned her Masters degree in Clinical Psychology at the University of Windsor. She is currently a doctoral candidate at the University of Windsor and is in the process of completing her pre-doctoral internship at SUNY Upstate Medical University in Syracuse, NY. She hopes to graduate in Fall 2006.

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