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Passive-avoidance learning and response inhibition in problem gamblers

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Abstract

Currently, problem gambling is classified as an impulse control disorder (American Psychiatric Association, 2000). Greater understanding of problem gambling's underlying pathologies is needed. The present study is an attempt to elucidate motor and cognitive disinhibition in problem gambling by applying two theoretical approaches researched with other "impulsive" groups. These theoretical approaches are the passive-avoidance learning paradigm (e.g., Newman 1987), derived mostly from the study of psychopaths, and the stop-signal paradigm (Logan & Cowan, 1984), which comes from the study of attention deficit/hyperactivity disorder.

The study compares 30 problem gamblers (PGs) to 37 controls (CTRLs) on two computer tasks of the above paradigms. Results on the stop-signal task show that PGs are slower in stopping their responses than CTRLs ($p = 0.06$, two-tailed), with no difference between the groups in 'go' reaction times. On the passive-avoidance task, a medium effect size (Cohen's $d = 0.37$) suggests that PGs are more likely to make passive-avoidance errors, and thus to exhibit learning deficits, than CTRLs when faced with both reward and punishment.

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Passive-avoidance learning and response inhibition in problem gamblers

Introduction

Although a seemingly uncomplicated question, the 'why?' of human behaviour has been of interest from the inception of psychology as a discipline. Learning theory has proposed the notion that, broadly speaking, rewarding[1] behaviour tends to cause the behaviour to repeat, but punishing it has the opposite effect (e.g., Brogden, Lipman, & Culler, 1938; Levis, 1989; McAllister & McAllister, 1991; Mowrer & Lamoreaux, 1942). More recently, a number of researchers have also broached the role of human personality, its various characteristics, and its relation to human actions. For example, a study by O'Gorman and Baxter (2002) looked at the relationship between personality variables such as conscientiousness and self-control to imprudent behaviours or criminal intent. They defined conscientiousness based on the scale of the same name of the NEO-PI-R (Costa & McCrae, 1989). This scale, comprised of a number of personality variables (e.g., dutifulness, self-discipline, and deliberation) is conceptually similar to the idea of self-control, or the ability to act (or withhold an action) based on thoughts of short-term and long-term consequences for oneself and others (Gottfredson & Hirschi, 1990).

O'Gorman and Baxter (2002) determined that self-control relates to whether or not an opportunity for behaviour holds excitement, rather than to the possibility of punishment. In other words, if a subject does not take part in imprudent or criminal behaviour, it is because that act is deemed unexciting or unattractive. Therefore, the possibility of punishment did not stop the subject from participating. Following this line

[1] Depending upon the precise learning theory in question "positive reinforcement" might be a more proper substitution for "reward". The contemporary theories underlying this study (i.e., Gray, 1985, 1991) can accommodate this.

of research, a number of different attempts have tried to explain many aspects of human behaviour. Of particular interest are those efforts focused on the concept of impulsivity and, more specifically, impulsivity and its relation to problem gambling.

Review of the Literature

Problem Gambling

Problem gambling would benefit from greater understanding of its underlying pathologies. With respect to a definition of gambling, Bergler (1957) states that there is general consensus over the core elements of what forms the context for gambling to occur. This includes: an agreement between at least two parties in which there is an exchange of items of value on the basis of the outcome of an undecided event; participation is voluntary; and, the participants are motivated to wager valuable items in the hopes of gaining profit and/or generating a subjective arousal. Notably, these elements do not have inherent moral, social, or religious value. Problem gambling, then, would be gambling behaviours taken to an extreme, resulting in excessively negative financial, social, and psychological consequences.

Archaeological evidence suggests that gambling is a ubiquitous pastime, and exists in almost every race and culture since 4000 B.C. (Blaszczynski, 1996). Even so, there is much to be learned from its development and history. While gambling itself is not inherently a social problem, social conventions have placed limits around what is acceptable and what is not. Problems arise when the consequences of excessive gambling require some mode of intervention to prevent or avoid further harm or side effects (i.e., financial strain, criminality, marital problems).

As Blaszczynski (1996) details, both competitive gambling and loss of control have long been correlated with social costs. For example, in his historical review of gambling, Blaszczynski (1996) recounts the story of a 1500 B.C. Indus River tribesman who gambled so heavily he lost 200 000 slaves, his kingdom, his brother, and his wife. In

more recent history, by 1822, the majority of European nations had some laws prohibiting gambling, which closely preceded similar laws in America. These laws followed an increase in problems with respect to family welfare, public safety, and juvenile delinquency.

Unfortunately, the current state of affairs does not appear to be much brighter. Some have suggested that any attempts to contain or limit gambling are destined to fail for three basic reasons: gambling continues to hold the prospect for fast easy money; excitement and entertainment are inherent to the activity; and gambling represents a lucrative, and therefore, attractive, source of revenue for governments and entrepreneurs alike (Blaszczynski, 1996).

Similar to the historical account, problem gambling is associated with many present day social issues: substance abuse, suicide, divorce, bankruptcy, and legal problems (Lesieur & Rosenthal, 1991). Early estimates of Canadian prevalence rates for problem gambling approach 2% in different areas of the country (Ladouceur, 1996; Shaffer, Hall, & Vander Bilt, 1999). A recent national survey of Canadian gambling (Cox, Yu, Afifi, & Ladouceur, 2005) compares prevalence rates across the provinces. Using the Canadian Problem Gambling Index on a sample of 34 770 community-based respondents aged 15 years or older, results show that Manitoba (2.9%) and Saskatchewan (2.9%) have the highest prevalence of gambling problems (moderate and severe), followed by Alberta (2.2%), Nova Scotia and Ontario (2%), Newfoundland, Prince Edward Island, and British Columbia (1.9%), Quebec (1.7%), and New Brunswick (1.5%). The highest prevalence is in areas with high concentrations of community based video lottery terminals and permanent casinos.

In addition to this existing scenario, Wynne (2002) has forecasted trends in Canadian gambling:

1) Gambling will continue to expand, and machine-based gambling such as video lottery terminals (VLT), coin slots, electronic Keno and bingo, and video poker will become the most pervasive form of gambling.
2) As people become increasingly high-tech, with more personal home computers, the future of gambling will include legalized Internet gambling on table games, horse races, sporting events, elections, etc.
3) The hospitality and tourism industry will successfully lobby governments to allow "gaming rooms" and "mini-casinos" in hotels, convention centres, and tourist attractions.
4) Funding for charitable organizations will increasingly rely on gambling revenues, such as from lotteries, raffles, casino nights, and grants from government sponsored gambling.

Along with these forecasted Canadian trends, the need to further study gambling exists since most of the available research investigating the nature of problem gambling utilizes samples from either the United States or Australia, and there is growing evidence that the Canadian experience is unique (Beaudoin & Cox, 1999; Wynne, 2002). In a study by Govoni, Frisch, Rupcich, and Getty (1999), the authors looked at the impact of casino gambling a year after it was introduced into a specific community. Although not statistically significant, the study found some evidence of gamblers spending more money after the opening of the casino.

Currently, problem gambling is classified as an impulse control disorder alongside kleptomania, pyromania, etc. (American Psychiatric Association, 2000). This diagnostic taxonomy perpetuates theory and speculation over the exact role of impulsivity in gambling. To this end, models of problem gambling have attempted to map the pathways to the development of gambling problems. These attempts include various typologies and routes to development.

Pathways to Problem Gambling

In 1970, Moran published an article that made one of the first references to impulsivity in problem gambling. In his article, he described five subtypes of gamblers: impulsive, subcultural, neurotic, psychopathic, and symptomatic. He described the impulsive subtype as presenting with increasing loss of control with an associated ambivalence towards the gambling activity. Higher degrees of disturbance also described the impulsive gamblers.

Current attempts by Blaszczynski and colleagues also suggest typologies of, and pathways to the development of, problem gamblers. In the conceptual pathway model, Blaszczynski (2002), and Blaszczynski and Nower (2002), identify three main subgroups: normal, emotionally vulnerable, and biologically based impulsive gamblers. This premise is a model that attempts to integrate biological, personality, developmental, cognitive, learning theory, and environmental factors. Although based primarily on clinical experience, there is scope for empirical investigation.

To begin with, the assumption is that all three groups are exposed to the common influences of ecological factors, cognitive processes, and contingencies of reinforcement. The ecological factors involve public policy issues that may promote the availability and

accessibility to gambling and gambling resources. The cognitive processes and contingencies of reinforcement draw from classical and operant conditioning. The postulate is that conditioning to gambling cues results in the urge to gamble which leads to habitual patterns of gambling. The development of cognitive schemas is thought to overlay this conditioning. For example, early and repeated wins may result in a set of irrational beliefs that promote gambling behaviour as a successful source of income. The reinforcing nature of gambling coupled with irrational schemas fortifies patterns of habitual gambling (Blaszczynski, 2002; Blaszczynski & Nower, 2002).

Although the three factors mentioned above are relevant to all gamblers, additional features differentiate the three subtypes of gamblers.

1. *Normal Gamblers.* This type of gambler meets the formal criteria for problem gambling at the peak of their gambling disorder, but does not have any premorbid psychopathology. Patterns of gambling behaviour fall within the range of regular to heavy gambling and excessive gambling. Generally, gambling is a result of bad judgments or poor decision-making, as opposed to psychological disturbance.

   Normal gamblers experience a preoccupation with gambling, chase losses, and manifest depression and anxiety because of the financial pressures associated with repeated losses. Clinically, their difficulties are the least severe of the three subtypes as there is generally no, or limited, premorbid psychopathology, substance abuse, or impulsivity. They are also more motivated to engage in treatment and comply with instructions, requiring minimal intervention.

2. *Emotionally Disturbed Gamblers.* Unlike the previous group, emotionally disturbed gamblers generally have psychological vulnerabilities that predispose them to gambling. They are motivated in their gambling behaviours to reduce or modulate various affective states, seeing gambling as a means of escaping their depression, anxiety, or other external stresses. This subtype often presents with histories that include problem gambling in the family, poor developmental experiences, neurotic personality traits, and negative life events. Because of the psychological disturbance, this group of gamblers is more resistant to change, and requires treatment that addresses both the underlying vulnerabilities as well as the gambling problem.
3. *Biologically Based Impulsive Gamblers.* Unlike the previous two groups, this type of gambler is defined by neurological or neurochemical dysfunction reflected in impulsive and antisocial features. For this type of gambler, the differential response to rewards and punishments is the result of biologically based impulsivity. They actively seek out rewarding activities, display difficulties in delaying gratification, and have impaired responses to punishment. Even when the consequences of their actions are painful, they do not alter their behaviours. As such, they are the most difficult to treat with respect to their gambling problems.

Although this model of problem gamblers is still preliminary, it provides a valuable heuristic for classifying the various types of gambling behaviours in both research and clinical situations. The third group in particular has been the focus of

considerable research. Despite the equivocal understanding of problem gambling, a large body of empirical investigation has focused on the various biological and psychological correlates in an attempt to understand gambling behaviours.

Biological Research in Problem Gambling

Research investigating the potential biological components of problem gambling has found that gamblers present with varied responses to neurochemicals. A number of researchers have shown that gamblers may respond to treatment with selective serotonin reuptake inhibitors (SSRIs) (Blanco, Petkova, Ibanez, & Saiz-Ruiz, 1999; Hollander, DeCaria, Mari, Wong, Mosovich, Grossman, & Begaz, 1998; Hollander, DeCaria, Hankell, Begaz, Wong, & Cartwright, 2000; Lopez-Ibor, 1988; Moreno, Saiz-Ruiz, & Lopez-Ibor, 1991).

Blanco, Ibanez, Blanco-Jerez, Baca-Garcia, and Saiz-Ruiz (2001) studied the plasma testosterone levels and psychological characteristics of male problem gamblers. They hypothesized that since impulsive behaviours in other groups who had abnormal serotonergic function and scores on measure of psychopathic behaviours had high levels of testosterone, perhaps this hormone was also involved in the pathophysiology of problem gambling. Unlike the relationship between problem gambling and serotonin, however, the results of this study found that problem gamblers did not have elevated levels of testosterone compared to healthy non-impulsive controls.

Research has also found altered dopaminergic and noradrenergic action in problem gamblers (Bergh, Eklund, Sodersten, & Nordin, 1997; Carrasco, Saiz-Ruiz, Hollander, Cesar, & Lopez-Ibor, 1994; Comings, Rosenthal, Lesieur, & Rugle, 1996). Bergh, et al. (1997) examine the possibility that problem gamblers have altered

neurotransmission of monoamine. With ten problem gamblers and seven controls, samples of the cerebrospinal fluid (CSF) were tested for monoamines and their metabolites. Results of the study suggest a change in the function of the dopaminergic system (which possibly mediates positive and negative reward) and the function of the noradrenergic system (which possibly mediates selective attention).

Similarly, the work of Comings, et al. (1996) outlines earlier research that showed the incidence of the D2A1 allele in substance abusers. As such, it is suggested the D2A1 allele for the dopamine D2 receptor gene is present in problem gamblers. Comings, et al. (1996) report that evaluations of problem gamblers based on severity of problems show that 63.8% of gamblers in the high severity range carry the D2A1 allele. Conversely, only 40.9% of gamblers in the low severity range carry the allele. Interestingly, 76.2% of problem gamblers in the sample who are comorbid alcohol abusers carry the gene compared to 49.1% of males who do not have a comorbid alcohol abuse/dependency issue.

Studying the EEGs (electroencephalograms) of gamblers shows a difference from controls and a similarity to the EEGs of attention-deficit disorder (ADD) children (Goldstein, Manowitz, Nora, Swartzburg, & Carlton, 1985). Further, when comparing nonsubstance dependent gamblers and "healthy" controls, gamblers are significantly more impaired in their memory and concentration (Regard, Knoch, Gutling, & Landis, 2003). In this sample, the EEGs of 67% of gamblers also reveal dysfunctional activity compared to only 26% of controls. The Regard, et al., study concludes that otherwise "healthy" problem gamblers evidence a greater number of brain injuries, fronto-temporo-limbic neuropsychological dysfunctions, and EEG abnormalities.

Psychological Research in Problem Gambling

With respect to psychological research in the area, Blaszczynski and McConaghy (1989) look at anxiety and depression as possible determinants in the maintenance of gambling behaviours. Results indicate some support for the notion that problem gambling is a behavioural stress reaction. While the problem gamblers in the study have a moderate level of depression, there are no differences from the normative sample on the state or trait anxiety scores.

A large body of research has also looked at the relationship between problem gambling and the use of substances. Results from the St. Louis epidemiological catchments survey have found that problem gamblers are at an increased risk for several diagnoses, including alcoholism and tobacco dependence (Cunningham-Williams, Cottler, Compton, & Spitznagel, 1998). Problem gamblers in an addictions treatment facility have greater disturbance when compared to non-problem gamblers on measures of premorbid risk, problem substance use, and psychiatric comorbidity (Langenbucher, Bavly, Labouvie, Sanjuan, & Martin, 2001). Finally, Maccallum and Blaszczynski (2002) have found that substance abuse is a common comorbid condition in problem gambling, and the rates for substance abuse disorders in a sample of treatment-seeking problem gamblers is higher than the general population.

In addition to substance use, Rosenthal and Lesieur (1992) have reported that problem gamblers experience withdrawal symptoms similar to those of individuals with substance-use disorders. Other clinical reports have noted that gamblers indicate "highs" similar to those experienced by using cocaine and other drugs. This has also involved a

distinct craving for the "feel" of gambling and developing a tolerance at which point greater risks are taken to achieve the same level of arousal (Blum, 2000).

Impulsivity and Disinhibition

Prior to the discussion of impulsivity and gambling, it is necessary to delineate the current understanding of impulsivity or disinhibition as a construct. Recent research in the area of impulsivity has focused on a wide range of disorders and personality characteristics. This research includes impulsivity's relationship to irritability and aggression (Stanford, Greve, & Dickens, 1995; Milligan & Waller, 2001), alcohol use/abuse and related perceptions (Marinkovic, Halgren, Klopp, & Maltzman, 2000; McCarthy, Miller, Smith, & Smith, 2001), personality disorders (Caseras, Torrubia, & Farre, 2001), cocaine use (Moeller, Dougherty, Barratt, Schmitz, & Swann, 2001b), sexual behaviours (McCoul & Haslam, 2001), and gambling (Langewisch & Frisch, 1998; McDaniel, 2002; Petry, 2001b; Steel & Blaszczynski, 1998).

Although impulsivity is a defining criterion in a number of psychological diagnoses, as described in the Diagnostic and Statistical Manual of Mental Disorders – 4th edition (DSM-IV-TR; American Psychiatric Association, 2000), there remains inconsistency in the definition of the term. This inconsistency and lack of agreement for a definition of impulsivity has also been the focus of considerable research (i.e., Barratt, 1985; Kindlon, Mezzacappa, & Earls, 1995; Moeller, Barratt, Dougherty, Schmitz, & Swann, 2001a).

Kindlon, et al. (1995), states that this disagreement emerged partly because impulsivity has several manifestations, from cognitive to emotional to motor presentations. As such, there is more than one plausible pathway resulting in what,

superficially, appears to be the same behaviour. Related to the multiple possible manifestations of impulsivity, Barratt (1985) made efforts to develop a definition of impulsiveness that was independent of other personality traits, had clinical utility, and was measurable by methods other than self-report. By reviewing past item analyses of item pools from self report questionnaires claiming to assess impulsiveness, a factor analysis of the impulsiveness items indicated this trait did not consist of a single dimension.

Barratt's (1985) research concludes there are three factors that consistently appear in the studies reviewed:

1) a motor impulsiveness subtrait that involves action without accompanying thought, and is similar to Eysenck and Eysenck's (1977) "impulsiveness narrow" factor
2) a cognitive impulsiveness subtrait which encompasses rapid cognitive decisions, separate from motor impulsiveness, and similar to the Eysenck and Eysenck (1977) idea of "impulsiveness broad"
3) a non-planning impulsiveness that identifies a lack of future planning or consideration of consequences.

In addition to the three factors, the factor analysis of the impulsivity items also led him to conclude that these subtraits were independent of other personality traits and had their own clinical utility. Barratt (1985) did note that, although several studies had recognized a risk-taking factor, this factor was more distinctive of socialization or sensation-seeking as opposed to impulsivity.

Kindlon, et al. (1995), elucidate the motivational and cognitive domains of impulsivity. The motivational domain entails individual differences in sensitivity to both punishment and reward (Bachorowski & Newman, 1990; Gray, 1985, 1987; Newman & Bachorowski, 1990), passive-avoidance learning (Newman, Patterson, & Kosson, 1987), and delay of gratification (Mischel, Shoda, & Rodriquez, 1989). The cognitive domain is more concerned with the (in)ability to inhibit a dominant response, as well as other processing impairments. Inhibitory control, therefore, is limited to those cortical functions responsible for response modulation, planning, or the inhibition of behaviour.

Moeller, et al. (2001a), published an exhaustive article in the hopes of bridging clinical and research work on the definition and measurement of impulsivity. Of interest is their biopsychosocial definition of impulsivity, which is similar to the work of Barratt (1985) and Kindlon, et al. (1995). This definition stipulates that any comprehensive understanding of impulsivity needs to include the decreased sensitivity to negative consequences of behaviour, the lack of regard for long-term consequences, and the rapid, unplanned reactions to stimuli without adequately processing available information. They further define impulsivity as a predisposition, and part of a pattern of behaviour, rather than a single act. Since impulsive behaviours involve rapid, unplanned actions without conscious processing and weighing of consequences, impulsivity should be distinct from compulsive behaviour or impaired judgment, and this distinction has both clinical and research relevance.

As previous conceptualizations of impulsivity indicate, there are both motor and cognitive components, as well as a motivational component. In addition, there is consistent agreement that there is a need for a clearer understanding of what impulsive

behaviour entails. The ubiquity of impulse control problems in psychopathology and limited understanding of impulsivity as a construct emphasizes the need to continue to investigate this phenomenon. Because of the pervasive and equivocal nature of impulse control problems, a goal of clinical research should be to understand which psychological processes, such as inhibitory functions, present abnormally in particular disorders (Wakefield, 1992).

Lack of inhibitions, or disinhibition, refers to the limited control over response inclinations in human behaviour (Gorenstein & Newman, 1980). As previously stated, this involves response modulation, planning, or the control of specific behaviours (Kindlon, et al, 1995). Gorenstein and Newman (1980) state this concept of disinhibition is useful to understand the main deficits in psychopathology (e.g., psychopathy, child hyperactivity, hysteria, and alcoholism). In addition, a more coherent connection of specific inhibitory processes to different disorders adds to a more advanced, process-based description of psychopathology (Nigg, 2000). To this end, two specific theoretical approaches to inhibition are provided: passive-avoidance learning and the stop-signal paradigm.

Passive-avoidance Learning Paradigm

The concept of passive-avoidance learning deficits is related to the idea that, in some individuals (i.e., impulsives), there is a diminished ability to inhibit reward (positive reinforcement) motivated responses, even when the possibility for punishment is present (Gorenstein & Newman, 1980). Newman and colleagues suggest that dysfunction caused by lesions in the septal-hippocampal region of the upper brainstem in rats (*septal syndrome* – hyperactivity, response perseveration, etc.) acts as an analogue

for the various psychological mechanisms that underlie a number of human disinhibitory pathologies such as substance abuse, alcoholism, and psychopathy. The *septal lesion model* (Patterson & Newman, 1993) provides a heuristic for the elaboration of the core processes of human disinhibition.

The septal lesion model, the premise for Gray's *Psychobiological Model of Personality* (1985, 1987, 1991), is the model that Newman and colleagues' extend in their passive-avoidance learning paradigm (Patterson & Newman, 1993). The psychobiological model works primarily from the principles of classical conditioning and motivation, and involves four systems: the behavioural activation system (BAS), the behavioural inhibition system (BIS), the fight/flight system (F/F), and the non-specific arousal system (NAS). The first two, the BIS and the BAS, are mutually competitive systems (see Table 1: Summary of Psychobiological Model of Personality).

The Behavioural Activation System (BAS). The BAS is responsible for approach behaviours. It activates motor responses to signals of reward, and activates avoidance behaviours when presented with frustrative nonreward (that is, the perception that pursuit of a previously rewarding stimulus would now be frustrating) or punishment. According to the model, ascending dopaminergic fibres in the appetitive (reward) system of the brain neurally mediate the BAS. These dopaminergic fibres extend from the subcortical structures of the brain to the prefrontal cortex.

When the BAS is engaged, by signals of reward and/or nonpunishment, there is a corresponding increase in cortical arousal which encourages approach (to rewards) and active avoidance (of punished) behaviours. If an individual has an overactive BAS, s/he routinely detects rewarding stimuli, as opposed to those with an under active BAS. As

such, when already primed for rewards, an overactive BAS leads to more repeated approach behaviours regardless of potential punishments.

The Behavioural Inhibition System (BIS). Also regarded as the anxiety system, the subcortical septal-hippocampal structure, as well as the thalamus and midbrain, mediate the BIS. There are two modes of operation within the BIS: checking and control. The checking mode, mediated by the hippocampus, functions to determine whether current stimuli is consistent with what is expected, and to estimate what will come next. The control mode is engaged when triggering cues (i.e., learned signals for punishment or nonreward, unconditional fear stimuli, etc.) are detected. Upon detection of the triggering cues, the BIS results in three behavioural outputs: stopping of the ongoing motor activity (passive-avoidance), heightened arousal, and increased attention to the environment and triggering stimuli.

In contrast to the action of the BAS, an overactive BIS results in a greater likelihood of inhibiting approach behaviours in the face of punishment, while an under active BIS increases the likelihood of not inhibiting such behaviours. An under active BIS, therefore, predisposes people to having a lower sensitivity to punishment cues.

The Fight/Flight System (F/F). Rather than responding to cues for punishment or cues for nonreward as the BIS does, the F/F system responds to actual pain (unconditional punishment) and actual nonreward. The F/F is hypothesized to involve the ventromedial hypothalamus and the amygdala. Activation of the F/F generates active avoidance or escape (flight) behaviours, or defensive aggression (fight), depending on

Table 1

Summary of Psychobiological Model of Personality

	COMPETITIVE SYSTEMS			
	BAS	**BIS**	**F/F**	**NAS**
WHERE?	mediated by dopaminergic fibres which extend from subcortical structures to prefrontal cortex	mediated by subcortical septal-hippocampal structure, midbrain, and thalamus	hypothesized to involve the ventromedial hypothalamus and amygdala	connected to the reticular activating system
HOW?	signals of reward and nonpunishment increase subcortical arousal and activate the BAS * encourages approach towards reward * encourages active avoidance of punishment	1) checking mode - determines whether current stimuli is consistent with what is expected and estimates what will come next 2) control mode – engaged when learned signals for punishment and nonreward are detected	unlike BIS which responds to *cues* for punishment and nonreward, F/F responds to *actual* pain (unconditional punishment)	responds positively to activation of either BIS or BAS
WHAT?	1) activates motor response to signals of reward 2) activates avoidance behaviour when presented with nonreward or punishment	detection of learned signals for punishment and nonreward results in 3 behavioural outputs: 1) stop the ongoing motor activity (passive avoidance) 2) heightened arousal 3) increased attention to the environment	depending on the environmental context: 1) generates active avoidance or escape (flight) behaviours, or 2) generates defensive aggression (fight)	because the presence of cues for punishment or reward results in heightened arousal and alertness, the behavioural response is intensified
When PROBLEMS arise	because the BAS is activated by cues for reward, if there is an overactive BAS, the person constantly looks for/detects rewarding stimuli and the prospect of reward overrides the possibility of punishment	an under active BIS predisposes people to have less sensitivity to (higher threshold for) cues for punishment, whereas an overactive BIS results in the greater likelihood of inhibiting approach		

what stimuli are present in the environmental context. Gray's (1987) model, however, also indicates that activation of the BIS can inhibit activation of the F/F.

The Non-specific Arousal System (NAS). Also considered the modulating system, the NAS connects to the ascending reticular activation system, and responds positively to activation of either the BIS or the BAS. Because the presence of cues for punishment or reward results in heightened arousal and alertness, the ensuing behavioural response is intensified. Heightened sensitivity to reward is a frequent presentation of disinhibited individuals who fail to learn from aversive feedback. It is this system (activated due to an overactive BAS) which Newman and his colleagues (1987, 1993) have expanded upon in their passive-avoidance learning model. In other words, in disinhibited people an overactive BAS makes it difficult to withhold appetitively-motivated responses in the face of potential punishment.

In 1987, J. P. Newman first published a detailed account of the passive-avoidance deficits present in extraverts and psychopaths[2], providing a potential mechanism for the impulsive behaviours of disinhibited individuals that tied into Gray's Psychobiological Model (see Figure 1). This four-stage mechanism of response modulation consisted of:

1) The establishment of an approach response set. Disinhibited individuals experience a deficiency in response modulation when they are acting within a context for reward. Any possibility of reward results in an emotional/motivational state, characteristic of which is a proclivity to

[2] The term "psychopath" is operationally defined as a score of 30 or greater on the Hare Psychopathy Checklist – Revised (PCL-R; Hare, 2003), and meets the descriptive/diagnostic criteria defined within the PCL-R. "Psychopathy" and "psychopath" are constructs which are different from, and should not be confused with, the DSM-IV-TR (American Psychiatric Association, 2004) diagnosis of Antisocial Personality Disorder.

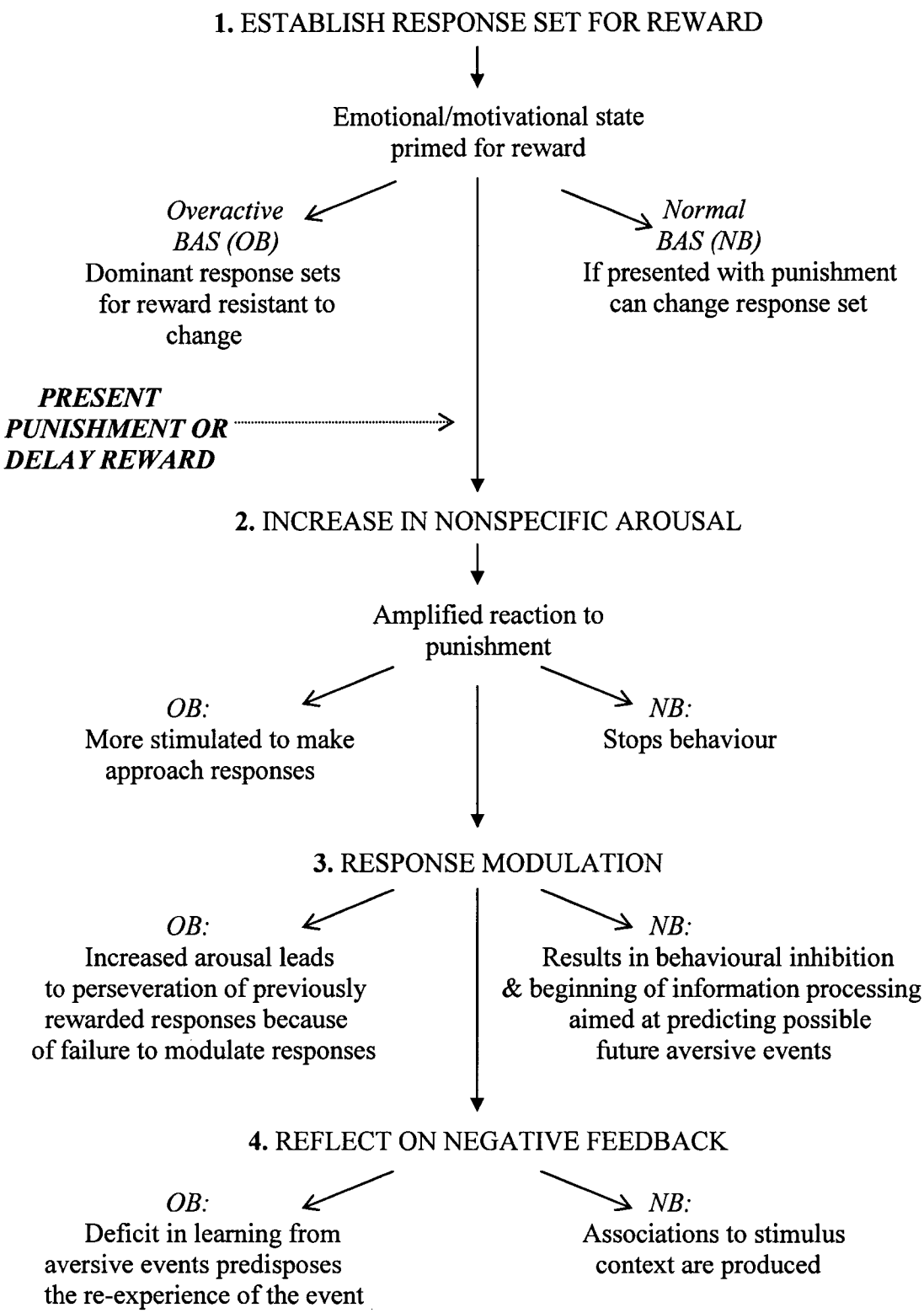
respond and a resistance to change the positive response set. Alternatively, if presented with a situation in which there is no demand to change the established response, a disinhibited person will perform as well as non-disinhibited individuals. According to Gray's model, disinhibited or impulsive individuals are more likely to establish dominant response sets for reward with greater frequency due to the BAS function of responding to reward situations.

2) In situations where there is punishment, omission of a reward, or a delayed reward, disinhibited individuals experience an incremental increase in non-specific arousal. The increase in non-specific arousal then leads to an amplification of the reaction to the aversive event. If an individual has an overactive BAS, s/he is more stimulated to make approach responses, thereby experiencing even greater levels of arousal following the aversive event.

3) When there is increased arousal, impulsive people tend to persevere with previously rewarded responses because of failures in response modulation or retrospective reflection. As such, disinhibited individuals are less likely to alter their response set, resulting in response facilitation. An overactive BAS determines this process. Conversely, an under active BAS results in a passive coping response such as behaviour inhibition and the beginning of information processing aimed at predicting possible future aversive events. It is important to note, however, that this is due to the normal operation of the BIS, assuming a normally functioning BIS. It is the

Figure 1

Four Stage Response Modulation (Newman, 1987)



*in*direct result of an under active BAS that allows a stronger BIS to predominate.

4) Finally, if reflecting on negative feedback (i.e., punishments, non reward, etc.) happens, associations to the stimulus context are produced. Since disinhibited people lack this retrospective reflection, learning from aversive events is deficient. As a result, an overactive BAS predisposes disinhibited individuals to experience the same aversive event in the future.

The implications of this mechanism for response modulation are two-fold. First, because of the emotional reaction to punishment, disinhibited individuals are more likely to respond in a reflex-like style when presented with negative feedback. Poor quality rapid responding, with limited forethought characterizes this style. Second, because of the emotional reaction, the negative feedback experiences are not adequately processed. These individuals, therefore, do not have the same network of associations regarding negative events as non-impulsive people, and are more likely to be lead by prospects of reward rather than the threat of punishment.

Avila (2001), using the theoretical framework of Gray, studied disinhibited behaviours, and attempted to assess the model of Newman and colleagues. Utilizing a point scoring reaction time (RT) procedure in four different experiments, Avila attempted to differentiate two disinhibitory mechanisms associated with the BIS and the BAS. Results were consistent with the work of Newman, et al. (1987, 1993), in that the BAS mediated mechanism was related to lacking inhibition in approach behaviours following the introduction of punishments. The BAS mediated mechanism was also related to

deficits in learning from aversive cues when responding for reward. The BIS mediated mechanism linked to a greater capacity to extinguish aversive associations, and linked to less interference of approach behaviours when presented with aversive stimuli. Corr (2003) also empirically demonstrated that the BIS and BAS systems do not always function independently. The two systems, however, often have a moderating effect on one another.

The theoretical approach to disinhibition described above has generated research to determine how the response modulation mechanism manifests in various psychopathologies. For example, Nichols and Newman (1986), studied the behavioural reactions of extroverts and introverts to punishments and rewards, and found that introverts were more likely to pause after punishment (allowing for response reflection), even while seeking reward. Extroverts, on the other hand, tended to form response sets that were resistant to interruption. Sometimes, these response sets were even more likely repeated following punishment.

Further to the development of the model, Newman and colleagues have developed a task, the Passive-Avoidance Task, to measure how individuals learn to inhibit responses. Briefly, it is a variant of the go/no-go task in which subjects are required to learn when responding to a stimulus results in either reward or punishment. In the primary task condition (reward and punishment, *PALR*), a subject is shown a set of eight numbers (one number at a time), and is required to learn which four of the eight numbers are 'good', or positive stimuli (S+), and which four of the eight numbers are 'bad', or negative stimuli (S-). When the subject correctly responds to the S+/'good' numbers s/he receives a small monetary reward. The subject is punished (loss of money) when s/he

responds to the S-/'bad' numbers. Additional tasks include a reward only condition (*RRI*) and a punishment only condition (*PANR*). All three conditions are described in greater detail in the Methods section.

At present, the majority of the research conducted with the Passive-Avoidance Task and paradigm have used samples of extroverts/introverts and psychopaths. Patterson, Kosson, and Newman (1987), used this task to explore the mechanism underlying passive-avoidance learning, and its relation to extraversion, neuroticism, and response latency after punishment. Results showed that extroverts commit more passive-avoidance errors (responding when they should not, and therefore, experiencing punishment) than introverts, and more frequently fail to pause following punished errors when compared to introverts. For both extroverts and introverts, instances of longer pausing following punishment result in better learning from the punishment. This suggests that, when cues for reward are present, the reaction of extraverts to punishment (i.e., not pausing) interferes with processing the punished errors, leaving them more likely to repeat these errors in an impulsive, non-reflective action. Similar findings are present in research with psychopaths (Newman & Kosson, 1986; Newman, Widom, & Nathan, 1985), and in the presence of monetary incentives (Newman, Patterson, & Kosson, 1987).

Patterson and Newman (1993) again look at extroverts and introverts on the passive-avoidance task using two conditions: one with both rewards and punishments (*PALR*), and one with punishments only (*PANR*). They report that extroverts in the *PALR* condition commit significantly more passive-avoidance errors (i.e., respond to more no-go cues) than introverts. In the *PANR* condition, however, extroverts and introverts are

equally effective in learning to inhibit responses when not being punished is the only incentive for responding correctly. Following this study, a new element to passive-avoidance deficits becomes apparent: the interaction between individual differences and situational variables. Therefore, when not required to switch between approach and avoidance contingencies, extroverts do not exhibit the same deficits.

In contrast to the research conducted with males (as most of the previous studies have been), Segarra, Molto, and Torrubia (2000) examine the manifestation of passive-avoidance in extroverted females. Results show that, unlike the research with males, the *PALR* condition does not produce passive-avoidance error differences between neurotic extroverts and stable introverts. Interestingly, when the *PALR* condition is the first administered, neurotic introverts make more passive-avoidance errors than other groups. In addition, stable extroverts display behavioural disinhibition when the *PANR* condition is administered second. Although the results are contradictory to previous studies (where introverts, in their ability to learn to passively avoid, are as deficient as extroverts), this research merely highlights the complexity of disinhibition.

Overall, the passive-avoidance learning theory, with the corresponding research, maintains that the impulsive behavioural style of disinhibited individuals arises due to their active, non-reflective reactions to aversive stimuli. When these individuals participate in reward-seeking behaviours, they experience a strong biopsychosocial response to events or stimuli that might fail to prevent, disturb, or punish their approach behaviours. This response style easily becomes maladaptive in that disinhibited individuals continue to experience negative consequences as they do not slow down and reflect to learn from experience.

Despite the promising nature of the passive-avoidance learning model, there are only a limited number of clinical populations used in research (i.e., psychopaths, introverts/extroverts). In order to determine if this pattern of disinhibition manifests similarly in other impulse-disordered psychopathologies, further research needs to be undertaken. This would allow for a greater understanding of impulsive presentations, and a more distinct understanding of impulsivity as a construct.

The Stop-Signal Paradigm

Similar to the passive-avoidance learning paradigm espoused by Newman et al., the Stop-Signal Paradigm is a model of disinhibition that also provides a method of understanding the cognitive and motor components of impulsive behaviours. In everyday situations, people are able to interrupt, restrain, or rapidly change behaviours deemed inappropriate by sudden changes or events. This ability implies internally generated cognitive and motor inhibitory responses, and has considerable functional and survival importance.

In one of their introductory articles, Logan and Cowan (1984) present the stop-signal paradigm, and attempt to link the ideas of motor control and of cognitive control by developing a model whereby either perspective explains control. For these researchers, 'control' is an exchange between the *executive* system and the *subordinate* system. The executive system is responsible for forming intentions, and issuing commands for these intentions to be fulfilled. The subordinate system interprets the commands issued by the executive system and completes the assignment. In this interaction, the sequence of *acts of control* is the actions of the executive system. It is these acts of control that are analyzed behaviourally.

For Logan and Cowan (1984), the act of control investigated is the ability to inhibit both thoughts and actions once the thoughts and actions are no longer relevant to current goals. Although quick to indicate that inhibition is not the only act of control for which the executive system is responsible, the researchers feel it is important for both motor control (e.g., stopping oneself from hitting a tennis ball that is already called out of bounds) and cognitive control (e.g., suppressing a negative comment).

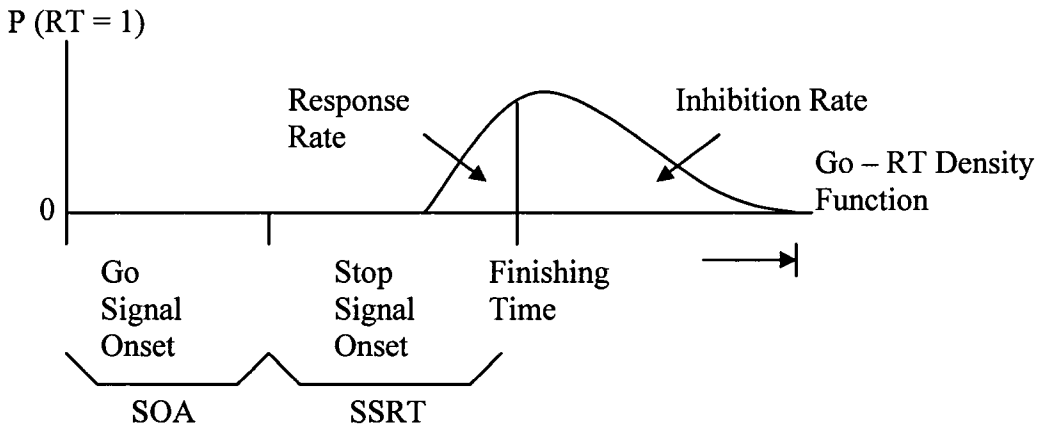
While other theories of performance and behaviour assume that two signals must be processed quickly and in sequence for action to occur, the stop-signal paradigm assumes there is no relationship between 1) the processes which respond to a stimulus, and 2) the processes which respond to information indicating an action should be inhibited (Logan, Cowan, & Davis, 1984). As a result, when a command is given to stop a response that has already started, the two processes start to race (race model). In other words, the response or 'go' processes are racing against the inhibiting or 'stop' processes.

The race model of response inhibition assumes there are two sets of processes that operate independently (as presented above). The first set of processes begins at the start of the task stimulus (the 'go' task), resulting in the launch and conclusion of a response; the 'go' process. In a lab setting, this might be a choice reaction time (RT) task where a subject responds to a specific stimulus. The second set of processes does not begin until a stop-signal occurs, and this results in the inhibition of the response; the 'stop' process. In the lab, this stop-signal could be a tone or loud noise. Accordingly, the response is either completed or withheld based on which process wins the race. Within this model, someone is more likely to inhibit his or her response when the stop-signal closely follows the task stimulus (*stop-signal delay).* If the stop-signal is delayed for too long after the

'go' signal, the 'go' signal process "wins the race" (see Figure 2: Race Model of the Stop-Signal Paradigm).

Figure 2

Race Model of the Stop-Signal Paradigm (Band, van der Molan, & Logan, 2003)



The stop signal is presented after a SOA (stimulus-onset asynchrony) relative to the go-signal. The stop-processes finish after the SSRT (stop-signal reaction time) relative to the stop-signal onset. When the SSRT is assumed to be constant, this finishing time intersects the density function of go-RT. Reponses from the left part of the go-RT density function are too fast to be inhibited whereas responses from the right part are stopped correctly. Therefore, the finishing time of the stop-process divides the go-RT density function into the RR (response rate) and the inhibition rate.

As stated, whether a response is inhibited is dependent upon the relative finishing times of the 'go' process and the 'stop' process. The term "finishing time" refers to the amount of time it takes between the start of the 'go' signal and the response, or the amount of time between the start of the 'stop' signal and its hypothetical ending. Note that the duration or finishing time of the 'go' process is directly observable in those trials when no subsequent stop-signal is given: the finishing time is the distance between the 'go' signal and the response. Duration or finishing times of 'stop' processes are not

directly observable since successful inhibition on those trials in which a stop-signal occurs does not result in a specific observable event. Rather, notable is the absence of an event, or, the absence of responding to the go signal. Determining the relative finishing times requires at least the following four factors: 1) the stop-signal delay; 2) the mean RT to the primary 'go' task; 3) the mean RT to the stop-signal; and, 4) the variance of RT to the 'go' task (Logan, et al., 1984). Knowledge of the probability of inhibition and the distribution of the 'go' task RT allow for estimation of the point in time at which the response to the stop-signal occurred. Once this estimate is determined, subtracting out the stop-signal delay predicts the stop-signal RT.

Although the actual mechanisms involved are still speculative due to limited empirical evidence, there are two mechanisms hypothesized to interrupt or withhold speeded responses. First is a central mechanism that operates by inhibiting response activation processes within cortical motor structures so that motor commands are not completed. The second mechanism is peripheral, and operates to prevent the peripheral motor structures from executing central motor commands (De Jong, Coles, & Logan, 1995).

In addition to the primary task, the model makes additional predictions about RTs on those trials which have a stop-signal, but the individual responds regardless (*signal-respond* trials). It states that the mean signal-response RTs should be faster than mean no-signal response RTs. In addition, the signal-respond RTs should increase as the stop-signal delay increases (Logan, et al., 1984).

Overall, the model predicts that the effects of the stop-signal delay and the 'go' task RT can compensate for each other. Therefore, the probability of responding given

the presentation of a stop-signal should not change if there are changes to other aspects (i.e., 'go' task RT, stop-signal delay, etc.). As such, the stop-signal paradigm and accompanying task (described in more detail in the Methods section), are useful in measuring the inherent ability to inhibit actions.

A number of research studies have used the stop-signal paradigm to assess the ability to inhibit thought and action. De Jong, et al. (1995) attempt to extend the study of motor inhibition beyond all-or-none situations to conditions that require selective motor inhibition where some responses are inhibited but not others. They also attempt to test the notion of two functionally distinct inhibition mechanisms, labelled executive and peripheral. Results of this study support the concept of functionally distinct mechanisms and strategies responsible for inhibitory motor control in various circumstances.

Another study utilizing the stop-signal paradigm is one by Logan, Schachar, and Tannock (1997), in which impulsivity is operationalized as a deficit in inhibitory processes. According to the race model of the stop-signal paradigm, poor inhibitory control has two possible causes: responding too quickly to the 'go' signal so that a response is executed before the person can respond to the stop-signal; or, responding too slowly to the stop-signal so that normally rapid responses to the 'go' signal evade inhibition. In studies of children with attention-deficit hyperactivity disorder (AD/HD), conduct disorder (CD), or both, slow stop-signal RTs are responsible for poor impulse control. Children with AD/HD inhibit less often than control children, even though their go-signal RTs are longer than normal children (Logan, et al, 1997; Oosterlaan, Logan, & Sergeant, 1998).

In a study with 136 undergraduate students, there was a significant relationship between impulsivity, as measured by the impulsivity subscale of the Eysenck Personality Inventory, and inhibitory control. Participants identified as highly impulsive had longer stop-signal RTs. This corroborated the idea that impulsive individuals did not necessarily have exceptionally rapid responding, but they did have exceptionally slow 'stop' responses (Logan, et al, 1997).

Similar to the passive-avoidance paradigm, in which impulsive individuals show deficits in response inhibition and have limited ability to inhibit strongly established response sets for reward, the stop-signal paradigm has a comparable dominant 'go' task. It is possible that the need to complete this 'go' task is so resilient, that there is impaired processing of a secondary cue like the stop-signal, resulting in a deficit in inhibitory control. Also similar to the passive-avoidance paradigm, limited clinical samples have been researched with this approach and it is not clear how it translates to other impulse control disorders, such as problem gambling.

Disinhibition and Gamblers. A number of studies have looked at Impulsive behaviour and disinhibition with respect to gamblers. Blaszczynski, Steel, and McConaghy (1997) investigate the role of impulsivity and antisocial personality features within 115 male problem gamblers. They report that impulsivity contributed to the behavioural and psychological disturbance experienced by the gamblers. They are unable, however, to demonstrate the exact relevance of impulsivity to problem gambling. Similarly, Carlton and Manowitz (1994) report only a small correlation between degree of problem gambling and impulsivity. In an earlier study, however, they state that

problem gamblers characterize themselves as having greater than normal numbers of ADD symptoms (Carlton & Manowitz, 1992).

Contrary to these studies, a number of researchers have reported strong associations between impulsivity and gambling. Castellani and Rugle (1995), in an analysis of data on 843 patients admitted to a Veteran's Association (VA) hospital, compare 126 problem gamblers without a history of substance dependence to 505 alcoholics and 212 cocaine addicts. The analysis shows that gamblers score significantly higher than the other groups on both impulsivity and the inability to resist cravings. The results of the study suggest that impulsivity in problem gamblers might be a function of lack of planning, rapid decision making, and taking action rather than waiting for entertaining or exiting experiences. These findings are consistent with those presented by Rugle and Melamud (1993) which suggest that gamblers exhibit significant problems with planning, anticipating consequences, learning from experiences, and inhibiting responses as required by complex situations.

Petry (2001a) examined the behavioural manifestations of impulsivity in problem gambling. The study finds that gamblers discount delayed rewards more frequently than controls, providing further evidence for the lack of planning and forethought suspected to be a core feature of impulse control in problem gamblers.

While there are a growing number of studies that have reported problem gamblers as attaining high scores on self-report measures of impulsivity (Blaszczynski, Steel, & McConaghy, 1997; Langewisch & Frisch, 1998), there remain significant discrepancies. As Castellani and Rugle (1995) note, the measurement scales utilized in the majority of studies assess impulsivity in terms of risk taking, excitement seeking, lack of planning,

making quick decisions, and taking action without thought. Although there might be a moderate correlation between these factors, they still represent separate, independently varying constructs. Furthermore, the instruments most commonly utilized to measure impulsivity in individuals (i.e., the Barratt Impulsiveness Scale, BIS-II; the Eysenck Personality Questionnaire, EPQ) do not assess the same sets of factors.

To illustrate this point, the definition of impulsivity in the psychological literature includes all or some of the following factors: motor impulsiveness such as unplanned and rapid reactions to stimuli without adequate processing of relevant information; a cognitive element such as a disregard for long-term consequences; and, a decreased sensitivity to negative consequences of behaviour (Barratt, 1985; Moeller, et al., 2001b). Seeing that there is growing evidence for an impulsive component to problem gambling, there is limited understanding as to how impulsivity manifests behaviourally and cognitively. Although some self-report investigations exist, there is little in the way of objective empirical research of the behavioural and cognitive expression of impulsivity within gamblers.

Limitations of Research

The preceding pages discuss the various research areas, studies, and theoretical models concerning impulsivity and gambling. As the discourse continues, however, it is clear that a number of shortcomings in the different topic areas exist. First, there is only partial agreement among researchers and theoreticians as to what impulsivity, as a construct, entails. Although impulsivity and impulsive behaviours are common diagnostic criteria for a variety of disorders, the measurement and description of the construct is equally as varied. As Castellini and Rugle (1995) point out, the scales and measures used

in current research are primarily self-report, and assess everything from lack of behavioural control to risk taking, with additional activities in between. Unfortunately, this lack of agreement between measurement instruments and definitions perpetuates the confused and sometimes contradictory accounts of what impulsivity describes within, and between, the impulse-related disorders.

Because of this confusion, two different sets of researchers have attempted to develop precise behavioural and cognitive measures of specific acts of control: learning from errors, and the ability to control thought and action. Newman and his colleagues have used the passive-avoidance learning paradigm to provide more specific accounts of individuals' ability to use discriminative stimuli (i.e., cues for punishment) to withhold inappropriate approach behaviour. Results with psychopaths, extraverts, and substance users have shown that all three groups are disinhibited when presented with both rewards and punishments. These groups are preoccupied with reward-seeking behaviours and are unable to learn to withhold responses from the punishments they experience. Further research, however, needs to determine if all the impulse-related disorders also experience this deficit. One such group that needs more research is gamblers.

In addition, the stop-signal paradigm is also a promising measure of both cognitive and motor disinhibition. Unfortunately, the primary research in this area has been with AD/HD and conduct disordered children, or adults who might present with ADD-like symptoms. Research using this paradigm with other populations can determine if cognitive and motor inhibition present themselves similarly in all impulsive psychopathologies and disorders.

Present Study

A number of studies show that gamblers experience impulsive urges and behaviours similar to extroverts, alcoholics, cocaine users, individuals with antisocial personality, and even adults and children with AD/HD (Blaszczynski, et al., 1997; Blaszczynski & Silove, 1996; Carlton & Manowitz, 1992; Castellani & Rugle, 1995; Goldstein, et al., 1985; Rosenthal & Lesieur, 1992; Roy, DeJong, & Linnoila, 1989; Rugle & Melamud, 1993; Steel & Blaszczynski, 1996).

Both the passive-avoidance learning model and the stop-signal paradigm have been studied in various impulse-related disorders. They have been able to illustrate the cognitive and motor processes involved in disinhibited behaviours within these disorders, but not within problem gamblers. Examining problem gamblers through these models will highlight objective aspects of problem gamblers from two validated theoretical approaches. This then provides another clinical and empirical benefit: by applying these models to problem gamblers, in addition to the populations that have already been studied, a clear link between specific inhibitory processes and different disorders adds to a more complete, process-based description of impulsive psychopathology (Nigg, 2000). Results from the tasks might clarify specific motor/behavioural and cognitive mechanisms, and provide specific objectives in assessment and treatment of those patterns that exacerbate and maintain gambling behaviours.

The stop-signal paradigm is a task that allows for the objective measurement of both cognitive and motor components of disinhibition, while the passive-avoidance paradigm provides more specific accounts of an individual's ability to use discriminative

stimuli (i.e., cues for punishment) to withhold inappropriate responses. Following from the literature available on these two models, it is hypothesized that:

1. a) Problem gamblers will exhibit a slow 'stopping' process when presented with a stop-signal while performing the primary RT task on the stop-signal paradigm.

   b) Problem gamblers will not have a corresponding increase in primary task RT. Therefore, RT to the primary task will be equal for problem gamblers and controls.

2. a) Problem gamblers will have difficulties withholding a response to avoid punishment when they are already primed for rewards (greater passive-avoidance errors compared to controls in the reward/punishment, *PALR*, condition)

   b) Problem gamblers will be equally proficient as controls on all other aspects of reward/punishment condition (*PALR*), reward only condition (*RRI*) or punishment only (*PANR*) conditions.

Methodology

Participants

Power Analysis. An a priori power analysis was conducted using the *G Power* computer program (Faul & Erdfelder, 1992). Power analysis revealed that an ideal situation for ANOVA measures, with high power (0.95) and a medium effect size (0.95), would require an N of 210 (105 participants in each group). Given that one of the groups was derived from a clinical population (Problem Gamblers), it was uncertain if this large an N would be feasible. A compromise analysis showed that a total N of 80 ($n = 40$ participants in each group) provided an acceptable power value of 0.82, with a medium effect size $d = .25$, at a $p = 0.05$.

Exclusion and Inclusion Criteria. Participants for this study were recruited through various community resources and the University of Windsor undergraduate participant pool. To be included in the study, subjects had to be a minimum of 18 years of age. Potential participants were directed to call the research lab. Those who called were asked to complete a telephone screening interview to determine suitability for the study. All potential participants were screened for a number of psychological diagnoses. For example, they were only included in the study if they did not endorse being depressed for a minimum of six months prior to participating, had not had a previous diagnosis, nor did they endorse the symptoms associated with the diagnosis, of Bipolar Disorder, Borderline Personality Disorder, or AD/HD.

Specific to the exclusion criteria, the requirement of no depression symptoms for at least six months was deemed important for the computer tasks which were reaction time based. Any motor or cognitive slowing which may accompany depressive symptoms

would confound the results of a reaction time task. In addition, the Bipolar Disorder, Borderline Personality Disorder, and AD/HD diagnoses were excluded from the current study as the larger Disinhibition study being conducted utilized these diagnostic categories as separate groups of clinical interest (please see Appendix E for complete list of measures in the larger research project).

Finally, the telephone interview also included the Eight Gambling Screen (Sullivan, 2001). This was a brief, eight item, yes/no questionnaire that was a rapid tool to identify possible gambling problems. Those who endorsed four or more of these items were invited down to the lab as potential PGs. Once a participant was in the office for the research appointment, the Canadian Problem Gambling Index (Ferris & Wynne, 2001) was completed. If the subject did not score greater than seven on this interview, their data was not included in the present research study as a Problem Gambler. Because of the use of a more stringent measure of problem gambling (see CPGI description in Tests and Materials for comparison to DSM-IV-TR diagnosis), there were fewer than anticipated subjects recruited into the problem gambling group.

With respect to the control group, participants again were screened using the same telephone screening interview. These potential subjects were deemed suitable if they met the same psychological diagnostic criteria as listed above. In addition, they would have answered fewer than four items in the keyed direction from the Eight Gambling Screen (Sullivan, 2001). All control subjects, however, did indicate that they had taken part in some type of gambling activity in the 12 months prior to the study. This was done to ensure that individuals in this group had exposure to gambling but did not experience any problems with it, nor were they in any way opposed to the idea of gambling in general.

Because of the nature of the study and inclusion criteria, it was difficult to find suitable control subjects. As a result, a large number of the controls came from the university undergraduate population.

In addition, some individuals self-referred to the study as potential experimental (PG) subjects. If these subjects did not meet criteria under the above mentioned guidelines, they were included in the study as potential CTRLs. As a result, three individuals were included as CTRLs which were subsequently dropped from the analysis. These three subjects were dropped because, although they did not meet criteria as PGs, nor did they currently meet criteria for any other Axis I or II diagnosis, two of these subjects reported (during the actual data collection session) that they had suffered two or more concussions requiring hospitalizations and two of three subjects reported heavy cocaine use in the past. As a result, the CTRL group was reduced to 37 subjects.

Participants categorized as problem gamblers had a score of 7.5 or higher on the Canadian Problem Gambling Index (CPGI). The non-problem gambling controls were individuals who indicated that they took part in some form of gambling in the 12 months prior to participating in the study (e.g., buying occasional lottery tickets, going to the casino), but had a CPGI score less than five. The CPGI categories are as follows:

0 = non-problem

1 – 2 = low risk gambling

3 – 7 = moderate risk gambling

8+ = problem gambling

Participant Demographics. The participants for this study consisted of a sample of 30 problem gamblers (PGs) (13 male, 17 female) and of 37 non-problem gambling

controls (CTRLs) (14 male, 23 female). There was a significant age difference between the PGs ($M = 35.2$, $SD = 16.60$) and CTRLs ($M = 25.78$, $SD = 9.45$), $t(43.85) = 2.77$, $p < 0.01$ (equal variances not assumed).

Although there were significant age differences found between the two groups, previous research by Logan, et al., had looked at age differences on the stop-signal task. It was found that there were no significant age differences between any of the adult age groups from young adults in their late teens and early 20's to older adults (55+). In addition, they found no gender differences (Logan, personal communication, April 10, 2006; Schachar, personal communication, April 10, 2006; Williams, Ponesse, Schachar, Logan, & Tannock, 1999; Bedard, Nichols, Barbosa, Schachar, Logan, & Tannock, 2002).

Unfortunately, no such data existed in the passive-avoidance literature so it is not clear how, if at all, age and gender influenced the results of the present study. Because this model is primarily based on psychopaths, limited work has been done with psychopathy in women. There are no studies that have used the passive-avoidance paradigm in mixed gender samples. Some might suspect that because passive-avoidance learning deficits have been empirically associated with psychopathy, and because psychopathy is more prominent in males, males ought thereby, be presumed to score higher on the passive-avoidance paradigm. Both Borderline and Histrionic Personality Disorder, however, have impulsivity among their diagnostic criteria, both are more frequently diagnosed among women, and scores on the passive-avoidance learning paradigm have been observed to be higher among incarcerated female borderline prisoners than among matched female prisoner controls (Hochhausen, Lorenz, &

Newman, 2002). Moreover, the present study concerns not impulsivity as such (which is at best, by all accounts already reviewed in the earlier chapter, a highly ambiguous construct), but rather a specific type of deficit in learning from experience. The passive-avoidance paradigm is based on Gray's model of behavioural inhibition in which such a deficit would be a heritable biological trait. There is, then, no reason to suspect that gender would substantially affect performance on a passive-avoidance learning task.

Regardless, the limited research that does exist has been inconsistent or irrelevant. One study by Vitale and Newman (2001) found that psychopathy was not associated with behavioural disinhibition in women. The study, however, was not directly assessing the performance of women on the passive-avoidance learning task, and to that extent is irrelevant. Gremore, Chapman, and Farmer (2005) assessed passive-avoidance learning in female inmates and found results that were consistent with the research conducted with men.

Participant Recruitment. Recruitment of subjects took place through a number of sources as part of a larger study conducted at the University of Windsor. These sources included the University of Windsor Undergraduate Participant Pool, Windsor Regional Hospital, and a number of other community mental health organizations. The community resources provided potential participants with an information sheet detailing the study and the participants' involvement. Upon handing out the information sheets, the individuals from the mental health treatment centres had no further involvement with any aspect of the research.

In addition to referrals from community treatment centres, posted flyers throughout the city indicated the purpose of the study, and contact information. These

flyers were a source of self-referral to the study posted in public areas such as the University of Windsor campus, libraries, public offices, and retail establishments.

Demographic data in the form of age, sex, etc., are described below. All participants (clinical and community) were at least 18 years of age.

Tests and Materials

Canadian Problem Gambling Index (CPGI) (Ferris & Wynne, 2001). The CPGI is a 129-item researcher administered interview. Of the 129-items answered by the participant, nine items are scored to produce a rating for degrees of gambling ranging from no problems with gambling to problem gambling. The CPGI consists of four primary dimensions: gambling involvement; problem gambling behaviour; adverse consequences; and problem gambling correlates. The items comprising the problem gambling behaviour and adverse consequences dimensions are scored (items Q75 to Q83). Each response, from item Q75 to Q83, is recoded into a numerical value (Never = 0, Sometimes = 1, Most of the time = 2, Almost always = 3, Don't know = 0, Refuse to answer = 0), and these numbers are then tabulated to calculate the total CPGI score (between 0 and 27).

Although the items in the gambling involvement and gambling correlates dimensions are not scored, the information provided by these items details the types of activities the gamblers engage in, the frequency of their play, the amount of money spent on different types of gambling behaviour, as well as the social and familial correlates that exist.

Upon scoring the items, individuals completing the CPGI fall into one of five groups: non-gambling, non-problem gambling, low-risk gambling, moderate-risk gambling, and problem gambling.

1. Non-gambling. Score on CPGI = 0. The non-gambling group consists of those individuals who have not gambled within the previous 12 months and, with the exception of the correlates and demographic questions, will have no responses for the majority of the items.
2. Non-problem gambling. Score on CPGI = 0. This group likely responds with 'never' to the behavioural problem indicators, although there is the possibility that there may be heavy monetary and time involvements. Gamblers presenting in this group will not have experienced many, if any, adverse consequences from their gambling and will not agree with the items assessing distorted cognitions.
3. Low-risk gambling. Score on CPGI = 1 to 2. Participants who score within this group will have responded 'never' to most of the behavioural problem items, but will still have one or more 'sometimes' or 'most of the time' responses. Although they may not have experienced any adverse consequences yet, they may be at risk if they are heavily involved in gambling and positively responded to at least two of the correlates questions.
4. Moderate-risk gambling. Score on CPGI = 3 to 7. Gamblers scoring within this group will have responded 'never' to most of the behavioural problem items, but will still have one or more 'most of the time' or 'always' responses. As with the previous group, gamblers may be at greater risk if

they are heavily involved with gambling and have responded positively to three or four of the problem gambling correlates.

5. Problem gambling. Score on the CPGI = 8 or greater. Although involvement in gambling may be at any level, it is most likely to be quite heavy for individuals in this group. These gamblers will have experienced adverse consequences from their gambling, may have lost control of their behaviour, and will be much more likely to endorse the cognitive distortion items.

With respect to reliability, utilizing Cronbach's alpha reliability coefficient, the CPGI had an alpha of 0.84, while the DSM-IV had an alpha of 0.76 when determining problem gambling. Upon retest, the CPGI had a test-retest reliability of 0.78 (Ferris & Wynne, 2001). Further, the criterion-related validity, or the accuracy with which the CPGI classifies participants using another measure as the reference point, is quite good with the DSM-IV, correlating at 0.83.

Within the present study, therefore, the PGs were those individuals who scored greater than 7.5 on this measure. These were individuals who regularly endorsed negative consequences associated with their gambling (e.g., significant loss of income, separation from family, etc.). These subjects, moreover, consistently endorsed those cognitive distortions identified as problematic, such as believing that gambling by a "system" will increase the chances of winning. The control subjects, on the other hand, while endorsing gambling behaviours, and possibly even some financial loss, did not have these same cognitive distortions, nor did they engage in those behaviours which would further negatively affect their lives as a result of gambling (e.g., committing crimes to get money to gamble).

Stop Signal Paradigm (computer-based task). The stop signal paradigm is based on the work of Logan, et al. (1984). The basic paradigm involves a standard choice reaction time task in which the subject discriminates between an X and an O. For example, the subject is instructed to press the '1' key if an X is presented on the computer screen. The subject is instructed to press the '3' key, if the letter O appears on the screen. Subjects get feedback with respect to their accuracy: 'correct' or 'incorrect'. On some of these trials, the subject is presented with a loud computer generated tone (stop-signal), telling them to stop their response to the primary discrimination reaction time task.

The entire task was on a computer. The subject sat before the monitor and was verbally described the task. Following the verbal description, written instructions were visible on the computer screen (Appendix A) and the participant completed a brief trial period to assure an understanding of what was required. The subject then completed three blocks of trials with a brief 10 second rest period between each block. Within each block, a small cross appeared in the middle of the screen for 500 milliseconds (ms) to orient the subject. The cross was followed by the presentation of the primary task stimulus for 5000 ms, at which point the subject had 2500 ms to respond before the next trial.

Each block consisted of 144 trials, of which 25% (36 trials) included the stop-signal. The stop-signal was randomly presented at one of six different intervals (with six presentations of each interval) after the primary task stimulus: 5 ms, 100 ms, 200 ms, 300 ms, 400 ms, and 500 ms. The most important data resulting from this task consisted of: 1) establishing if the subject was able to stop when told to (i.e., when the stop-signal sounds); and 2) the time it took for the subject to inhibit responding (stop-signal reaction time).

Passive-Avoidance Learning Task (computer-based task). The passive-avoidance learning task, based on the work of Newman, et al. (1985), is a successive go/no-go discrimination task presented on the computer. The subject sat before the monitor and was verbally described the task. Following the verbal description, the participant reviewed a written version of the instructions on the computer monitor (Appendix B). This task consists of three conditions presented in the following order: punishment and reward (*PALR*), reward only (*RRI*), and punishment only (*PANR*). In each of the conditions participants learned through trial and error that four of eight numbers are "good"/S+ and four are "bad"/S-. The S+ numbers were those that required an active response (pressing the space bar) and the S- numbers were those that required a passive response (not pressing the space bar). Each condition involved a different set of randomly selected eight numbers (four S+, four S-) with each number presented eight times (64 trials per condition, 192 total trials).

All of the numbers consisted of two digits, with no number repeated twice between conditions. In addition, all characteristics of the numbers (i.e., odd or even numbers, high or low numbers, numbers greater or less than 50, etc.) were differentially associated with either the S+ or S- numbers. For each trial, the stimulus number was presented for 2500 ms.

Through trial and error, participants were required to learn to press the spacebar when a S+ number appeared (active response), and not press the spacebar if a S- number appeared (passive response). For the *PALR* condition, participants received a reward, one dime, for a correct active response to an S+ only. Within this condition, they were also punished (lost a dime) for each incorrect active response to an S- (i.e., pressing spacebar

for "bad" number). In the *RRI* condition, each time the participant made a correct active response to an S+ (i.e., pressing spacebar for "good" number), or a correct passive response to an S- (i.e., not pressing spacebar for "bad" number), they received a reward (one dime). There was no punishment of incorrect responses. In the final condition, *PANR*, participants only received punishments (losing a dime). Therefore, they were punished for not responding to S+ numbers (incorrect passive responses) and for responding to S- numbers (incorrect active responses). Both the reward and punishment consequences were administered immediately after the subject made a response.

Throughout the conditions, there were two types of errors: errors of commission (active responding to S-) and errors of omission (failing to respond to S+). Errors of commission are passive-avoidance errors in that they represent a failure to inhibit responding that result in punishment.

For the first task, every participant started with 10 dimes. As previously stated, rewarding added a dime while punishing removed a dime. Prior to beginning the research trials, participants were primed for reward by an initial successive presentation of each of the S+ numbers in the first condition. This priming was immediately followed by the *PALR* condition without a noticeable break in the presentation of trials. The second and third conditions began with the number of coins left over from the previous condition, and the rewards and punishments continued based on the task design. At the completion of the task, a tally was taken of the total number of dimes won and participants kept their winnings.

The purpose of the task was to determine if a subject, when presented with appropriate cues for responding or not responding (i.e., S+ and S-), was able to learn

from previous experience of an anticipated reward, and was able to passively avoid responding (i.e., learning to withhold a response to get a reward rather than constantly responding in the hopes of a reward).

Procedure

Initial communication involved the potential subject contacting the researcher and indicating their interest in participating. At this point, they completed a brief telephone-screening questionnaire (Appendix C) to determine their suitability. During the initial contact with participants (i.e., during the telephone screen), individuals were presented with a verbal description of the study, told of the voluntary nature of the study, and told of their right to discontinue participation at any point. The researcher made note of verbal consent and signed that all procedures and expectations were disclosed. If a participant declined to consent, they were thanked for their time and no further contact was made.

The telephone interview was approximately 15 minutes in length and consisted of questions that assessed briefly for potential comorbid mood disorders, Axis II conditions, substance use/dependence, ADHD, eating disorders, and gambling problems. If the individual met inclusion criteria with respect to gambling problems and met all other inclusion/exclusion criteria, they were scheduled for a testing session. For the control group, individuals were included if they did not meet criteria for any Axis I disorder or Axis II disorder in the last 6 months, and met all other inclusion/exclusion criteria as detailed above.

For people who did not meet criteria, they were thanked for their time and informed that they were not accepted into the study. Some individuals did not clearly meet inclusion or exclusion criteria. These potential subjects were told that they would be

contacted regarding their participation within 48 hours. Their information was discussed between the researcher and her supervisor, and a decision was made regarding the study.

Once deemed suitable, the subject had the study explained to them, as well as the limits of confidentiality, and right to withdraw without consequences. Following the verbal explanation, the subject was asked to read and sign the consent form; a copy of which they received for their own records (Appendix D). The testing session involved administering the Canadian Problem Gambling Index, the stop-signal paradigm computer task and the passive-avoidance learning task to the participant, as well as all other tasks included in the larger study (see Appendix E). All tasks were presented in random order to avoid administration and presentation effects. The clinical and non-clinical control groups were tested in the research lab of Dr. Stephen Hibbard, located in Chrysler Hall South, University of Windsor campus.

Results

The following analyses did not include any statistical corrections for Type I error rate. Because of the small sample sizes and low power associated with a small *N*, a correction would exacerbate the problems of low power. Applying a correction would imply concern only for controlling the probability of a Type I error (rejecting the null hypothesis when the null hypothesis is true), without thought to limiting the probability of a Type II error (accepting the null hypothesis when the null hypothesis is false; Simon, 2006).

Participant Characteristics

With respect to individual characteristics, 13 problem gamblers and 11 controls had a previous psychiatric or psychological diagnosis provided by another practitioner. There was no significant difference in the rate of diagnoses between the two groups, $\chi^2(1) = 0.005, p = 0.94$. Although none of the participants had a previous diagnosis of Problem Gambling, a number of subjects from both groups (20 problem gamblers, 14 controls) acknowledged feeling depressed at some point in their lives. There was no significant difference in the rate of depression reported, $\chi^2(2) = 0.79, p = 0.67$.

There were some interesting significant differences between the two groups on a number of different gambling related variables. For example, 12 PGs bought lottery tickets at least once a month or greater, while 31 CTRLs purchased them less than once a month or never, $\chi^2(3) = 8.97, p < 0.05$. Similarly, nine PGs indicated they went to bingo once a month or more, and 11 others endorsed a slightly lower frequency of bingo playing, whereas 25 CTRLs stated they did not go to bingo at all, $\chi^2(3) = 10.05, p < 0.05$. Finally, 14 PGs gambled with VLTs or slots at least once a month or greater (six of

whom indicated playing the slots at least once a week) compared to 31 CTRLs who played the slots/VLTs less than once a month or never, $\chi^2(3) = 12.95, p < 0.01$.

With respect to their experiences with, and beliefs about, gambling, PGs and CTRLs exhibited a number of significant differences. Within the sample, 14 PGs versus 7 CTRLs indicated having a "big loss" when they first started gambling, $\chi^2(2) = 6.54$, $p < 0.05$; 24 PGs compared to 12 CTRLs had a "big win" when they first started gambling, $\chi^2(2) = 15.51, p < 0.001$; 18 PGs believed a strategy would increase their odds of winning while only nine CTRLs held that belief, $\chi^2(3) = 9.29, p < 0.05$; and, 20 PGs believed after losing a number of times in a row they were more likely to win, compared to only six CTRLs, $\chi^2(3) = 17.52, p < 0.01$.

In addition to their beliefs and experiences, PGs also endorsed a number of gambling related urges and problems that CTRLs did not. These significant differences included PGs having the urge to gamble if something bad were to happen [$\chi^2(1) = 20.31$, $p < 0.001$], PGs were more likely to think about or attempt suicide as a result of gambling [$\chi^2(2) = 7.50, p < 0.05$], more PGs had family members with a gambling problem [$\chi^2(1) = 8.19, p < 0.01$], and PGs had experienced more difficulties as a result of someone else's gambling [$\chi^2(2) = 6.51, p < 0.05$].

Finally, PGs and CTRLs differed significantly on the amount of time and money spent on all gambling activities, with PGs spending much more time, winning more money, and losing more money than the non-problem gambling comparison group (see Table 2). Overall, PGs were more likely to endorse those risk factors that have been associated with maintaining and exacerbating problem gambling behaviours (Ferris & Wynne, 2001).

Table 2

Independent t-tests for Time and Money spent Gambling in past 12 months (N = 67)

Group	*M*	*SD*	*t*	*df*	Cohen's *d*
Time spent gambling (minutes)					
Problem Gamblers	1142.95	2013.71			
Non-problem Gamblers	93.38	194.23	2.84	29.51* [t]	0.73[a]
Money spent gambling (dollars)					
Problem Gamblers	6651.67	9078.30			
Non-problem Gamblers	866.94	2687.44	3.36	33.48* [t]	0.86[a]
Money won gambling (dollars)					
Problem Gamblers	5520.90	7868.20			
Non-problem Gamblers	254.96	585.62	3.66	29.28* [t]	0.94[a]
Money lost gambling (dollars)					
Problem Gamblers	8867.57	13732.28			
Non-problem Gamblers	128.24	194.46	3.49	29.01* [t]	0.90[a]

* significant $p < 0.01$
[t] equal variances not assumed
[a] large effect size

The questions posed in this study were organized into three sections. Note, then, that the four main hypotheses stated on p.36 above are hereby addressed within the

context of related but ancillary analyses. While we couch the central principles of the theories undergirding Logan's theory and Newman's theory in these four hypotheses, there are also ancillary related analyses that are worth following up on.

The first hypothesis (Hypothesis 1a, p.36) and supporting analyses focused on the stop-signal paradigm and the 'stopping' process. It was hypothesized that PGs would exhibit slower 'stopping' processes than CTRLs. In other words, for PGs, on those 25% of all the trials in which a stop-signal sound occurred, the PGs would be slower to inhibit their 'go' process than would CTRLs. To address this hypothesis, a Repeated Measures Analysis of Variance compared PGs and CTRLs on 'stop' times across the three blocks of the stop-signal task. In addition to providing overall group differences, and although no hypotheses were formulated directly relating to block effects or block by group interactions, examining the results by block would allow us to see if the various blocks themselves had any effect.

It was also hypothesized (Hypothesis 1b, p.36) that PGs would not have a faster 'go' process than CTRLs. Similar to the analysis above, and with the same rationale regarding possible block effects or interactions, a Repeated Measures Analysis of Variance compared PGs and CTRLs on 'go' times across the three blocks of the stop-signal task.

The remaining two hypotheses focus on the performance of PGs on the passive avoidance paradigm: 1) that PGs would have greater commission (passive-avoidance) errors than CTRLs on trials which have the possibility of both punishment and reward (*PALR* trials), and 2) that PGs would be as proficient as CTRLs when presented with reward only conditions or punishment only conditions. Analyses involved an Independent

t-test to address Hypothesis 2a (p.36) comparing PGs and CTRLs on passive-avoidance errors. The second analysis was a Repeated Measures Analysis of Variance to examined type of error (commission versus omission) and type of consequence (*PALR*, *RRI*, *PANR*) for PGs and CTRLs (Hypothesis 2b, p.36).

Analysis 1: The 'stopping' process.

To begin, a review of the dataset identified instances of 11 cases with some missing data with respect to stopping times ($SSRT_{obs}$). Further examination of these cases showed two causes for the missing data: 1) responding on all, or almost all, trials (stop-signal and non-stop signal), and 2) correctly inhibiting all responses on stop-signal trials. That is, missing data was due to either near universal failure of stopping or near universal success in stopping.

For those subjects who responded to all (or nearly all) trials, these cases were further broken down into two categories: a) four subjects that were unable to inhibit their responding during experimental trials but correctly inhibited responses during practice trials; and, b) three subjects that did inhibit some responses, but not enough to meet mathematical requirements to determine the $SSRT_{obs}$ value as detailed in the stop-signal paradigm literature. The remaining four cases with missing data fell into the second causal category: they inhibited all responses on stop-signal trials in a given block of trials.

Cases with missing data were included in the following analyses. In order to not lose subjects due to missing $SSRT_{obs}$ data, various solutions to supply missing data were considered. One of the most obvious is to simply use the average from those cases for which the data are not missing. If the overall average is used, this completely washes out

any contribution from group membership, whereas if group average is used, this tends to bias unreasonably in favour of the between groups difference. One alternative method is to use observed values of variables demonstrated to be related to the targeted missing values in order to estimate those missing values; but use values of these variables that are drawn across the whole sample, rather than separately for each group. Thus, some contribution due to group membership is maintained, since the related variables are themselves determined by group membership. In the present case, values on $SSRT_{obs}$ were determined to be strongly related to commission errors[3] on stop-signal trials and to non-stop signal reaction times (non-SSRT).

Therefore, using subjects for whom data was complete, three distinct regression equations were computed, predicting $SSRT_{obs}$ from commission errors on stop-signal trials and from non-SSRT, for each block. These three equations were used to estimate values of $SSRT_{obs}$ for each instance in which the data were missing on that variable. That is, missing Block 1 $SSRT_{obs}$ values were estimated with an equation based on Block 1 commission errors and Block 1 non-SSRT; missing Block 2 $SSRT_{obs}$ values were estimated with an equation based on Block 2 commission errors and Block 2 non-SSRT; and missing Block 3 $SSRT_{obs}$ values were estimated with an equation based on Block 3 commission errors and Block 3 non-SSRT.

For Block 1, R for regression was significantly different from zero, $F(2, 53) = 32.65, p < 0.001$, with R^2 at 0.65 and the adjusted R^2 at 0.62. Block 2 R for regression was also significantly different from zero, $F(2, 53) = 19.18, p < 0.001$, with R^2 at 0.57 and the

[3] The term "commission error" in the stop-signal task refers specifically to those errors made on trials with a stop-signal sound and should not be confused with the commission errors referenced in the Passive-avoidance paradigm.

adjusted R^2 at 0.54. Finally, for Block 3, R for regression was significantly different from zero, $F(2, 53) = 69.05$, $p < 0.001$, with R^2 at 0.72 and the adjusted R^2 at 0.71. Altogether, 65% (62% adjusted) of Block 1, 57% (54% adjusted) of Block 2, and 72% (71% adjusted) of Block 3 of the variability in $SSRT_{obs}$ was predicted by number of commission errors on stop-signal trials and non-SSRT. Because these effect sizes are so large, use of these equations to estimate missing $SSRT_{obs}$ values was regarded as preferable to simply substituting means for $SSRT_{obs.}$

To address the first hypothesis, a three (within subjects) by two (between groups) two-way Repeated Measures Analysis of Variance was conducted, using stopping time across the three blocks of the study as the within-subjects variable and the two groups (PGs vs. CTRLs) as the between subject variables. This would provide information about the main effects of blocks, the main effects of groups, and any interaction between these two. A significant interaction would mean that Block 1, 2, or 3 or their pattern influenced mean stopping time ($SSRT_{obs}$) differently for the PGs than for the CTRLs. The results of this 3 x 2 analysis are diagrammed in Figure 3.

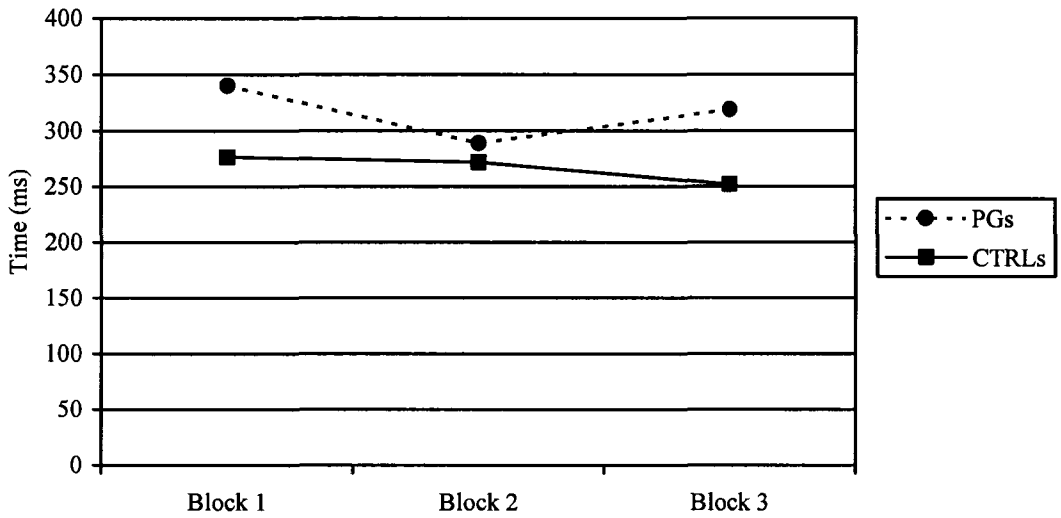
The main effect of blocks on the stopping time was nearly significant, $F(2, 130) = 2.51$, $p = 0.08$, $\eta_p^2 = 0.04$. There was no significant interaction between group membership and block, $F(2, 130) = 2.17$, $p = 0.12$, $\eta_p^2 = 0.03$, showing that any differences between the blocks were not moderated by group membership.

As predicted, there was a (marginally) significant difference between PGs and CTRLs, $F(1, 65) = 3.79$, $p = 0.056$, $\eta_p^2 = 0.06$, indicating a difference between the two groups on $SSRT_{obs}$ across the three blocks. For all three blocks, PGs had slower stopping times (Block 1: $M_{SSRTobs} = 340.32$ ms, $SD = 163.64$; Block 2: $M_{SSRTobs} = 288.87$ ms, $SD =$

100.33; Block 3: $M_{SSRTobs}$ = 319.00 ms, *SD* = 155.62) than CTRLs (Block 1: $M_{SSRTobs}$ = 276.57 ms, *SD* = 95.95; Block 2: $M_{SSRTobs}$ = 271.62 ms, *SD* = 102.42; Block 3: $M_{SSRTobs}$ = 251.82 ms, *SD* = 101.88) [see Figure 3: Mean Stopping Time ($SSRT_{obs}$) Across Blocks].

Figure 3

Mean Stopping Time ($SSRT_{obs}$) across Blocks (ms)



The effect sizes were large for Block 1 and for Block 3, while moderate for Block 2. PGs stopping times were considerably slower than CTRLs on Block 1 and on Block 3. Effect sizes for the differences between the two groups across blocks are provided in Table 3. Because this finding was in the predicted direction with a two-tailed significance level $p < 0.06$, we interpreted it as confirming the hypothesis of slower stopping times for the PG group.

By comparison, means, standard deviations, and effect sizes, collapsed across blocks, were computed for those participants for whom there was originally complete $SSRT_{obs}$ data. For PGs, $SSRT_{obs}$ across blocks was M_{Total} = 282.94, *SD* = 85.74, and for

CTRLs $SSRT_{obs}$ across blocks was $M_{Total} = 248.31$, $SD = 67.88$. The effect size for $SSRT_{obs}$ across blocks was Cohen's $d = 0.44$. These data were consistent in direction and size with that just reported which used the estimated values for $SSRT_{obs}$.

Table 3

Means, Standard Deviations, and Effect Sizes of Stopping Times ($SSRT_{obs}$) (N = 67)

Block	Group	*M*	*SD*	Cohen's *d*
1	Problem Gamblers	340.32	163.64	
	Non-problem Gamblers	276.57	95.95	0.48
2	Problem Gamblers	288.87	100.33	
	Non-problem Gamblers	271.62	102.42	0.17
3	Problem Gamblers	319.00	155.62	
	Non-problem Gamblers	251.82	101.88	0.51
Total	Problem Gamblers	316.03	124.62	
	Non-problem Gamblers	266.67	82.16	0.47

Given that the inhibitory response is known to be influenced by the offset from the 'go' signal, further Repeated Measures Analyses were performed based on the time of signal presentation following onset of the primary task presentation (sound delay; within-subject) by group (between-subjects). Although this is not directly related to the primary hypothesis of differences between the two groups on the stop-signal task, it would allow

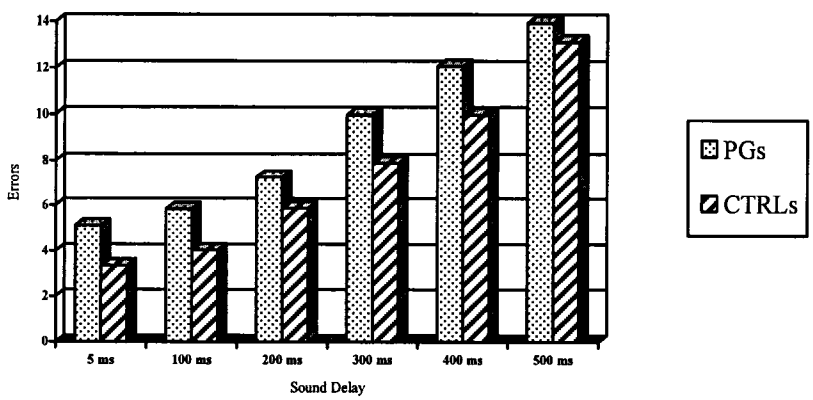
for an examination of whether the results of the present study are consistent with the literature and if the two groups might differ in the number of commission errors on stop-signal trials.

The analysis found a significant main effect for number of errors per sound delay, $F(5, 325) = 119.37, p < 0.001, \eta_p^2 = 0.65$, with the greatest errors at sound delays of 500 ms ($M_{errors} = 13.48$, $SD = 4.63$), followed by 400 ms ($M_{errors} = 10.91$, $SD = 5.61$), 300 ms ($M_{errors} = 8.82$, $SD = 6.16$), 200 ms ($M_{errors} = 6.48$, $SD = 6.34$), 100 ms ($M_{errors} = 4.84$, $SD = 5.84$), and 5 ms ($M_{errors} = 4.13$, $SD = 5.45$). This finding is to be expected and is almost universal in the stop-signal paradigm because the longer the stop-signal delay, the longer the response to the 'go' signal has had opportunity to develop to completion.

There was no significant interaction between group membership and the number of errors per sound delay, $F(5, 325) = 0.53, p = 0.75, \eta_p^2 = 0.01$. There was also no main effect for group, indicating that differences in the number of commission errors between PGs ($M_{errors} = 9.02$, $SD = 5.82$) and CTRLs ($M_{errors} = 7.37$, $SD = 5.49$) was not significant, $F(1, 65) = 1.73, p = 0.19, \eta_p^2 = 0.03$ (see Figure 4: Stop-signal errors by sound delay).

Figure 4

Stop-signal Errors by Sound Delay



Analysis 2: The 'go' process

As previously stated, this set of analyses looked to examine the differences between PGs and CTRLs on their 'go' processes, or their responses to the primary task. Given that the two processes, 'stop' and 'go' are hypothesized to be two independent processes, the 'go' process was not expected to be different for PGs and CTRLs.

To address this, a Repeated Measures, 3 (within-subjects) x 2 (between-groups) two-way Analysis of Variance was conducted, using 'go' reaction time across the three blocks of the study as the within-subjects variable and the two groups (PGs vs. non-PGs) as the between-subject variables. As with the analysis regarding stopping times, this would provide information about the main effects of blocks, the main effects of groups, and any interaction between these two. A significant interaction would mean that block 1, 2, or 3 or their pattern influenced mean 'go' time (RT) differently for the PGs than for the CTRLs.

Tests of between-subjects found no significant difference between PGs and CTRLs, $F(1, 65) = 0.13, p = 0.72, \eta_p^2 = 0.002$. In addition, there was no main effect of blocks on the go time, $F(2, 130) = 0.81, p = 0.45, \eta_p^2 = 0.012$. Finally, there was no significant interaction between group membership and block, $F(2, 130) = 0.57, p = 0.57, \eta_p^2 = 0.01$, showing that any differences between the blocks were not moderated by group membership.

Although the logic of hypothesis testing does not allow us to affirm the null hypothesis (no group differences), the high observed p value in this sample provides evidence to support the view that the two groups are not different in reaction time to the primary task.

Analysis 3: Passive Avoidance Paradigm

As stated, the hypotheses regarding the passive-avoidance paradigm posit that PGs would have greater commission (passive-avoidance) errors (but not omission errors) than CTRLs on trials that have the possibility of both punishment and reward (*PALR* trials), and PGs would respond similar to CTRLs when presented with *RRI* conditions or *PANR* only conditions.

To determine if PGs committed more passive-avoidance errors than CTRLs on *PALR* trials as predicted, an Independent t-test was run based on this *a priori* hypothesis. Results did not show a significant difference between PGs ($M_{\text{PAerrors}} = 11.93$, $SD = 5.49$) and CTRLs ($M_{\text{PAerrors}} = 10.06$, $SD = 4.79$ on the number of passive-avoidance errors committed on *PALR* trials, $t(66) = 1.49$, $p = 0.12$, two-tailed. There was a medium effect size, however, Cohen's $d = 0.37$, which suggests that perhaps the hypothesized effect was not observed because of lack of power. Effect sizes did not indicate similar performance for omission errors on the reward/punishment trials (see Table 4 for all means, standard deviations, and effect sizes). A 2 (error type) x 1 (consequence type) x 2 (group) planned comparison of omission versus commission errors on the *PALR* trials for PGs and CTRLs was also conducted. The main effect for error type was nearing significance on the reward/punishment trials, $F(1, 66) = 2.89$, $p = 0.08$, $\eta_p^2 = 0.04$. There was no significant interaction for error type by group, $F(1, 66) = 1.14$, $p = 0.30$, $\eta_p^2 = 0.02$.

To examine the performance of PGs and CTRLs further, a mixed two (type of error; i.e., commission error or omission error) by three (consequence type; i.e., *PALR*, *RRI*, *PANR*) Repeated Measures Analysis of Variance was conducted to determine if type of error and/or type of consequence varied by group membership. This analysis would

begin to identify if, and in what way, PGs and CTRLs differed in their performance on the passive-avoidance task (hypothesis 2b).

To begin, the three-way interaction for group by error type by consequence was not significant, $F(2, 128) = 0.45, p = 0.64, \eta_p^2 = 0.01$. This means that there was no effect predicated on simultaneous moderation of any two of the variables. There was no two-way interaction between type of error and group membership, $F(1, 64) = 0.14$, $p = 0.71$, $\eta_p^2 = 0.002$, indicating that group membership did not influence the difference between commission versus omission errors. Similarly, there was no two-way interaction between consequence type and group membership, $F(2, 128) = 0.26, p = 0.77, \eta_p^2 = 0.004$, showing that group membership also did not determine any differences in performance based on the three different types of consequences for error versus success.

The interaction between type of error and consequence type, however, is significant, $F(2, 128) = 8.65, p < 0.001, \eta_p^2 = 0.12$. As indicated in Figure 5 and Table 4, the difference between commission and omission errors is greatest on reward only trials and least on mixed consequence trials. Examination of the columns in Table 4 depicting cell means by error type reveals an interesting data point; namely, the rate of commission errors ascends monotonically across the mixed, punishment, and reward conditions, whereas rate of omission errors does the opposite, descending monotonically. Group does not moderate this effect. The greatest numbers of commission errors on the reward only trials is consistent with the literature in that the possibility of reward increases the likelihood of responding. This task makes it explicit that there is no possibility of punishment, thereby possibly increasing the proclivity to respond as opposed to inhibiting (therefore, less omission errors). The presentation of the tasks, with the punishment only

task following the reward/punishment and reward only tasks, may have created a responding or commission response set which was somewhat resistant to change, similar to that in the reward/punishment task; again resulting in greater responding.

Table 4

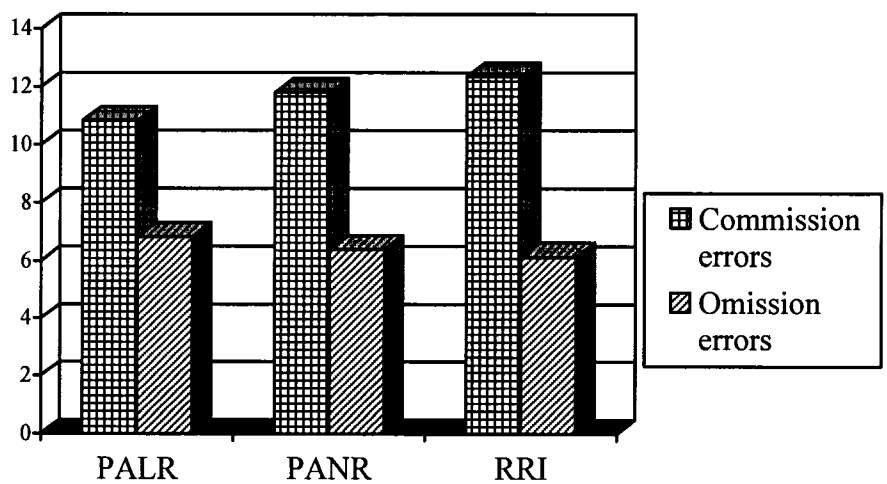
Means, Standard Deviations and Effect Sizes across Type of Trial and Consequence (N = 66)

	Commission Errors			Omission Errors		
	M	*SD*	Cohen's *d*	*M*	*SD*	Cohen's *d*
1.Reward/Punishment Trials						
Problem Gamblers	11.93	5.49		9.07	6.76	
Non-problem Gamblers	10.06	4.79	0.37	8.89	6.16	0.03
Total	10.91	5.16		8.97	6.39	
2. Punishment Only Trials						
Problem Gamblers	12.70	6.09		7.33	4.67	
Non-problem Gamblers	10.94	5.88	0.29	5.72	4.30	0.36
Total	11.74	6.00		6.45	4.51	
3. Reward Only Trials						
Problem Gamblers	13.03	6.24		6.70	6.40	
Non-problem Gamblers	12.22	6.71	0.12	5.56	4.36	0.21
Total	12.59	6.47		6.08	5.37	

With respect to the main effects, there was a significant main effect for type of error, $F(1, 64) = 46.48$, $p < 0.001$, $\eta_p^2 = 0.42$, with more commission errors (M_{errors} = 11.82, SD = 5.89) than omission errors (M_{errors} = 7.21, SD = 5.48). Using η_p^2 as the measure of association, type of error (commission or omission) accounted for 42% of the total variability in accuracy rate. Results of the analysis showed no significant main effect for total number of errors across type of tasks, $F(2, 128) = 1.18$, $p = 0.31$, $\eta_p^2 = 0.02$, indicating there was no significant difference between the number of errors on reward/punishment (M_{errors} = 9.94, SD = 5.78), punishment only (M_{errors} = 9.10 SD = 5.25), and reward only (M_{errors} = 9.34, SD = 5.92) trials.

Figure 5

Number of Commission and Omission Errors by Task



For overall between group effects, the difference between PGs and CTRLs collapsed across error type and consequence type, was not significant, $F(1, 64) = 2.13$, $p = 0.15$, $\eta_p^2 = 0.03$. Results of these analyses are presented in Table 5, means and standard deviations are listed in Table 4.

When further examining the performance of PGs and CTRLs on this task (see Table 5), the two groups do not appear to differ on the number of omission errors or on

Table 5

Mixed Repeated Measures ANOVA for Group by Error Type by Consequence – Main effects and Interactions (N = 66)

	M	*SD*	*F*	*df*	*P*	η_p^2
Interactions						
Error type x Consequence x Group			0.45	2, 128	0.64	0.01
Error type x Consequence			8.65	2, 128	<0.001	0.12
Consequence x Group			0.26	2, 128	0.77	0.004
Error type x Group			0.14	1, 64	0.71	0.002
Main Effects						
Error type			46.48	1, 64	<0.001	0.42
Commission Errors	11.82	5.89				
Omission Errors	7.21	5.48				
Consequence			1.18	2, 128	0.31	0.02
Reward/Punishment Trials	9.94	5.78				
Punishment Only Trials	9.10	5.25				
Reward Only Trials	9.34	5.92				
Problem Gamblers vs. Controls			2.13	1, 64	0.15	0.03

the number of commission errors on the reward only task. The Cohen's *d* values do suggest, however, that the two groups may perform differently on the punishment only tasks. That is, there are medium effect sizes for omission errors and commission errors for PGs versus CTRLs on the punishment only task with PGs having a greater number of errors. This is in contrast to the expectation that the PGs and CTRLs mean errors would not differ on all other conditions (e.g., everything except passive avoidance errors on reward/punishment trials).

As previously reported, the order of the tasks, with the punishment only task following the reward/punishment and reward only tasks, may have created a responding or commission response set which was somewhat resistant to change and the PGs may be more prone to this resistance than the CTRLs.

Discussion

The objective of this study was to apply and analyze the passive-avoidance learning model and the stop-signal paradigm to problem gamblers. Both of these models have been empirically studied with various impulse-related disorders, and have illustrated a number of cognitive and motor processes associated with disinhibition.

The analyses recounted above followed the sequence presented in the hypotheses. The first set of analyses, therefore, looked at the stop-signal task and the performance of the problem gamblers compared to controls. It was hypothesized that PGs would have slower stopping processes compared to the CTRL group. This position was originally presented and supported by the research of Logan, et al, with AD/HD children (e.g., Logan & Cowan, 1984; Logan, Cowan, & Davis, 1984; Logan, Schachar, & Tannock, 1997.).

As the model posits, and research has subsequently supported (see above references), all individuals are more likely to stop a response that has already started if a signal to stop comes closely after the initial task onset signal. The results of the current study are also consistent with this portion of the model. When this condition is met, and responses are *not* inhibited, the literature suggests one of two causes: 1) the individual is responding too slowly to the 'stop' signal even though the 'go' response is comparable within normal limits, or 2) the individual is responding too quickly to the 'go' signal.

The first cause, 1) above, is what the researchers have found in their studies of children with AD/HD. In other words, when compared to non-AD/HD controls, the AD/HD group did not have significantly faster response times on a forced choice task. In fact, the AD/HD group often had *slower* RTs on the primary 'go'

task, as well, although this feature is not always noted. Consistently, however, the AD/HD group did exhibit slower responses to the stop-signal (Logan, Schachar, & Tannock, 1997). The present study extended this finding to the sample of PGs vs. CTRLs: across the three blocks of stop-signal trials, the PGs were reliably slower than CTRLs, as predicted.

The second of the causes of disinhibition, that the individual is responding too quickly to the 'go' signal, was the basis of the next hypothesis: PGs would not have a faster 'go' response time. The work of Logan, et al, states that it is not a faster response time on the primary task that is responsible for disinhibition in AD/HD children but, rather, a slower 'stopping' time. It was expected, therefore, that a faster 'go' process would not be present in PGs. This was, in actuality, the case. Not only were PGs and CTRLs not significantly different in their respective 'go' RTs, PGs were actually, on average, somewhat slower than CTRLs on the primary go task, although not significantly slower.

The somewhat slower 'go' RT of the PGs could also help explain the similar number of commission errors for the two groups. In other words, while a slower stopping time would, in most instances, result in a corresponding increase in the number of commission errors (PGs had a slightly, though not significant, greater number of errors than CTRLs), the associated slower 'go' time would decrease the number of overall errors, as seen with the PGs.

Important is the similarity in presentation between PGs and the AD/HD children with whom the stop-signal paradigm was originally developed. One of the objectives of this study was to ascertain if other impulse-related disorders would have comparable

presentations on previously researched and supported models. This study, then, aids in clarifying motor and cognitive disinhibition in another diagnostic group. That is, the PGs in this study, like AD/HD children, are disinhibited not by their excessive or rapid responding to a go task. The deficit lies in their ability to shift from starting a response to stopping the response once the "normally" paced 'go' response is set in motion. As previously suggested, the stop-signal task establishes a dominant 'go' task in which the need to 'go' is so strong that the processing of any secondary direction (like 'stop') is impaired. As a result, there is a deficit in inhibitory control.

The passive-avoidance learning paradigm attempts to explain (dis)inhibitory processes in another impulse-related group: psychopaths. The research by Newman, et al., (e.g., Newman, 1987; Newman & Kosson 1986; Patterson & Newman, 1993) puts forth the idea that, for psychopaths and extroverts, the possibility of reward results in over-responding or the inclination to respond. In addition, there is a resistance to change behaviours regardless of actual or probable punishment. As a result, when rewards, or the possibility of rewards, are present, if an individual is punished, s/he is even more prone to responding with a greater disregard for the negative consequences. In other words, psychopaths lack response modulation because of their limited or non-existent consideration of behaviours or negative consequences when the possibility for reward exists. They do not learn from aversive events.

Based on this model, it was expected that PGs, like psychopaths, would not withhold responding under conditions where they could receive both rewards and punishments, and have a greater number of commission errors (passive-avoidance errors) than CTRLs. The results of the present study suggested a difference between the two

groups in number of passive-avoidance errors: the effect size was moderate. *Cohen's d* = 0.37, and in the predicted direction, that is, the problem gambling group had a greater number of passive-avoidance errors than the control group. This difference is sufficient to warrant further investigation, perhaps with a larger sample size. Consistent with the work of Newman, et al., the suggestion did exist that the PGs in this sample were comparable to psychopaths and extroverts on this task.

Extrapolated from the septal-hippocampal lesion model by Gray, this tendency to over respond in the face of both reward and punishment may be related to the exaggerated influence of reward. Although still only a suggestion, future study with a greater sample size may find that, following the model, any possibility of reward for the PGs produces an emotional/motivational state which, according to Gray, is related to the behavioural activation system (BAS), and results in both over-responding and an inability to shift response sets. When punishment or non-reward is presented, the PGs experience even greater arousal which, unlike with the CTRLs, results in even greater responding as opposed to response modulation.

The theory, however, goes on to state that, when there is only one type of consequence possibility (e.g., either reward or punishment but not both), this proclivity to over-respond or ignore consequence is not present since the individuals are not required to shift response sets (between avoidance and approach). The study results show that the two groups did not differ in commission or omission errors on the reward only task. On the punishment only task, however, PGs appeared to have a greater number of total errors than CTRLs. This result may be explained by the presentation of tasks within the study. The punishment only task was the third task completed after the reward/punishment and

reward only tasks. The PGs, based on Newman, et al's model, had an approach response set develop and become strengthened over the course of the previous two trials, making it more difficult for them to then shift to not responding.

As with the stop-signal task, the performance of PGs on the passive-avoidance learning task is a step towards linking problem gamblers in this study to the psychopaths upon whom this model was first developed. The PGs in this study, like the previously studied clinical populations, are possibly disinhibited in that they are not reflective upon negative consequences to moderate behaviours if the possibility of reward also exists. When both reward and punishment are likely, the PGs have difficulty shifting from dominant reward-seeking responses to punishment prevention. In situations with punishment only or reward only (e.g., non-competing response sets), the PGs in this study do not present with this deficit. Again, the results of this study help meet the objective of connecting two distinct impulse-related disorders with an objective measure of behavioural and motivational aspects of disinhibition.

Overall, the results of this study have shown that the disinhibition manifest in the PGs is not due to faster or indiscriminate responding compared to the control group. In fact, PGs are *slower* to even begin responding to the primary task. Their deficits are likely to be found in their unsuccessful ability to shift between a 'go' response that has already started and a secondary signal which negates the first, telling them to stop. It is not that they do not hear this signal to stop, but that they are slower (than controls) in their ability to stop their response from happening.

Secondly, the influence of consequences is also varied for PGs. Unlike assumptions that PGs do not learn from negative consequences, their difficulty in

inhibiting themselves is more apparent in situations where the possibility of a positive consequence is also present. In other words, knowing that a reward might be coming to them, PGs are emotionally/motivationally aroused to seek out that reward and are less likely to pay attention to the effects of a negative consequence/punishment, resulting in disinhibited behaviours such as indiscriminate gambling despite significant losses. If, however, there is no competition of consequences, if the PGs know they will only be rewarded and do not have to worry about a punishment, they are no more disinhibited than anyone else. A reward only (or a punishment only) possibility does not result in over-responding or indiscriminate actions.

Clinical Implications

In terms of clinical issues, Cummings and Gambino (1992) asked a group of 75 clinicians those dimensions considered important in the treatment of gambling problems. The paper detailed five main clusters identified by the clinicians as important to treatment: self-help/social support, crisis interventions, behavioural resources for change, psychodynamics of treatment, and crisis severity. Maurer (1994) has also reported the sincerity of the gambler's motivation for change as important to treatment efficacy.

In addition to the complexities inherent in treatment and diagnosing are the differences of opinion on what constitutes the appropriate treatment goals for problem gambling: abstinence versus controlled gambling. Abstinence proponents believe in the disease model of addictions and that, as an addiction, gambling cannot be cured, only arrested (Schwarz & Lindner, 1992). As a treatment goal, controlled gambling offers the advantage of appealing to gamblers who believe they would not be able to fully abstain from the problem behaviour. In addition, early success with controlled gambling would

have the benefit of increasing self-efficacy, possibly resulting in the gambler more readily accepting the idea of later abstinence (Ladouceur, 2005).

Cognitive theories of gambling posit that problem behaviours are maintained by erroneous beliefs or perceptions (Ladouceur & Walker, 1996). These erroneous beliefs include having the ability to predict outcome and having control over the game while excluding the idea of chance, and these need to be addressed in treatment. Cognitive treatments have differed in their goals with some aiming for controlled gambling while others focus on abstinence. For example, a social learning model based approach would have the gamblers learn the relationship between their emotions and gambling behaviours, how to effectively manage emotions, and return to appropriate social gambling (Sylvain & Ladouceur, 1992). In addition, treatment emphasizes an awareness of the variables influencing behaviours, learning self-management skills, and recognizing negative emotional states (Das, 1990).

Psychodynamic treatments, also with a treatment goal of controlled gambling, include intensive psychotherapy with an emphasis on finding the meaning behind, and consequences of, gambling behaviours (Rosenthal, 1992). This treatment focuses on the patient's omnipotence, confusion over boundaries, idealization of destructiveness, and lying. The effects of financial and interpersonal losses are considered in detail, and challenges are made to the compulsion to live within a fantasy world. Within the therapeutic relationship, work is centred on the realization and symbolization of early childhood dependence and the therapist's task is to encourage the transformation from financial gamble to emotional vulnerability (Whitman-Raymond, 1988).

Conversely, the abstinence proponents, such as Gamblers Anonymous (GA), believe the only treatment goal is to take responsibility for the gambling behaviours and stop gambling (Schwarz & Lindner, 1992). The four factors for success in GA include: identifying oneself as a gambler, relying on the support of the group, spirituality, and relabelling oneself. As with other 12-step programs, GA involves variations of the following: admitting being powerless over gambling; believing in a greater power; considerable self-reflection on thoughts, feelings and actions; and, making amends to others who have been harmed by the gambling.

According to Custer (1982), GA is successful for the following reasons: it demands honesty and responsibility; is non-judgemental; undercuts projection, rationalization, and denial; gives affection, personal support and concern; develops substitutes for the void left by no longer gambling; identifies serious implications of gambling; and identifies and corrects character deficits.

The present study, however, has identified other components of disinhibited behaviours in gambling which may be relevant to the treatment of gambling and can be incorporated to the treatments mentioned above. For example, on a behavioural level, the study has identified that, like AD/HD children, the gamblers in this study are not rapid responders, but are delayed in their stopping of a response that has already started. Within a social learning or cognitive-behavioural model, this awareness could be used to tailor the type of self-management skills which need to be taught.

Similarly, the gamblers in this study also have comparable behaviours to psychopaths in learning from rewards and punishments. Recognizing the deficits in learning from punishment if reward is also present would alter the course of teaching

behaviour management skills and countering erroneous beliefs and perceptions. It may then be beneficial to address the emotional or motivational arousal experienced by gamblers related to a physiological response. With an identified physiological response biological approaches to treatment, such as medications, can be considered.

In relation to presentation similar to other groups researched in greater depth, applying the treatments and interventions found to be helpful with these other groups may be an appropriate treatment avenue with gamblers as well. Most important to note, however, is the understanding that gamblers, as with other diagnostic groups, vary in the etiology of the problem, the factors which maintain the behaviours, and the impact of treatment. Because of the diversity of and within problem gamblers the treatment programs available should be equally varied to meet the needs of this population.

Limitations and Future Research

One of the more apparent limitations in this study was the relatively small sample size of problem gamblers and controls. With only 30 gamblers and 37 controls, a number of analyses showed nearing statistical significance, while the effect sizes for these same analyses were in the medium to large range. As such, future research on disinhibition and problem gambling would benefit from an even larger sample of moderate to serious problem gamblers and non-problem gambling controls. In addition, while stop-signal research has shown no gender differences or differences between younger and older adults, this data is not as clearly available for the passive-avoidance learning paradigm. Future research may need to pay closer attention to age and gender makeup of the various experimental and control groups.

Researchers, however, would do well to consider the difficulties inherent in not only recruiting subjects, but also screening for those subjects that meet research criteria. While a number of potential participants referred to the study subjectively felt or believed that they had a problem with gambling or were concerned about their gambling behaviours, these participants did not always meet research criteria according to the CPGI. For example, the CPGI addresses not only gambling behaviours (e.g., frequency of gambling, different types of gambling involvement), but also assesses for the distorted cognitions and adverse consequences associated with problem gambling (e.g., go back another day to win back losses; steal or sell things to fund gambling). For some individuals, while there may be heavy monetary expenditure or time involvement, they do not experience many, if any, adverse consequences from their gambling and will not agree with items that assess distorted cognitions.

Related to the idea of proper diagnosis is also that of definition. There is still considerable variety in the definition of problem gambling which, in turn, influences the measurement of the ill-defined construct. As a result, a heterogeneous group of gamblers are often combined into one group and one study (such as this) while they manifest the various dimensions of gambling in many different ways.

In addition to the difficulties inherent in recruiting a suitable sample, and defining what a suitable sample consists of, there were also some concerns with the administration of the measures themselves. As the tasks were computerized, instructions were given on the computer and were verbally provided by the research assistant running the subject. Following the written and verbal instructions, practice trials were run prior to the start of the actual research trials. For example, on the stop-signal task, the primary task was to

respond to an 'X' or an 'O' with a '1' or a '3', respectively. If a sound was presented, the subject was told to withhold the response.

Before beginning the research trials, the subject had the opportunity to practice 20 trials with and without sound. During this time, the research assistant was observing the subjects' responses to assess if they were, in fact, inhibiting the response when there was a sound, or displaying some nonverbal or verbal feedback at being aware that they have not withheld a response when they should have. It was found, however, that there were some participants who did not comprehend the task requirements, and did not follow through with the instructions. As a result, these subjects' data were not included in the analyses.

Finally, the results of the present study have shown some interesting relationships between motor and cognitive disinhibition and problem gambling. Future research that includes other diagnostic groups also characterized as "impulsive" will help to further clarify the manifestation of motor and cognitive disinhibition across a greater number of disorders, helping to refine diagnoses and diagnostic criteria. This refinement, in turn, will help to guide both the research on, and treatment of, these disorders. The direct application of these models to problem gambling has started the process of unifying the presentation of disinhibition across a number of diagnoses.

APPENDIX A
Written instructions for Stop-signal task (computer-based task)

GENERAL INSTRUCTIONS

1. The purpose of this study is to determine how quickly you can respond to a stimulus while also measuring accuracy. You will be presented with two different stimuli: an 'X' and an 'O'.
2. For this study, you will first see a plus sign (+) in the middle of the screen.
3. Immediately after this you will see either the letter 'X' or the letter 'O'
   When you see the letter 'X', hit the '1' key as quickly as you can.
   When you see the letter 'O', hit the '3' key as quickly as you can.
   HOWEVER, if you hear a sound after the letter, DO NOT hit either of the keys.
   Do not wait for the sound since it will not occur after each letter.
   Remember, we want to see how quickly and accurately you respond.
4. Before we begin, find the number '1' key and the number '3' key on the keypad which is on the right hand side of the keyboard. Place your left pointer finger in the '1' key and your right pointer finger on the '3' key.
5. Press any key to begin a practice trial.

INSTRUCTIONS FOR PRACTICE TRIAL 1

1. For this practice trial, you will first see a plus sign (+) in the middle of the screen.
2. Immediately after this you will see either the letter 'X' or the letter 'O'.
3. When you see the letter 'X', hit the '1' key (left finger) as quickly as you can.
4. When you see the letter 'O', hit the '3' key (right finger) as quickly as you can.
5. Press any key to continue with the practice trial.

INSTRUCTIONS FOR PRACTICE TRIAL 2

1. For this practice trial, you will first see a plus sign (+) in the middle of the screen.
2. Immediately after this you will see either the letter 'X' or the letter 'O'.
3. When you see the letter 'X', hit the '1' key (left finger) as quickly as you can.
4. When you see the letter 'O', hit the '3' key (right finger) as quickly as you can.
5. HOWEVER, if you hear a sound after the letter, DO NOT hit either of the keys.
6. Do not wait for the sound since it will not occur after each letter. Remember, we want to see how quickly and accurately you respond.
7. Before we begin, find the number '1' key and the number '3' key on the keypad which is on the right hand side of the keyboard. Place your left pointer finger on the '1' key and your right pointer finger on the '3' key.
8. Press any key to begin the practice trial.

INSTRUCTIONS BEFORE BEGINNING STUDY TRIALS

1. You have now completed the practice trials and are about to begin the study. You will be asked to complete these trials in the same way as the ones you just did.
2. In between sets of trials, you will see a blue screen. Continue to watch the screen until the next trial begins.
3. If you are ready to begin the study, please hit any key now.

APPENDIX B

Written instructions for Passive-avoidance task (computer-based task)

GENERAL INSTRUCTIONS BEFORE BEGINNING TASK

1. The purpose of this study is to determine how quickly you can respond to a stimulus while also measuring accuracy. You will be presented with one of eight different numbers.
2. If you choose to respond to a number, hit the zero '0' key as quickly as possible. If you choose not to respond do not hit anything.
3. Press any key to begin.

INSTRUCTIONS FOR TASK 1

1. For this next set of trials, you will be presented with one of eight different numbers.
2. Some of these numbers are "good" numbers and some these numbers are "bad" numbers. For the "good" numbers, if you hit the zero '0' key you will get a reward, 10 cents. For the "bad" numbers, if you hit the zero '0' key you will lose 10 cents.
3. Your task is to learn which numbers are "good" and which ones are "bad".
4. Press any key to begin.

INSTRUCTIONS FOR TASK 2

1. For the next set of trials, you will be presented with a different set of eight numbers from last time.
2. On these trials, you will get a reward (10 cents) when you respond to the "good" numbers, AND, you will also get a reward when you do not respond to the "bad" numbers. You will not lose any money for an incorrect response.
3. Press any key to begin.

INSTRUCTIONS FOR TASK 3

1. For the next set of trials, you will be presented with another different set of eight numbers.
2. On these trials, you will lose 10 cents when you do not respond to the "good" numbers, AND, you will also lose 10 cents when you do respond to the "bad" numbers. You will not win any money for a correct response.
3. Press any key to begin.

APPENDIX C

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 (Sullivan, 2001)

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.

APPENDIX D
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.
_____ Initials

APPENDIX E

Full List of Measures in Disinhibition Study

1. Beck Anxiety Inventory (BAI)

Beck, A. T., & Steer, R. A. (1993) *Beck Anxiety Inventory manual.* San Antonio, TX: Psychological Corporation.

2. Beck Depression Inventory (BDI – II)

Beck, A. T., & Steer, R. A. (1993) *Beck Depression Inventory manual.* San Antonio, TX: Psychological Corporation.

3. Baratt Impulsiveness Scale (BIS – 11)

Barratt, E. S. (2000). Barratt Impulsiveness Scale, Version 11 (BIS 11). In American Psychiatric Association, *Handbook of psychiatric measures* (pp. 691-693). Washington, DC: American Psychiatric Publishing Inc.

4. Behavioral Inhibition/Activation Scale (BIS/BAS)

Carver, C. S., & White, T. L. (1994). Behavioral inhibition, behavioral activation, and affective responses to impending reward and punishment: The BIS/BAS scales. *Journal of Personality and Social Psychology, 67,* 319–333.

5. Balanced Inventory of Desirable Responding – 7 (BIDR – 7)

Paulhus, D. L . (1998). *Manual for the Balanced Inventory of Desirable Responding (BIDR – 7).*Toronto, Canada: Multi-Health Systems Inc.

6. Connors' Adult ADHD Rating Scales

Conners, C. K., Erhardt, D., & Sparrow, E. (1999). Connors' Adult ADHD Rating Scales (CAARS). Toronto, Canada: Multi-Health Systems Inc.

7. Connors' Continuous Performance Test – II (CPT – II)

Connor, C. K. (2004). *Connors' Continuous Peformance Test – II Version 5.0 for Windows*. Toronto, Canada: Multi-Health Systems Inc.

8. Eating Disorders Questionnaire (EDQ)

Mitchell, J. E., Hatsukami, D., Eckert, E., & Pyle, R. (1985). Eating Disorders Questionnaire. *Psychopharmacology Bulletin, 21*, 1025-1043.

9. Eysenck Impulsiveness Questionnaire (I7)

Eysenck, B. G., Pearson, P. R., Easting, G., & Allsopp, J. F. (1985). Age norms for impulsiveness, venturesomeness and empathy in adults. *Personality and Individual Differences, 6*, 613-619.

10. Eysenck Personality Questionnaire – Revised (EPQ – R)

Eysenck, H. J., & Eysenck, S. B. G. (1993). Eysenck Personality Questionnaire – Revised. San Diego, CA: Educational and Industrial Testing Service.

11. Goldberg Factor Markers

Goldberg, L. R. (1992). The development of markers for the Big-Five factor structure. *Psychological Assessment, 4*, 26-42.

12. Generalized Reward and Punishment Expectancy Scale (GRAPES)

Ball, S. A., & Zuckerman, M. (1990). Sensation seeking, Eysenck's personality dimensions and reinforcement sensitivity in concept formation. *Personality and Individual Differences, 11*, 343–353.

13. Millon Clinical Multiaxial Inventory – III (MCMI – III)

Millon, T., Millon, C., Davis, R. (1994). Millon Clinical Multiaxial Inventory – III: Manual for MCMI – III. Minneapolis: National Computer Systems.

14. Personality Diagnostic Questionnaire 4+ (PDQ-4+)

Hyler, S. E. (1994a). *The Personality Diagnostic Questionnaire 4+*. New York: New York State Psychiatric Institute.

15. Relationship Questionnaire (RQ)

Bartholomew, K. & Horowitz, L. M. (1991). Attachment styles among young adults: A test of a four-category model. *Journal of Personality and Social Psychology, 61,* 226-244.

16. Relationship Scales Questionnaire (RSQ)

Griffin, D., & Bartholomew, K. (1994). Models of the self and other: Fundamental dimensions underlying measures of adult attachment. *Journal of Personality and Social Psychology, 67*, 430-445.

17. Structured Clinical Interview for DSM-IV Axis II Personality Disorders Questionnaire (SCID – II PQ)

First, M. B., Spitzer, R. L., Gibbon, M., Williams, J.B.W., & Benjamin, L. (1994). *Structured clinical interview for DSM-IV axis II personality disorders (SCID – II), version 2.0.* New York: Biometrics Research Department, New York State American Psychiatric Association.

18. Sensitivity to Punishment and Sensitivity to Reward Questionnaire (SPSRQ)

Torrubia, R., Avila, C., Molto, J., & Caseras, X. (2001). The sensitivity to punishment and sensitivity to reward questionnaire (SPSRQ) as a measure of Gray's anxiety and impulsivity dimensions. *Personality and Individual Differences*, 31, 837-862.

19. Shipley Institute of Living Scale (SILS)

Zachery, R. A. (1986). Shipley Institute of Living Scale: Revised manual. Los Angeles: Western Psychological Services.

20. Self-report Psychopathy Scale (SRP – II)

Hare, R. D., Harpur, T. J., & Hemphill, J. F. (1989). [Scoring pamphlet for the self report psychopathy scale: SRP – II]. Unpublished document, Simon Fraser University, Vancouver.

21. State-Trait Anger Expression Inventory – II (STAXI – II)

Spielberger, C. D. (1999). *Manual for the State-Trait Anger Expression Inventory-2.* Odessa, FL: Psychological Assessment Resources.

22. Tridimensional Personality Questionnaire (TPQ)

Cloninger, C. R. (1987) A systematic method for clinical description and classification of personality variants. A proposal. *Archives of General Psychiatry, 44*, 573–588.

23. Urgency, Premeditation, Sensation Seeking, Perseverance Scale (UPPS)

Whiteside, S.P., Lynam, D.R., Miller, J.D., & Reynolds, S.K. (2005). Validation of the UPPS impulsive behavior scale: a four-factor model of impulsivity. *European Journal of Personality, 19,* 559-574.

24. Weinberger Adjustment Inventory (WAI)

Weinberger, D. A., Feldman, S. S., Ford, M. E., & Chastain, R. L. (1989). *Construct validity of the Weinberger Adjustment Inventory.* Unpublished manuscript.

25. Yale-Brown Obsessive Compulsive Scale (Y – BOCS)

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., Heninger, G. R., Charney, D. S. (1989). The Yale – Brown Obsessive Compulsive Scale. *Archives of General Psychiatry, 46*, 1006-1011.

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