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Internalizing and externalizing psychopathology in problem and pathological gamblers: Factor structure and gambling subtypes

Aleksandar Milosevic
University of Windsor

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INTERNALIZING AND EXTERNALIZING PSYCHOPATHOLOGY IN PROBLEM
AND PATHOLOGICAL GAMBLERS:
FACTOR STRUCTURE AND GAMBLING SUBTYPES

by
Aleksandar Milosevic

A Dissertation
Submitted to the Faculty of Graduate Studies
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in Partial Fulfillment of the Requirements for
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Windsor, Ontario, Canada
2011
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Internalizing and Externalizing Psychopathology in Problem and Pathological Gamblers:
Factor Structure and Gambling Subtypes

by

Aleksandar Milosevic

APPROVED BY:

Dr. R. Gupta, External Examiner
McGill University

Dr. K. Gorey
Department of Social Work

Dr. A. Scoboria
Department of Psychology

Dr. R. Frisch, Co-Advisor
Department of Psychology

Dr. D. Ledgerwood, Co-Advisor
Department of Psychology

Dr. F. Omorodion, Chair of Defense
Faculty of Graduate Studies

26 August 2011

DECLARATION OF PREVIOUS PUBLICATION

This dissertation includes one original paper that has been previously published in a peer reviewed journal, as follows:

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Chapter 2	Milosevic, A., & Ledgerwood, D. M. (2010). Subtyping of pathological gambling: A comprehensive review. <i>Clinical Psychology Review</i> , 30(8), 988-998.	Published

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ABSTRACT

Problem and pathological gamblers (PPGs) present with various forms of psychopathology and maladaptive personality traits. It is unknown how psychopathology and personality traits are related in PPGs. Furthermore, some suggest the heterogeneity of PPGs supports classification of gamblers into distinct subtypes. The current study examined the utility of the internalizing-externalizing model (e.g., Krueger, 1999) in conceptualizing the structure of psychopathology in gamblers, and explored differences in gambling subtypes derived from the pathways model of PPG (Blaszczynski & Nower, 2002). One hundred and fifty (N = 150; 50% male) PPGs were recruited from the community, and assessed using measures of psychopathology, personality, and gambling behaviour. Results suggest the structure of psychopathology in PPGs consists of latent internalizing and externalizing dimensions associated with negative emotionality and impulsivity, respectively, and behaviourally conditioned (or low pathology) and antisocial impulsivist (or externalizing) gamblers can be differentiated from one another. Clinical implications of results, as well as directions for future research, are discussed.

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TABLE OF CONTENTS

DECLARATION OF PREVIOUS PUBLICATION	iii
ABSTRACT	iv
ACKNOWLEDGEMENTS	v
LIST OF TABLES	x
LIST OF FIGURES	xi
LIST OF APPENDICES	xii
CHAPTER	
I. INTRODUCTION	1
II. REVIEW OF THE LITERATURE	5
Problem and Pathological Gambling (PG): Definition and Prevalence	5
Psychiatric Comorbidity in PGs	6
PG and substance use disorders (SUDs)	7
PG and unipolar mood disorders	11
PG and anxiety disorders	14
PG and attention-deficit/hyperactivity disorder (ADHD)	16
PG and antisocial personality disorder (ASPD)	18
PG and Personality Traits	21
PG and impulsivity/sensation seeking	21
PG and negative emotionality	25
Covariation of Psychopathology in PGs: Utility of the	

Internalizing-Externalizing Model	27
Internalizing-Externalizing Model in PGs?	32
Subtyping PGs based on Psychopathology and Personality	33
Early subtyping of PGs (1970 – 2001)	34
The emergence of three pathways subtypes (2002 – 2010)	41
Aims of Current Study	51
III. METHODOLOGY	55
Sample	55
Recruitment	55
Measures	56
Problem and PG	56
Gambling motivation	57
Multidimensional Personality Questionnaire	57
Unipolar mood, anxiety, substance use, and antisocial behaviour disorders	59
Attention-deficit hyperactivity disorder (ADHD)	59
Impulsivity	60
Childhood maltreatment	62
Additional variables	63

Procedure	63
Data Analysis	65
IV. RESULTS	66
Descriptive Statistics	66
Hypothesis 1: Factor Structure of Psychopathology	68
Hypothesis 2: Subtypes of PPGs	79
Comparing Gambling Subtypes	81
Substance Use Variables	85
Psychiatric Variables	87
Antisocial and ADHD-related Variables	89
General Personality Traits and Impulsivity Traits	91
Childhood Maltreatment Variables	92
V. DISCUSSION	98
Hypothesis 1: Factor Structure of Psychopathology	98
Hypothesis 2: Subtypes of PPGs	105
Limitations of Current Study	114
Sample	114
Cross-sectional design	115
Measures	115
Data	115
Pathways model	116

Strengths of Current Study	116
Sample	116
Measures	117
Internalizing-externalizing model	118
Pathways model	118
Future Directions	118
REFERENCES	121
APPENDICES	157
VITA AUCTORIS	193

LIST OF TABLES

Table 1. Pattern matrix for two-factor structure of psychopathology (N = 150)	76
Table 2. Structure matrix for two-factor structure of psychopathology (N = 150)	77
Table 3. Correlations between psychopathology factors and Multidimensional Personality Questionnaire (MPQ) personality traits (N = 150)	79
Table 4. Subtype comparisons on Multidimensional Personality Questionnaire (MPQ) personality traits	81
Table 5. Subtype comparisons on demographic and gambling-related variables	83
Table 6. Subtype comparisons on substance use variables	86
Table 7. Subtype comparisons on psychiatric variables	88
Table 8. Comparing subtypes on antisocial and ADHD-related variables	90
Table 9. Correlations between impulsivity variables (N = 150)	93
Table 10. Subtype comparisons on general personality and impulsivity traits	95
Table 11. Subtype comparisons on childhood maltreatment variables	96

LIST OF FIGURES

Figure 1. Percentage of participants engaging in gambling activities (at least twice per week in past year) (N = 150)	68
Figure 2. Percentage of participants with at least one DSM-IV-TR disorder criteria (N = 150)	74
Figure 3. Prevalence rates of DSM-IV-TR disorders (N = 150)	75

LIST OF APPENDICES

Appendix 1. Comparison by gender	157
Appendix 2. Comparison by recruitment source	164
Appendix 3. Scree plot	172
Appendix 4. Pattern and structure matrices for three-factor structure of psychopathology	173
Appendix 5. Pattern and structure matrices for four-factor structure of psychopathology	177
Appendix 6. Community advertisement	181
Appendix 7. Participant pool advertisement	182
Appendix 8. Community population consent form	183
Appendix 9. Undergraduate population consent form	187
Appendix 10. Compensation receipt form	191
Appendix 11. Treatment referral form	192

CHAPTER I

INTRODUCTION

Pathological gambling (PG) is characterized by a failure to resist the impulse to gamble despite serious personal and social consequences (American Psychiatric Association, APA, 2000). It is a disorder typified by various comorbid psychiatric conditions and underlying maladaptive personality traits. For example, pathological gamblers (PGs) exhibit elevated rates of current and lifetime substance use (el-Guebaly et al., 2006; Petry, Stinson, & Grant, 2005), mood (Kim, Grant, Eckert, Faris, & Hartman, 2006; Potenza, Xian, Shah, Scherrer, & Eisen, 2005), anxiety (Black & Moyer, 1998; Kerber, Black, & Buckwalter, 2008), and personality disorders (Blaszczynski & Steel, 1998; Fernandez-Montalvo & Echeburua, 2004), as well as marked levels of impulsivity (Blaszczynski, Steel, & McConaghy, 1997; Ledgerwood, Alessi, Phoenix, & Petry, 2009; Vitaro, Arseneault, & Tremblay, 1999) and neuroticism (Bagby et al., 2007; MacLaren, Best, Dixon, & Harrigan, 2011) compared with the general population. These psychiatric disorders and maladaptive personality traits, however, are not present in all PGs, and considerable heterogeneity is found in the presentation of individuals with PG.

The heterogeneity of psychiatric disorders and maladaptive personality traits in PGs may be understood from the perspective of the *internalizing-externalizing* model of psychopathology. The internalizing-externalizing model postulates that patterns of psychiatric comorbidity adhere along *internalizing* (i.e., the tendency to express psychological distress inward) and *externalizing* (i.e., the tendency to express psychological distress outward) dimensions influenced by core, underlying personality processes. At a disorder level, the internalizing dimension consists of unipolar mood (i.e.,

major depressive disorder, dysthymic disorder) and anxiety (e.g., generalized anxiety disorder, posttraumatic stress disorder) disorders, while the externalizing dimension consists of substance use disorders (SUDs) and antisocial behaviour disorders (i.e., conduct disorder, antisocial personality disorder) (Kramer, Krueger, & Hicks, 2008; Krueger, 1999; Krueger & Finger, 2001; Waldman & Slutske, 2000). Furthermore, the personality traits of neuroticism/negative emotionality and impulsivity underlie the internalizing and externalizing dimensions, respectively, and are considered factors that partially explain the specific covariations between internalizing and externalizing disorders (Krueger et al., 2001; Krueger, McGue, & Iacono, 2001). Given that these psychiatric disorders and maladaptive personality traits are elevated in PGs, it is possible the internalizing-externalizing model offers utility in conceptualizing the heterogeneity seen in these individuals.

The internalizing-externalizing model has yet to be studied in PGs. However, recent theoretical work has attempted to organize the heterogeneity of PGs by conceptualizing gambling *subtypes* with a distinct underlying psychopathology and a unique presentation, and this work has similarities with the internalizing-externalizing model. The *pathways model* of problem and PG (PPG; Blaszczynski & Nower, 2002) identifies three subtypes of gamblers, each arriving at disordered gambling through a specific mechanism. *Emotionally vulnerable* gamblers, for example, present with pre-morbid unipolar mood and/or anxiety disorders, and gamble to escape dysphoric and distressful feelings. *Antisocial impulsivist* gamblers present with increased rates of SUDs and antisocial personality disorder, and their gambling is associated with elevated impulsivity and potentially with neurobiological deficits. Finally, the *behaviourally conditioned* gambler's gambling is based primarily on the behavioural contingencies

offered by gambling rather than on latent psychopathological processes. Research directly investigating the pathways model is beginning to emerge (Stewart, Zack, Collins, Klein, & Fragopoulos, 2008; Turner, Jain, Spence, & Zangeneh, 2008; Vachon & Bagby, 2009), and the existing gambling subtyping literature appears to support the validity of these subtypes (see Milosevic & Ledgerwood, 2010, for a review).

From the perspective of the internalizing-externalizing model, the emotionally vulnerable and antisocial impulsivist gamblers postulated by the pathways model can be conceptualized as internalizing and externalizing gamblers, respectively, while behaviourally conditioned gamblers can be considered ‘low pathology’ gamblers who present with little co-occurring psychopathology. That is, the emotionally vulnerable gambler exhibits elevated levels of unipolar mood and/or anxiety disorders, disorders characteristic of the internalizing dimension. The antisocial impulsivist gambler has SUDs, antisocial personality disorder, and elevated impulsivity, disorders and traits characteristic of the externalizing dimension. Furthermore, the behaviourally conditioned gambler demonstrates relatively low levels of psychopathology and can be understood as neither having major tendencies toward the internalization or externalization of psychological distress. Given the apparent congruence between the gambling subtypes postulated by the pathways model and the internalizing and externalizing dimensions of psychopathology, the internalizing-externalizing model may be a useful framework to study the pathways model of PPG.

There are two specific aims of the current study:

1. Examining the underlying structure of psychopathology in PPGs, as well as correlations between factors and higher-order personality traits, to explore the

utility of the internalizing-externalizing model in conceptualizing the heterogeneity seen in individuals with gambling disorders.

2. Examining differences in internalizing and externalizing psychopathology, and gambling and other psychosocial variables in gambling subtypes. Gamblers were classified using the pathways model as a guide, and predictions about subtype differences were made using both the internalizing-externalizing and pathways models.

Providing a heuristic (i.e., the internalizing-externalizing model) for organizing and conceptualizing the heterogeneity in PPGs is critical to advancing knowledge of the etiology and course of psychopathology in individuals with gambling disorders. In addition, examining the structure of psychopathology in gamblers will reveal underlying psychological dimensions that may have relevance to the pathogenesis and maintenance of PPG. Studying gambling subtypes can aid in the development of assessment and treatment strategies that address individual differences in clinical presentation. If it can be shown that subtypes of gamblers differ on psychopathological and personality variables, assessment tools can be developed to differentiate gambling subtypes to allow treatment providers to adequately address the unique psychological factors that underlie specific gamblers' disordered gambling. In addition, identification of subtypes of gamblers will facilitate the study of underlying genetic and neurobiological mechanisms, advance understanding of diagnostic comorbidity, and help identify psychiatric and personality factors that influence differential responses to disordered gambling treatment.

CHAPTER II

REVIEW OF THE LITERATURE

Problem and Pathological Gambling (PG): Definition and Prevalence

Pathological gambling (PG) is categorized as an impulse control disorder in the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV-TR; APA, 2000), and is defined as a pattern of “maladaptive gambling behavior that disrupts personal, family, or vocational pursuits” (p. 671). The diagnosis of PG is made when an individual meets at least five of the 10 DSM-IV-TR diagnostic criteria (p. 674; e.g., is preoccupied with gambling, needs to gamble with increasing amounts of money in order to achieve the desired excitement). Distinguished from PG, *problem gambling* is a condition in which an individual experiences distress or impairment as a result of gambling but the severity of the gambling behaviour does not meet the diagnostic threshold for PG. Problem gambling is typically defined as an endorsement of three or four criteria on assessment measures such as the South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987) or other gambling instruments (e.g., National Opinion Research Centre DSM-IV Screen for Gambling Problems, NODS, Gerstein et al., 1999).

Lifetime prevalence estimates of PG have been found to range between 0.4% to 2.0% in epidemiological studies of adults variably conceptualizing PG using gambling screening measures or DSM-IV diagnosis (Petry, Stinson, & Grant, 2005; Shaffer, Hall, & Vander Bilt, 1999; Welte, Barnes, Wieczorek, Tidwell, & Parker, 2001). In addition, community prevalence rates of problem gambling for adults have typically been found to be approximately 4% (Welte et al., 2001). A meta-analysis (Shaffer et al., 1999) of 119

prevalence studies in North America found that the mean 12-month SOGS probable PG rate was 1.12% while the mean problem gambling rate was 2.16% among adults.

Psychiatric Comorbidity in PGs

Elevated rates of psychiatric disorders have been extensively documented in clinical (Specker, Carlson, Edmonson, Johnson, & Marcotte, 1996) and epidemiological (Petry, Stinson, & Grant, 2005) samples of PGs. Comorbidity with psychiatric conditions, such as SUDs and unipolar mood disorders, appears to be the rule rather than the exception in PGs (Crockford & el-Guebaly, 1998; Petry et al., 2005). Furthermore, some studies (e.g., Ibanez et al., 2001) have found that PG severity increases linearly in treatment-seeking gamblers with the number of comorbid psychiatric diagnoses. While the increased prevalence of psychopathology in PGs is evident, our understanding of the effect of co-occurring psychiatric disorders on gambling disorders remains limited (Crockford & el-Guebaly, 1998; Hodgins & el-Guebaly, 2010; Raylu & Oei, 2002).

The study of psychiatric comorbidities in PGs can enhance knowledge of the determinants of disordered gambling. That is, concurrent psychiatric conditions may contribute to the development and maintenance of PG. In this regard, Petry and colleagues (2005) have noted that understanding co-occurring PG and psychiatric conditions is necessary to generate hypotheses regarding the etiology of PG. Furthermore, Raylu and Oei (2002) have suggested that knowledge regarding the comorbidity of psychiatric disorders and PG is especially relevant due to the lack of a comprehensive model of the 'pathogenic process' from controlled gambling to PG. Understanding rates of psychiatric disorders in gamblers is also important for establishing intervention strategies for affected individuals (Petry, Stinson, & Grant, 2005). Gamblers with any, or

specific, comorbid psychiatric conditions may differentially respond to various forms of pharmacological and/or psychotherapeutic treatment interventions. Furthermore, psychiatric comorbidity may impact the recommended duration or intensity of PG treatments (Crockford & el-Guebaly, 1998).

A comprehensive review of psychiatric comorbidity in PG Crockford and el-Guebaly (1998) concluded that PG is highly comorbid with *specific* psychiatric disorders. Others (e.g., Petry, Stinson, & Grant, 2005; Raylu & Oei, 2002) have also noted associations between certain psychiatric disorders and PG, and have suggested these covariations are likely the result of basic factors that cause or contribute to comorbid disorders. Very little is known, however, about what factors contribute to covariation between PG and comorbid psychiatric disorders. The following review of psychiatric comorbidity in PGs explores disorders that are relevant to the internalizing-externalizing model, that is, unipolar mood disorders, anxiety disorders, SUDs, and antisocial behaviour disorders. These psychiatric factors, in addition to attention-deficit/hyperactivity disorder (ADHD), also play prominent roles in differentiating Blaszczynski and Nower's (2002) pathways model subtypes. The literature review also highlights personality factors (i.e., impulsivity, negative emotionality) that are suggested by the internalizing-externalizing model to explain the covariation between these psychiatric disorders. These personality traits are important in the pathways model as well.

PG and substance use disorders (SUDs). Substance use disorders (SUDs; i.e., alcohol abuse and dependence, and drug abuse and dependence) are among the most common psychiatric disorders associated with PG, and are a form of externalizing psychopathology specifically relevant to the antisocial impulsivist gambler (Blaszczynski

& Nower, 2002). Extensive literature using clinical and community samples of PGs has shown that 25% to 75% of PGs have a SUD in their lifetime (Crockford & el-Guebaly, 1998; Raylu & Oei, 2002). This rate is elevated relative to the lifetime prevalence of SUDs of approximately 15% in the general population (Kessler et al., 2005).

Correspondingly, 10% to 25% of patients with SUDs meet criteria for PG (Crockford & el-Guebaly, 1998; Raylu & Oei, 2002). PG and SUDs share numerous features in diagnostic, clinical, physiological, and behavioural domains (Wareham & Potenza, 2010).

A number of studies have examined rates of lifetime and current SUDs in clinical samples of PGs. These studies have included samples of inpatient (Ciarrocchi & Richardson, 1989; Kausch, 2003; Ramirez, McCormick, Russo, & Taber, 1983) and outpatient (Maccallum & Blaszczyński, 2002; Specker, Carlson, Edmonson, Johnson, & Marcotte, 1996; Toneatto, Skinner, & Dragonetti, 2002) gamblers, and have found elevated rates of lifetime alcohol and drug use disorders in treatment-seeking PGs. For example, Kausch (2003) reported that 66.4% of inpatient PGs had a lifetime history of substance abuse or dependence, with lifetime prevalence of alcohol dependence being 42.5% and drug dependence being 30.1%. In an outpatient sample of PGs, Specker and colleagues (1996) found that 50% had an alcohol use disorder and 10% had a drug use disorder in their lifetime. Compared with those without SUDs, treatment-seeking PGs with a history of substance abuse or dependence have been shown to have greater gambling severity (Ibanez et al., 2001), psychopathology (McCormick, Taber, & Kruedelbach, 1989; Ibanez et al., 2001), suicide attempts (Ciarrocchi, 1987; Kausch, 2003), impulsivity (McCormick et al., 1989), and stress-related physical illnesses (Ciarrocchi, 1987). The literature, however, is conflicting with regards to the effect SUDs

have on PG treatment outcomes (Echeburua, Fernandez-Montalvo, & Baez, 2001; Toneatto et al., 2002).

Rates of *lifetime* alcohol and drug use disorders have also been reported to be elevated in PGs in epidemiological and other community samples (Black & Moyer, 1998; Bland, Newman, Orn, & Stebelsky, 1993; Cunningham-Williams, Cottler, Compton, & Spitznagel, 1998; Lynch, Maciejewski, & Potenza, 2004; Petry, Stinson, & Grant, 2005; Smart & Ferris, 1996). For example, in a large sample of Canadian community residents, individuals who reported disordered gambling had a four-fold increased risk of lifetime SUDs (Bland et al., 1993). Black and Moyer (1998) found rates of lifetime alcohol abuse and dependence of 63% and lifetime drug abuse and dependence of 27% in non-treatment-seeking PGs. Furthermore, Petry, Stinson, and Grant (2005) reported lifetime rates for any alcohol or drug use disorder in PGs were over 73% and over 38%, respectively. SUDs in PGs in the community are associated with increased gambling severity (Hardoon, Gupta, & Derevensky, 2004; Rush, Bassani, Urbanoski, & Castel, 2008; Welte, Barnes, Wieczorek, Tidwell, & Parker, 2001), psychopathology (Smart & Ferris, 1996), and risk of relapse after recent quitting (Hodgins & el-Guebaly, 2010).

Current SUD rates in treatment-seeking and community samples of PGs have typically been found to be lower than lifetime rates (Black & Moyer, 1998; Maccallum & Blaszczyński, 2002; Petry, Stinson, & Grant, 2005; Specker, Carlson, Edmonson, Johnson, & Marcotte, 1996), although are considerably higher than rates in the general population (i.e., 3.8%; Kessler, Chiu, Demler, & Walters, 2005). For example, Maccallum and Blaszczyński (2002) noted that 16% of outpatient PGs had past year alcohol abuse and 8% had past year alcohol dependence. A similar finding was reported in a sample of community PGs (Black & Moyer, 1998).

Rates of PG have also been explored in a variety of alcohol and substance abuse treatment samples (Blume & Lesieur, 1987; Ciarrocchi, 1993; Daghestani, Elenz, & Crayton, 1996; Feigelman, Kleinman, Lesieur, Millman, & Lesser, 1995; Giacomassi, Stitt, & Vandiver, 1998; Hall et al., 2000; Langenbucher, Bavly, Labouvie, Sanjuan, & Martin, 2001; Ledgerwood & Downey, 2002; Lesieur, Blume, & Zoppa, 1986; Lesieur & Heineman, 1988; McCormick, 1993; Petry, 2000; Petry & Tawfik, 2001; Rupcich, Frisch, & Govoni, 1997; Sellman, Adamson, Robertson, Sullivan, & Coverdale, 2002; Shepherd, 1996; Spunt, Lesieur, Hunt, & Cahill, 1995; Spunt, Lesieur, Liberty, & Hunt, 1996; Steinberg, Kosten, & Rounsaville, 1992; Weinstock, Blanco, & Petry, 2006). For example, increased rates of PG have been found in patients in alcohol dependence treatment (Daghestani et al., 1996; Giacomassi et al., 1998; Sellman et al., 2002), with PG being associated with earlier onset and longer duration of alcohol dependence as well as with an increased number of alcohol detoxifications (Lejoyeux et al., 1999). Furthermore, studies of treatment-seeking cocaine dependent individuals have found rates of probable PG to be between 15% to 30% (Blume & Lesieur, 1987; Steinberg, Kosten, & Rounsaville, 1992), and cocaine dependent individuals with PG were more likely to have antisocial traits and behaviours (Hall et al., 2000). In methadone maintenance samples, the rates of current problem and PG have been found to be between 3% to 15% and 7% and 52.7%, respectively (Feigelman et al., 1995; Ledgerwood & Downey, 2002; Spunt et al., 1996; Weinstock et al., 2006; Petry, 2006), with disordered gambling being associated with interpersonal conflicts, criminal activity, recent drug use, a history of problem drinking, poorer physical health, and treatment drop-out (Feigelman et al., 1995; Ledgerwood & Downey, 2002; Weinstock et al., 2006). Furthermore, it appears that PG

rates may be the greatest among polysubstance abusers (Langenbucher et al., 2001; McCormick, 1993; Shephard, 1996).

As noted by Petry, Stinson, and Grant (2005), evidence for the relationship between SUDs, particularly alcohol abuse or dependence, and PG is unequivocal. Furthermore, Crockford and el-Guebaly (1998) stated the overall picture is consistent with a strong association between SUDs and PG, and alcohol is almost always found to be the most common substance of abuse. This significant association, and similarities between PG and SUDs, has influenced the decision to consider the placement of PG into a proposed section of the upcoming revision of the DSM-IV-TR (i.e., the DSM-5) referred to as “Substance Use and Addictive Disorders” (see dsm5.org; Petry, 2006; Potenza, 2006). Despite the high rates of SUDs among PGs as a group, not *all* individuals with gambling disorders have a history of SUD. Accordingly, as suggested by the pathways model of PPG (Blaszczynski & Nower, 2002), SUD rates may be elevated in only certain gamblers (i.e., antisocial impulsivist gamblers). Differential levels of SUD symptoms by gambling subtype were explored in the current study.

PG and unipolar mood disorders. Kim and colleagues (2006) reported most of the clinically-related literature on psychiatric comorbidity in PGs points toward an association between unipolar mood disorders (i.e., internalizing conditions such as major depressive disorder and dysthymic disorder) and PG. As with SUDs, not all PGs have a history of a unipolar mood disorder. Crockford and el-Guebaly (1998) have suggested that at least a subpopulation of PGs have a co-occurring unipolar mood disorder, and these disorders may play a role in perpetuating their gambling. Furthermore, Blaszczynski and Nower’s (2002) pathways model postulates that emotionally vulnerable gamblers

have elevated rates of unipolar mood disorders, and symptoms of these disorders play a role in the development of their disordered gambling.

The majority of studies on depression in PGs have examined samples of individuals in PG treatment. In studies comparing treatment-seeking PGs to non-gambling controls, most have found elevated levels of depression in PGs, as measured by self-report instruments such as the Minnesota Multiphasic Personality Inventory (MMPI; (Graham & Lowenfeld, 1986; Moravec & Munley, 1983) and the Beck Depression Inventory (BDI; Becona, Del Carmen Lorenzo, & Fuentes, 1996; Blaszczynski & McConaghy, 1988, 1989; Blaszczynski, McConaghy, & Frankova, 1990; Getty, Watson, & Frisch, 2000; Maccallum & Blaszczynski, 2003; Maccallum, Blaszczynski, Ladouceur, & Nower, 2007). Elevated rates of lifetime major depressive disorder have been found in treatment-seeking PGs using clinical diagnoses as well (Linden, Pope, & Jonas, 1986; McCormick, Russo, Ramirez, & Taber, 1984; Ramirez, McCormick, Russo, & Taber, 1983; Specker, Carlson, Edmonson, Johnson, & Marcotte, 1996). For example, Linden and colleagues (1986) reported that 72% of Gamblers Anonymous members experienced a DSM-III major depressive episode, with 28% experiencing DSM-III recurrent major depressive episodes. In addition, Specker and colleagues (1996) found a 70% lifetime and 35% current rate of major depressive disorder and a rate of 7.5% for lifetime dysthymic disorder in treatment-seeking PGs. These rates are elevated relative to the lifetime prevalence rates of 16.6% and 2.5% for major depressive disorder and dysthymic disorder, respectively, in the general population (Kessler et al., 2005). Some literature suggests that depression in treatment-seeking gamblers is associated with increased gambling severity (Becona et al., 1996; Moodie & Finnigan, 2006), increased risk for uncontrolled gambling following treatment (Blaszczynski, McConaghy, & Frankova,

1991), and decreased abstinence rates (Hodgins, Peden, & Cassidy, 2005). However, depression does not appear to be associated with PG treatment drop-out (Brown, 1986; Echeburua, Fernandez-Montalvo, & Baez, 2001; Milton, Crino, Hunt, & Prosser, 2002; Robson, Edwards, Smith, & Colman, 2002).

Additional literature has examined the relationship between PG and unipolar mood disorders in non-clinical samples (Black & Moyer, 1998; Bland, Newman, Orn, & Stebelsky, 1993; Cunningham-Williams, Cottler, Compton, Spitznagel, & Ben-Abdallah, 2000; Grant & Kim, 2001; Petry, Stinson, & Grant, 2005). For example, 50% of PGs from the community were found to have major depressive disorder or dysthymic disorder in one study (Black & Moyer, 1998). Furthermore, in a large epidemiological sample Petry and colleagues (2005) reported a lifetime rate of approximately 50% for unipolar mood disorders in PGs, including almost 37% for major depressive disorder and 13% for dysthymic disorder. Some studies, however, have reported that rates of major depressive disorder are not elevated in PGs relative to non-gamblers (Bland et al., 1993; Cunningham-Williams et al., 2000). In a sample of community gamblers who recently quit gambling, individuals with a lifetime history of a unipolar mood disorder were slower to achieve abstinence at a three-month follow-up (Hodgins & el-Guebaly, 2010).

The majority of studies using clinical samples of PGs suggest a relationship between unipolar mood disorders and PG (Crockford & el-Guebaly, 1998; Petry, Stinson, & Grant, 2005). The association between mood disorders and PG in non-treatment-seekers, however, is less clear. For example, Moodie and Finnigan (2006) statistically combined and also separated community and treatment-seeking PGs when examining the relationship between depression and disordered gambling behaviour. When treatment-seekers were removed from analysis, the non-treatment-seeking PGs no longer had

elevated depression scores. Some recent studies using non-clinical samples of PGs, however, are beginning to show an association between *any* mood disorder and PG, with mixed results for specific mood disorders (e.g., Petry et al., 2005). It is clear that not all individuals with disordered gambling have unipolar mood disorders. Accordingly, as postulated by the pathways model (Blaszczynski & Nower, 2002), unipolar mood disorders may be elevated in only certain types of gamblers (i.e., emotionally vulnerable gamblers). The current study examined this possibility in relation to different gambling subtypes.

PG and anxiety disorders. While the pathways model of PPG also highlights the importance of anxiety disorders, another form of internalizing psychopathology, in the development of disordered gambling in emotionally vulnerable gamblers (Blaszczynski & Nower, 2002), relatively few studies have explored the association between PG and anxiety disorders (i.e., panic disorder with or without agoraphobia, social phobia, specific phobia, obsessive-compulsive disorder, generalized anxiety disorder, and posttraumatic stress disorder [PTSD]) (Crockford & el-Guebaly, 1998). In treatment-seeking samples of PGs, rates of panic disorder and agoraphobia have been found to be approximately 20% (Linden, Pope, & Jonas, 1986; Specker, Carlson, Edmonson, Johnson, & Marcotte, 1996). Roy and colleagues (1988) reported that 12.5% of treatment-seeking PGs had either specific phobia or generalized anxiety disorder, while Specker and colleagues (1996) found that 37.5% of treatment-seeking PGs had an anxiety disorder diagnosis. Furthermore, some authors have examined rates of lifetime PTSD among treatment-seeking PGs (Ledgerwood & Petry, 2006; McCormick, Taber, & Kruegelbach, 1989; Specker et al., 1996; Taber, McCormick, & Ramirez, 1987), with 12.5% to 29% of PGs meeting criteria for PTSD. PTSD is generally associated with greater lifetime gambling

severity, impulsivity, and general psychiatric symptoms (Ledgerwood & Petry, 2006; Najavits, Meyer, Johnson, & Korn, in press).

Additional research has examined the presence of anxiety disorders in non-clinical samples of PGs (Black & Moyer, 1998; Bland et al., 1993; Cunningham-Williams et al., 1998; Petry, Stinson, & Grant, 2005). For example, Bland and colleagues (1993) found 26.7% of a community sample of PGs had an anxiety disorder in their lifetime. Other research has revealed that non-clinical samples of PGs are more likely to have specific phobias but not other anxiety disorders (Cunningham-Williams et al., 1998). Finally, in a large epidemiological sample (Petry et al., 2005), over 40% of PGs had a lifetime anxiety disorder, with approximately 5% having panic disorder with agoraphobia, 13% panic disorder without agoraphobia, 10% social phobia, 23% specific phobia, and 11% generalized anxiety disorder. The lifetime prevalence estimate of anxiety disorders in the general population is 28.8% (Kessler et al., 2005).

Studies examining self-reported anxiety levels in PGs have yielded inconsistent results. Trait anxiety has been associated with PG and gambling severity in some studies (Coman, Burrows, & Evans, 1997; Fernandez-Montalvo & Echeburua, 2004; Rodda, Brown, & Phillips, 2004), but not in others (Blaszczynski & McConaghy, 1989; Burton, Netemeyer, & Andrews, 2000). In addition, while some studies failed to discover a relationship between self-reported anxiety and gambling treatment dropout (Echeburua, Baez, Fernandez-Montalvo, 1996; Leblond, Ladouceur, & Blaszczynski, 2003; Milton, Crino, Hunt, & Prosser, 2002; Robson, Edwards, Smith, & Colman, 2002), others have reported that anxiety levels in individuals who dropped out of treatment were greater than those who completed treatment (Echeburua, Fernandez-Montalvo, & Baez, 2001).

While the literature examining anxiety disorders in treatment-seeking or non-clinical samples of PGs is relatively scant (Crockford & el-Guebaly, 1998; Petry, Stinson, & Grant, 2005), anxiety disorders appear to be elevated in PGs relative to the general population. Research on self-reported anxiety in PGs has produced inconsistent findings. Blaszczynski and Nower (2002) noted that increased anxiety has etiological significance for emotionally vulnerable gamblers. Very little research has explored the possibility that elevated anxiety disorder symptoms are associated with one specific type of PG; the current study examines this issue.

PG and attention-deficit/hyperactivity disorder (ADHD). Due to its potential role in the development of disordered gambling, some researchers have studied the association between PG and ADHD. Despite the preliminary nature of this research (Crockford & el-Guebaly, 1998), these studies have consistently shown a relationship between PG and ADHD. For example, Carlton and colleagues (Carlton & Manowitz, 1992; Carlton, Manowitz, McBride, Nora, Swartzburg, & Goldstein, 1987) found that self-reported childhood behaviours related to ADHD were strongly correlated with PG, a relationship that persisted even when substance use was statistically controlled. Furthermore, these authors reported that differential patterns of EEG activity and self-reported symptoms of ADHD in PGs were similar to those found in childhood ADHD. Other studies have similarly found elevated ADHD-related behaviours in the childhoods of PGs (Langenbucher, Bavly, Labouvie, Sanjuan, & Martin, 2001; Specker, Carlson, Christenson, & Marcotte, 1995).

In addition, Rugle and Melamed (1993) compared non-substance abusing PGs to healthy controls and discovered deficits in higher-order attention and increased ADHD-related childhood behaviours in PGs. These authors concluded that childhood behaviours

related to over-activity, destructibility, and inhibitory difficulties were primarily important in differentiating gamblers from controls. Furthermore, ADHD-related symptoms reflecting impulsivity predated the onset of disordered-gambling behaviour.

Rodriguez-Jimenez and colleagues (2006) found that almost 30% of treatment-seeking male PGs reported a history of ADHD. This rate is elevated relative to the lifetime prevalence of ADHD in the general population (i.e., 8.1%; Kessler et al., 2005). Gamblers with ADHD, when compared to healthy controls, self-reported greater levels of impulsivity and performed less efficiently on a behavioural measure of inhibitory control. These authors concluded that PGs with childhood ADHD had an impaired ability to delay gratification and lower control of impulses than PGs without ADHD.

Finally, Breyer and colleagues (2009) examined the association of gambling behaviours among young adults with their longitudinal history of ADHD. Notably, ADHD persists (i.e., those with ADHD at both periods of assessment spanning over a decade) were significantly more likely to meet criteria for possible problem gambling than ADHD desisters (i.e., those with ADHD at the first but not the second assessment period). Furthermore, ADHD moderated the relationship between gambling and legal and work difficulties, and mediated the relationship between gambling and psychological symptoms.

While this research remains preliminary, its consistency and potential etiological significance suggests that ADHD may be a psychiatric disorder that warrants increased attention in the PG literature. Furthermore, the pathways model of disordered gambling (Blaszczynski & Nower, 2002) suggests the developmental significance of ADHD in antisocial impulsivist gamblers. No previous research has examined the differential

relationship between ADHD and gambling subtypes; the current study explores this possible relationship.

PG and antisocial personality disorder. The most extensively researched personality disorder in PGs is antisocial personality disorder, and its etiological relevance for certain gamblers (i.e., antisocial impulsivist gamblers) has been postulated (Blaszczynski & Nower, 2002). Early studies have examined PGs' subscale scores on personality measures of antisocial personality, and these studies have found elevated MMPI Psychopathic Deviate scores (Glen, 1979; Lowenfeld, 1979; Moravec & Munley, 1983; Roston, 1961) and low CPI Socialisation scores (McCormick, Taber, Kruegelbach, & Russo, 1987) in treatment-seeking PGs. Later studies employing more current personality disorder definitions have found self-reported rates of antisocial personality disorder of almost 30% in PGs in treatment (Blaszczynski & Steel, 1998).

Several studies of treatment-seeking PGs have rendered clinical diagnoses of antisocial personality disorder in gamblers. In a sample of treatment-seeking PGs, Blaszczynski and colleagues (Blaszczynski & McConaghy, 1994; Blaszczynski, McConaghy, & Frankova, 1989; Blaszczynski, Steel, & McConaghy, 1997) found that approximately 15% met DSM-III criteria for antisocial personality disorder. Additional studies have found 14.5% to 29% of PGs in treatment met diagnostic criteria for antisocial personality disorder (Ibanez et al., 2001; Pietrzak & Petry, 2006; Steel & Blaszczynski, 1998). These rates are elevated relative to the lifetime prevalence rates of antisocial personality disorder in the general population (i.e., 3.6%; Compton, Conway, Stinson, Colliver, & Grant, 2005). While most studies have found elevated rates of antisocial personality disorder in treatment-seeking PGs, Specker, Carlson, Edmonson,

Johnson, and Marcotte (1996) did not find any individuals in a sample of treatment-seeking PGs with antisocial personality disorder.

Treatment-seeking PGs with and without antisocial personality disorder have been compared on demographic, psychiatric, and gambling-related variables. PGs with antisocial personality disorder were more likely to be male, began gambling earlier in life, experienced more gambling and employment problems, engaged in more gambling-related illegal activity, and reported higher levels of psychological distress than PGs without antisocial personality disorder (Blaszczynski et al., 1997; Blaszczynski & Steel, 1998; Pietrzak & Petry, 2006). Furthermore, PGs with antisocial personality disorder experienced increased depression, anxiety, suicidal ideation, and a history of problem drinking and illicit substance use (Pietrzak & Petry, 2006; Steel & Blaszczynski, 1998).

In non-treatment seeking samples of PGs in the community, 15% to 40% of PGs have been found to meet diagnostic criteria for antisocial personality disorder (Bland, Newman, Orn, & Stebelsky, 1993; Black & Moyer, 1998; Cunningham-Williams et al., 1998; Petry, Stinson, & Grant, 2005; Slutske et al., 2001). Notably, Slutske and colleagues (2001) found a lifetime prevalence of conduct disorder (i.e., the childhood precursor of antisocial personality disorder) of 23%, adult antisocial behaviour of 37%, and antisocial personality disorder of 15% among men with a lifetime history PG.

Much of the literature on PG and antisocial personality disorder has emphasized the causal link between the disorders (Bergh & Kuhlhorn, 1994; Blaszczynski & McConaghy, 1994; Blaszczynski, McConaghy, & Frankova, 1989; Blaszczynski & Silove, 1996; Brown, 1987; Ladouceur, Boisvert, Pepin, Loranger, & Sylvain, 1994; Lesieur & Rosenthal, 1991; Meyer & Fabian, 1992; Rosenthal & Lesieur, 1996; Rosenthal & Lorenz, 1992; Slutske et al., 2001). Notably, Blaszczynski and colleagues

stated that individuals committing gambling-only related offenses showed a significant increase in antisocial features after 15 years of age, leading these authors to conclude that antisocial features in the majority of cases emerge as a consequence of PG. They also noted that for gamblers exhibiting high levels of antisocial features in pre-adolescence, gambling behaviour may increase the risk for committing gambling-related offenses.

Antisocial personality disorder may increase the propensity to engage in criminal and gambling behaviours independently of each other, or increase the risk of offending in response to gambling-induced financial problems. Alternatively, PG may produce personality changes phenotypically similar to antisocial traits as a consequence of attempts to conceal gambling-induced problems. Slutske and colleagues (2001) reported that in their study of community PGs the higher rate of adult antisocial behaviour than conduct disorder among individuals with PG is consistent with the hypothesis that part of the association between PG and antisocial behaviour may be due to the influence of disordered gambling behaviour. However, the high rate of conduct disorder in their study also suggested that much of the association could not be explained by this causal influence. In this regard, Welte and colleagues (2009) found a strong positive relationship between current problem gambling and current conduct disorder, and the relationship was strongest in individuals whose problem gambling began in their early to mid-teens than for those whose disordered gambling began later. These authors noted a cluster of problem behaviours emerge early in life, and problem gambling can be part of that cluster. This is consistent with the antisocial impulsivist gambler in the pathways model (Blaszczynski & Nower, 2002).

Evidence appears to be fairly convincing that treatment-seeking PGs have high rates of antisocial personality disorder. Furthermore, it appears that only some PGs

exhibit antisocial personality disorder (Blaszczynski & Nower, 2002), and for these gamblers it has been suggested that this form of externalizing psychopathology may have significance in the development of disordered gambling behaviour. The current study examined the relationship between antisocial personality and conduct disorder traits in subtypes of individuals with disordered gambling.

PG and Personality Traits

The following review highlights maladaptive personality traits that are central to the internalizing-externalizing model of psychopathology, and that are either explicitly presented in Blaszczynski and Nower's (2002) pathways model of PPG or that have relevance to the model. That is, impulsivity is the core personality process that underlies externalizing forms of psychopathology and is reportedly characteristic of antisocial impulsivist gamblers. In addition, negative emotionality has been empirically linked to internalizing disorders such as unipolar mood and anxiety disorders, and these conditions are suggested to characterize emotionally vulnerable gamblers.

PG and impulsivity/sensation seeking. Individual differences in impulsivity and sensation seeking have long been assumed to be central to the development and maintenance of PG. While impulsivity is typically defined as the failure to resist an impulse, Nower and Blaszczynski (2006) noted the relevant issue is, "Does the failure to resist an impulse result from an inability to act without sufficient forethought to take into account consequences, an unwillingness to deter gratification, or a lack of restraint despite the capacity to do so?" Definitions of impulsivity in the literature have variably emphasized concepts of acting with lack of forethought (Dickman, 1990), rapid decision making without consideration (Jaspers, 1963), non-planning, risk-taking, and sensation

seeking (Eysenck & Eysenck, 1977, 1978), motor activation (Barratt, 1983, 1985), lack of deliberation (Dickman, 1990), and delay discounting (Green, Fristoe, & Myserson, 1994). Accordingly, impulsivity is best conceptualized as a multi-dimensional construct that takes into account several unique underlying processes that lead to impulsive behaviours that appear phenomenologically similar.

With few exceptions (e.g., Allcock & Grace, 1988), studies employing self-report measures of impulsivity have found that treatment-seeking and community PGs have elevated scores of impulsivity relative to non-PG controls (Blaszczynski, Steel, & McConaghy, 1997; Carlton & Manowitz, 1994; Castellani & Rugle, 1995; Clarke, 2006; Loxton, Nguyen, Casey, & Dawe, 2008; Maccallum, Blaszczynski, Ladouceur, & Nower, 2007; Steel & Blaszczynski, 1996). The majority of these studies have explored impulsivity using cross-sectional research designs. The few longitudinal studies demonstrate that impulse control difficulties precede disordered gambling behaviour (Vitaro, Arseneault, & Tremblay, 1997, 1999; Slutske, Caspi, Moffitt, & Poulton, 2005). For example, in a longitudinal study of male adolescents, Vitaro and colleagues (1997) found that self-reported and teacher-rated impulsivity at 13 years of age predicted PG at 17 years of age, even after statistically controlling for early gambling behaviour. In addition, in a community sample Slutske, Caspi, Moffitt, and Poulton (2005) found that PG at 21 years of age was associated with lower levels of constraint at 18 years of age, even after controlling for SUDs.

Elevated self-reported impulsivity in PGs in treatment has been shown to be associated with increased gambling severity (Loxton, Nguyen, Casey, & Dawe, 2008; Vitaro, Arseneault, & Tremblay, 1997), increased psychological distress and depression (Blaszczynski, Steel, & McConaghy, 1997; Steel & Blaszczynski, 1996), elevated

number of suicide attempts (Blaszczynski et al., 1997), and non-response to treatment (Gonzalez-Ibanez, Mora, Gutierrez-Maldonado, Ariza, & Lourido-Ferreira, 2005). In regards to the relationship between impulsivity and PG treatment dropout, however, results have been inconsistent. Impulsivity has not been associated with treatment dropout in some studies (Echeburua et al., 2001), while it was a significant predictor of treatment failure in others (Leblond, Ladouceur, & Blaszczynski, 2003; Maccallum, Blaszczynski, Ladouceur, & Nower, 2007).

Most studies of impulsivity in PGs have employed self-report measures of impulsivity. Emerging research, however, is examining impulsivity in PGs as conceptualized by behavioural and neuropsychological measures. This research supports self-report findings that PGs are characteristically impulsive (e.g., Ledgerwood, Alessi, Phoenix, & Petry, 2009). For example, the rate at which rewards delayed in time are subjectively devalued is considered a behavioural marker of impulsivity. Petry and colleagues (Alessi & Petry, 2003; Ledgerwood et al., 2009; Petry, 2001; Petry & Casarella, 1999) have studied the relationship between delayed discounting and PG. Petry and Casarella (1999) compared substance abusers with and without PG and found that substance abusers with PG discounted delayed rewards at three times the rate of their substance abusing-only counterparts. In another study using a sample of PGs, Petry (2001) found that PGs discounted delayed rewards at higher rates than control participants, and gamblers with SUDs discounted delayed rewards at higher rates than non-substance abusing gamblers. Finally, Alessi and Petry (2003) found that impulsive choices on a delay discounting task were predicted by gambling severity. Furthermore, severity of gambling problems predicted the degree of impulsivity on the delayed

discounting task above and beyond the variance accounted for by self-reported impulsivity.

Fuentes, Tavares, Artes, and Gorenstein (2006) explored the effect of psychiatric comorbidity on neuropsychological and self-reported impulsivity in treatment-seeking PGs. While PGs with and without psychiatric comorbidities produced more errors on neuropsychological measures of impulsivity than controls, PGs with comorbidities reported being more impulsive than non-comorbid PGs and controls. The authors noted that impulsivity assessment in PGs is best performed by a combination of methodologically distinct tests, comprising neuropsychological and self-report measures. This suggestion is in line with research by Goudriaan and colleagues (2008), who showed that relapse in PGs was predicted by neuropsychological measures of executive functioning but not self-report measures of impulsivity.

The majority of studies examining the multi-dimensional construct of impulsivity in PGs have examined the dimension of impulsivity referred to as sensation seeking. The construct sensation seeking can be traced back to the work of Zuckerman (1971) and Zuckerman, Kolin, Price, and Zoob (1964). Sensation seeking has been defined as a trait involving the “seeking of varied, novel, complex and intense sensations and experiences” (Zuckerman, 1994, p. 27). Sensation seeking has been an integral component of some theories of the etiology and maintenance of PG (e.g., Zuckerman, 1999), with PGs being considered the prototypical sensation seeker.

Studies examining sensation seeking in PGs in treatment, however, have found lower or equivalent sensation seeking scores in PGs when compared to healthy controls (Blanco, Orensanz Munoz, Blaco Jerez, & Saiz Ruiz, 1996; Blaszczyński, McConaghy, & Frankova, 1990; Blaszczyński, Wilson, & McConaghy, 1986; Carrasco, Saiz-Ruiz,

Hollander, & Cesar, 1994; Raviv, 1993). In addition, non-clinical samples PGs have been generally found to have lower or equivalent sensation seeking scores when compared to controls (Anderson & Brown, 1984; Bonnaire, Lejoyeux, & Dardennes, 2004; Coventry & Brown, 1993; Dickerson, Cunningham, England, & Hinchy, 1991; Dickerson, Hinchy, & Fabre, 1987; Dickerson, Walker, England, & Hinchy, 1990; Lejoyeux, Feuche, Loi, Solomon, Ades, 1998; Powell, Haroon, Derevensky, & Gupta, 1999). Furthermore, when compared to substance dependent individuals, PGs are often found to be indistinguishable on measures of sensation seeking (Castelli & Rugle, 1995; Lejoyeux, Feuche, Loi, Solomon, & Ades, 1998; Steinberg, Kosten, & Rounsaville, 1992), suggesting sensation seeking is not characteristic of PGs and is distinct from other forms of impulsivity.

In a review of sensation seeking in PGs, Hammelstein (2004) reported the phenomenologically-derived suggestion that the PG is the prototype of the sensation seeker (Zuckerman, 1999) cannot be reconciled with empirical results. However, Hammelstein also suggested it is possible that PGs may not be high sensation seekers in a variety of contexts, as conceptualized in various measures of sensation seeking, but rather use *only* gambling to satisfy the need for intense and novel stimulation. Hammelstein noted it is more reasonable to conceive sensation seeking as a *need* for stimulation, rather than, as conceptualized by Zuckerman (1999), highly specific behaviours. By conceptualizing sensation seeking as a need it can be easily differentiated from impulsivity, which is related to the control of behaviour and has been highly correlated with PG.

PG and negative emotionality. Several theories on the etiology of PG have implicated the personality dimension of negative emotionality as an important risk factor

for the subsequent development of disordered gambling (Dickerson & Baron, 2000; Hand, 1998). Furthermore, given that negative emotionality is associated with and is considered a vulnerability factor for psychopathology in general (e.g., Malouff, Thorsteinsson, & Shutte, 2005), it is possible it is implicated in the development of PG. The available empirical literature, while relatively limited, appears to support the notion that negative emotionality, also referred to as neuroticism, is associated with PG.

A number of studies of PGs in treatment have reported elevated levels of neuroticism compared to controls (Blanco, Ibanez, Blanco-Jerez, Baca-Garcia, & Saiz-Ruiz, 2001; Blaszczynski, Buhrich, & McConaghy, 1985; Blaszczynski, Steel, & McConaghy, 1997; Blaszczynski, Wilson, & McConaghy, 1986; Graham & Lowenfeld, 1986; Roy et al., 1989). Similar findings have been observed in community PGs relative to non-PGs (Bagby et al., 2007; Potenza et al., 2003). Furthermore, neuroticism has been associated with severity of PG (McCormick, 1993), treatment failure (Echeburua, Fernandez-Montalvo, & Baez, 2001), uncontrolled gambling following treatment (Blaszczynski, McConaghy, & Frankova, 1991), and earlier relapse following treatment (Daughters, Lejuez, Strong, Brown, Breen, & Lesieur, 2005).

In conclusion, empirical literature suggests that impulsivity and negative emotionality are elevated in PGs relative to controls. Furthermore, both impulsivity and negative emotionality appear to have significance to the development and maintenance of disordered gambling behaviour. The pathways model of PPG (Blaszczynski & Nower, 2002) suggests that impulsivity plays an important etiological role for some gamblers (i.e., antisocial impulsivist gamblers), and implies the importance of negative emotionality in the genesis of disordered gambling for other gamblers (i.e., emotionally vulnerable gamblers). Despite literature showing elevated impulsivity and negative

emotionality in PGs as a group, little empirical work has specifically examined how these personality traits are related to subtypes of PPGs. In addition, little research exists on the relationship between internalizing and externalizing forms of psychopathology and these personality traits.

Covariation of Psychopathology in PGs: Utility of the Internalizing-Externalizing Model

PG is a disorder that is highly comorbid with other psychiatric conditions, notably SUDs, unipolar mood disorders, and antisocial personality disorder. In this regard, PG is similar to other psychiatric conditions. That is, high rates of comorbidity have been observed among purportedly discrete and mutually exclusive psychiatric disorders in numerous clinical and epidemiological samples (Clark, Watson, & Reynolds, 1995; Kessler et al., 1994; Maser & Cloninger, 1990). Furthermore, literature suggests that specific psychiatric disorders are highly associated with one another. For example, a large number of studies document the covariation between unipolar mood and anxiety disorders (Maser & Cloninger, 1990; Merikangas et al., 1996; Mineka, Watson, & Clark, 1998). In addition, SUDs, conduct disorder, and antisocial personality disorder co-occur at well beyond chance levels (Armstrong & Costello, 2002; Waldman & Slutske, 2000). While it has yet to be studied, patterns of comorbidity in PGs appear to parallel patterns found in the general psychopathology literature. For example, it has been suggested that some gamblers experience comorbid depression and anxiety, while others experience comorbid SUDs and antisocial personality disorder (see Blaszczynski & Nower, 2002, for a review).

Krueger and colleagues (2001) suggested that comorbidity among psychiatric conditions reflects the fact that common psychiatric disorders, rather than being discrete

and unique conditions, are reliable indicators of core psychopathological processes. Consistent with this proposition, Mineka, Watson, and Clark (1998) proposed a model to account for patterns of comorbidity among unipolar mood and anxiety disorders that posits a higher order dimension of personality, namely negative emotionality, which influences all disorders within this realm. Furthermore, a wide body of research suggests that high negative emotionality is a non-specific predictor of a broad class of psychopathology encompassing the unipolar mood and anxiety disorders. First, epidemiological and twin-based studies have shown that covariations between depressive and anxiety symptoms and disorders is due largely to a common genetic factor that also influences negative emotionality (Fanous et al., 2002; Jang & Livesley, 1999; Kendler et al., 1993; Markon, Krueger, Bouchard, & Gottesman, 2002; Mineka, Watson, & Clark, 1998; Roberts & Kendler, 1999). Second, individuals with and without diagnoses of major depression and/or generalized anxiety can be separated on the dimension of negative emotionality (Trull & Sher, 1994; Watson, Clark, & Carey, 1988; Widiger & Trull, 1992). Third, longitudinal studies have shown that negative emotionality is a predictor of the onset of major depressive disorder (Hirschfeld et al., 1989) and panic attacks (Hayward, Killen, Kraemer, & Taylor, 2000). Finally, research on the latent structure of unipolar and anxiety disorders suggests that negative emotionality is a higher-order facet that accounts for covariation among these disorders (Spence, 1997; Zinbarg & Barlow, 1996). Research from a variety of sources indicates negative emotionality is associated with unipolar mood and anxiety disorders as well as their comorbidity. Negative emotionality, therefore, is postulated to be the core psychopathological process that underlies these disorders and is responsible for their strong pattern of comorbidity.

Extensive research additionally documents correlations between SUDs, conduct disorder, antisocial personality disorder, and personality traits such as novelty seeking, impulsivity, disinhibition, and constraint (Howard, Kivlahan, & Walker, 1997; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; McGue, Slutske, & Iacono, 1999; McGue, Slutske, Taylor, & Iacono, 1997; Patrick & Zempolich, 1998; Sher & Trull, 1994; Verona & Parker, 2000; Watson & Clark, 1993). In clinical populations, numerous studies have demonstrated that substance abusers score higher than controls on personality inventories of impulsivity (Allen et al., 1998; Chalmers et al., 1993; Cookson, 1994; Eisen et al., 1992; McCormick et al., 1987; Patton et al., 1995; Rosenthal et al., 1990; Sher & Trull, 1994). Longitudinal studies have also shown that children with elevated novelty seeking are at a greater risk to develop subsequent substance abuse (Cloninger, Sigvardsson, & Bonham, 1988; Masse & Tremblay, 1997) and delinquency (Tremblay, Pihl, Vitaro, & Dobkin, 1994). Furthermore, impulsivity observed as early as age three foretells alcohol dependence and criminal behaviour in early adulthood (Caspi, Moffitt, Newman, & Silva, 1996). Finally, lack of constraint in late adolescence predicts substance dependence and antisocial behaviour in early adulthood (Krueger, 1999). Research from a variety of sources suggests dimensions of impulsivity are associated with SUDs and antisocial behaviours as well as their comorbidity. Impulsivity, therefore, is postulated to be the core psychopathological process that underlies these disorders and is believed to be responsible for their strong pattern of comorbidity.

In an effort to explain the extensive comorbidity that exists among psychiatric conditions, a number of empirical studies have examined the higher order structure of the common psychiatric disorders. These studies have found consistent and meaningful groupings of mental disorders (Krueger, Caspi, Moffitt, & Silva, 1998; Krueger et al.,

2001, 2003; Krueger, 1999; Vollebergh et al., 2001; Cox, Clara, & Enns, 2002; Kendler et al., 2003; Kessler et al., 2005). For example, Krueger and colleagues (1998) examined the latent structure underlying ten psychiatric disorders and found that a two-factor structure offered the best account of the correlations observed among the disorders. They discovered what they referred to as an *internalizing* dimension which was comprised of symptoms of the unipolar (i.e., major depressive disorder, dysthymic disorder) and anxiety (i.e., generalized anxiety disorder, agoraphobia, social phobia, simple phobia, obsessive-compulsive disorder, and posttraumatic stress disorder) disorders. In addition, they discovered an *externalizing* dimension comprised of symptoms of alcohol and drug abuse and dependence, conduct disorder, and antisocial personality disorder. Further research has confirmed the two-factor higher-order structure underlying major psychiatric disorders (Kramer, Krueger, & Hicks, 2008; Krueger, 1999; Krueger & Finger, 2001; Slutske & Watson, 2006), which offers a model that organizes psychopathology around the inward or outward expression of distress.

Krueger and colleagues (Krueger, Caspi, Moffitt, & Silva, 1998; Krueger & Silva, 2001) hypothesized that the internalizing and externalizing dimensions of psychopathology mapped onto the higher order trait dimensions of adult personality. Specifically, they speculated that internalizing disorders were associated with high negative emotionality, whereas externalizing disorders were associated with low levels of constraint (i.e., high impulsivity). In a study examining psychopathology dimensions and personality dimensions in a joint factor analysis, Krueger, McGue, and Iacono (2002) provided evidence for the association between high negative emotionality and the internalizing dimension and the association between low constraint and the externalizing dimension. These associations were found in other research studies as well (Krueger,

Hicks, Patrick, Carlson, Iacono, & McGue, 2001). High negative emotionality may reflect the personality substrate for internalizing disorders (Achenbach & Edelbrock, 1978, 1984; Clark, Watson, & Mineka, 1994; Krueger et al., 2001) whereas low constraint may reflect the personality substrate for the externalizing disorders (Kendler, Davis, & Kessler, 1997; Krueger et al., 2002; Sher & Trull, 1994; Widiger & Clark, 2000). Krueger and colleagues hypothesized that comorbidity occurs, then, because basic dimensions of personality variation confer risk for a broad range of psychopathological outcomes.

Research on the structure and organization of psychiatric disorders suggests that patterns of behavioural disturbance and psychiatric comorbidity tend to cohere along internalizing and externalizing dimensions. The *internalizing-externalizing model* offers utility in organizing psychopathology into a coherent structure, reducing the complexity of comorbidity and postulating underlying personality factors that may account for the covariation. Emerging research (Miller, Fogler, Wolf, Kaloupek, & Keane, 2008; Miller, Greif, & Smith, 2003; Miller, Kaloupek, Dillon, & Keane, 2004; Miller & Resick, 2007) has suggested the internalizing-externalizing model can help provide coherence to psychopathology in individuals with an index disorder that is associated with high levels of comorbid psychiatric conditions. Using a broad measure of personality to identify personality-based subtypes within a heterogeneous sample of veterans with PTSD, Miller and colleagues (2003) discovered three subtypes of PTSD that were differentiated based on personality and psychopathology. The *internalizing* cluster was characterized by low scores on positive emotionality and high scores on negative emotionality, and exhibited elevated levels of unipolar mood and anxiety disorders. The *externalizing* cluster was characterized by high scores on negative emotionality coupled with low scores on constraint, and exhibited elevated levels of SUDs and antisocial personality disorder.

Finally, a *low pathology* cluster was characterized by normative levels of the three higher-order personality traits, and exhibited low levels of all psychiatric disorders. The same subtypes were found utilizing different measures of personality and with different PTSD samples (Miller, Fogler, Wolf, Kaloupek, & Keane, 2008; Miller, Kaloupek, Dillon, & Keane, 2004; Miller & Resick, 2007). Miller and colleagues suggested the internalizing-externalizing model helped in developing a typology of PTSD designed to account for the heterogeneity of posttraumatic symptomatology and comorbid psychopathology.

Internalizing-Externalizing Model in PGs?

Miller and Resick (2007), in their work applying the internalizing-externalizing model to conceptualize heterogeneity in individuals with PTSD, noted that the internalizing-externalizing model can be applied to other disorders that show extensive patterns of comorbidity and heterogeneity. PG is a disorder associated with considerable heterogeneity of psychopathology and personality traits. Accordingly, the internalizing-externalizing model may provide a useful heuristic in conceptualizing the various psychiatric comorbidities and personality variables associated with PG.

No research to date has explored the possibility that psychiatric symptoms and behavioural disturbances in PGs cohere along latent internalizing and externalizing dimensions. Slutske and colleagues (2000, 2001, 2005), however, suggested the overlap between PG, SUDs, and antisocial personality disorder may be explained in part by the existence of a latent externalizing factor associated with impulsivity. They noted, along with Petry (2001), that this possibility should be explored. Furthermore, Potenza, Xian, Shah, Scherrer, and Eisen (2005) observed that prior studies supporting the clustering of common psychiatric disorders into internalizing and externalizing types have generally

not included measures of PG. They noted that PG shares features of impulsivity with other externalizing disorders; however, the genetic overlap between PG and major depressive disorder is substantial enough to raise questions regarding the nature of the relationship between PG to internalizing disorders. Therefore, as Potenza and colleagues (2005) suggested, direct investigation of the most appropriate categorization of PG as an internalizing or externalizing disorder is needed.

Subtyping PGs based on Psychopathology and Personality

While it is clear that PGs are heterogeneous in terms of forms of comorbid psychopathology and maladaptive personality traits that characterize them, a lack of clarity remains on how best to conceptualize the heterogeneity with which PGs present. That is, it is unknown how co-occurring psychiatric disorders and maladaptive personality traits in PGs are associated with one another, with the onset and maintenance of PG, and with the severity of disordered gambling behaviour. While the internalizing-externalizing is one method of conceptualizing the heterogeneity in PPGs, it remains to be studied. However, Blaszczynski and colleagues (Blaszczynski, McConaghy, & Frankova, 1990; Blaszczynski & Nower, 2002; Steel & Blaszczynski, 1996) and others (e.g., Graham & Lowenfeld, 1986; Gonzalez-Ibanez, 1994; Lesieur, 1993; Lesieur & Blume, 1991; Lesieur, 2001; Lesieur & Mark, 1993; McCormick, 1987; Moran, 1970; Walker & Kruedelbach, 2000; Zimmerman, Meeland, & Krug, 1985) have suggested the importance of grouping PGs into subtypes based on etiological factors, psychopathology, personality and motivational factors, and demographics in order to adequately account for the heterogeneity seen in individuals with this disorder. This substantial literature has presented possible PG subtypes, and results are consistent in some aspects with the

internalizing-externalizing model of psychopathology. That is, some gambling subtypes present primarily with internalizing disorders and others present primarily with externalizing disorders.

Early subtyping of PGs (1970 – 2001). The earliest empirical attempt to separate PGs into distinct subtypes is the classification system presented by Moran (1970). Moran noted that PG, because it is a disorder classified based on problematic *behaviour*, is likely a heterogeneous group of conditions that share the feature of excessive gambling but differ in underlying etiological and motivational factors. Based on information obtained through structured clinical interviews (including questions about the details of gambling problems, gambling in early life, and psychiatric difficulties) with 50 male PGs referred for psychiatric treatment, Moran developed a qualitative taxonomy that categorized PGs into five subtypes based on the relative importance of individual characteristics and social influences.

According to Moran's classification system, the *subcultural* gambler is an individual who initiates gambling and maintains disordered gambling behavior as a function of pressures from family and/or peers. While social pressures are paramount for this type of gambler, individual characteristics also partially determine that gambling reaches a pathological level. The *neurotic* gambler, on the other hand, develops a gambling disorder not because of interpersonal pressures but rather in response to stressful life situations and/or emotional difficulties. According to Moran, the activity of gambling for the neurotic gambler provides relief from underlying feelings of tension. The *impulsive* gambler, which Moran states is the most serious subtype of PG, experiences a loss of control over his or her gambling, has strong urges to gamble, and suffers serious social and economic dysfunction as a result of gambling. The

psychopathic gambler's gambling is a function of his global psychopathic personality disturbance. Finally, the *symptomatic* gambler's gambling is best understood as a symptom of another psychiatric condition and not as a primary disorder in its own right. That is, this group's gambling is only one among many other symptoms characteristic of a particular disorder (most commonly depression). As with neurotic gamblers, gambling for symptomatic gamblers provides relief from the symptoms of tension and depression. While Moran's classification draws attention to the intricate relationship between individual factors and social pressures in the etiology and maintenance of PG, he did not specify how he derived this typology and provided no data analyses to support his model.

Zimmerman and colleagues (1985) noted that previous studies of PG, including the work of Moran (1970), failed to 'objectively' investigate the disorder and the behavioural manifestations that define it. These authors factor analyzed Inventory of Gambling Behavior responses from 83 PGs in Gamblers Anonymous and 61 non-gambling control participants to explore the factor structure underlying PG-related behaviors. Five factors were extracted that significantly differentiated PGs from non-gambling controls. The first factor contained items representing underlying anxiety and maladjustment and was considered an index of general psychological distress. Based on Moran's delineation of the neurotic gambler, Zimmerman and colleagues labeled their first factor *Neurotic Gambling*. High scorers on this factor experienced gambling as a release from frustration and worry. The second factor captured a variety of antisocial behaviours and was labeled *Psychopathic Gambling*. High scorers on this factor reported a history of school truancy, vandalism, and theft beginning in early adolescence, and also being prone to easily becoming bored. The third factor was labeled *Impulsive Gambling*, and was comprised of items indicating high energy levels and risk-taking behaviors. High

scorers on this factor described themselves as risk takers who are energetic. Finally, the fourth and fifth factors related to *White Collar Crime* and *Employment Problems* because they reflected criminal activities (e.g., fraud, tax evasion) and work difficulties related to gambling, respectively. Zimmerman and colleagues concluded that PG is a complex expression of neurotic, psychopathic, and impulsive factors which are correlated but relatively independent of one another.

While empirical research on the characteristics of PGs was beginning to emerge at the time (e.g., Zimmerman et al., 1985) Graham and Lowenfeld (1986) sought to address the relative lack of studies examining the personality traits of gamblers. Furthermore, given that previous research reported PGs variably show strong antisocial tendencies as well as signs of dysphoria or depression (Moravec & Munley, 1983), Graham and Lowenfeld examined whether personality characteristics could be used to distinguish meaningful subgroups of PGs. Using medical chart data from a sample of 100 males receiving inpatient PG treatment at a Veterans Administration Hospital, Graham and Lowenfeld cluster analyzed MMPI profiles and generated four distinct clusters of gamblers.

The first cluster, which represented a *personality disordered* profile, included individuals described as immature, rebellious, restless, grandiose, and hostile, and who were also seen as having emotional problems. The second cluster, which was characterized by heightened *paranoia*, represented a type of gambler described as suspicious, jealous, rigid, and withdrawn. In addition, this subtype was considered irritable and hostile and prone to excessive alcohol use. The third cluster demonstrated a MMPI profile with a combination of *depressive or anxious symptoms and alcoholism*. Finally, the *passive-aggressive or emotionally unstable* personality cluster of gamblers

tended to be impulsive, immature, and irresponsible. In addition, this PG had low frustration tolerance and was often moody, tense, and depressed. Their history of impaired academic and vocational adjustment suggested this group was the most antisocial of Graham and Lowenfeld's PG clusters. Although Graham and Lowenfeld's taxonomy provides a basis for understanding psychopathology among gamblers, they did not validate these clusters by comparing them by using additional independent variables.

In reviewing previous research on PG, including the work of Graham and Lowenfeld (1986), McCormick (1987) concluded that PGs vary tremendously in their presentations and motivations for gambling. McCormick suggested the literature at the time supported generalization at the level of subtypes, and there may be both explanatory value and clinical utility to conceptualizing subtypes of gamblers. In an attempt to integrate the literature on the differential motivations of PGs into a parsimonious model, McCormick used "psychological observations" to derive two clinically meaningful subtypes of PGs. Accordingly, he presented a PG classification system based on the "need state" that drives and is satisfied by gambling behavior. The two subtypes he postulated were both characterized by chronic states of hypoarousal but were differentiated according to the presence of depression or boredom proneness. Gamblers in the first subtype, which he referred to as the *recurringly depressed gambler*, experience depression that predates disordered gambling and tend to have histories of childhood traumatic experiences. Pervasive depressogenic cognitive styles, interacting with biochemical abnormalities, are considered instrumental in establishing a need state in this type of gambler that is relieved by the affect-enhancing excitement produced by gambling. For the recurringly depressed gambler, gambling serves the function of providing a euphoria that allows him or her to escape dysphoric feelings. Gamblers in the

second subtype, which McCormick called the *chronically understimulated gambler*, do not experience dysphoria but rather excessive boredom, low frustration tolerance, and a need for constant and varied stimulation. These gamblers also exhibit deficiencies in impulse control and may have narcissistic personality traits. The inherent arousal produced by gambling acts as a reinforcer for this type of gambler, reducing his or her boredom and consequently perpetuating continued gambling.

McCormick's subtyping scheme emphasized the importance of both psychological and physiological factors in the development of PG. He noted the model is general enough to be consistent with the data available at the time, yet he hoped it would be empirically investigated. Existing empirical research appears to support both the recurrently depressed gambler (Linden, Pope, & Jonas, 1986; Petry & Steinberg, 2005; Ramirez, McCormick, & Lowie, 1988) and the chronically understimulated gambler (Goldstein, Manowitz, Nora, Swartzburg, & Carlton, 1985; Rugle & Melamed, 1991). Furthermore, McCormick's PG model is consistent with Jacobs' (1986) general theory of addiction, which proposes that abnormal physiological resting states (i.e., chronically overstimulated or understimulated) in combination with negative childhood experiences results in feelings of inadequacy, rejection, and/or guilt that predispose gamblers to use gambling behaviour to escape psychological distress.

Comparing 48 patients attending a specialized hospital PG therapy program to 40 patients attending a family physician for non-gambling related problems, Blaszczynski and colleagues (1990) found that PGs showed elevated boredom proneness and depression scores which suggests PG is in part a maladaptive coping strategy to deal with affective disturbances. They noted that high scores on depression were consistent with McCormick's recurrently depressed gambler, while high scores on boredom proneness

was similar to the chronically understimulated gambler. However, given that depression and boredom proneness were correlated in their sample, Blaszczynski and colleagues acknowledged the existence of a third subtype of gamblers who are both prone to depression and boredom.

Additional support for McCormick's two PG subtypes comes from Lesieur and Blume (1991), who interviewed 50 females attending Gamblers Anonymous and classified these PGs into two subtypes called *escape seekers* and *action seekers*. Escape seekers reported using gambling to numb feelings of dysphoria, and their gambling could be seen as a response to increased depression and anxiety, and to traumatic experiences. Action seekers, on the other hand, reported gambling to stimulate feelings of excitement and to fulfill a desire to impress others. The subtypes identified by Lesieur and Blume are virtually identical to the recurrently depressed and chronically understimulated PGs proposed by McCormick.

The work of Moran (1970) and Zimmerman and colleagues (1985) identified an impulsive type of gambler, suggesting that impulsivity underpins gambling behavior in at least some gamblers. To further the empirical investigation of impulsivity and associated variables (i.e., psychological distress, antisocial personality disorder) in PGs, Steel and Blaszczynski (1996) analyzed various measures of these constructs completed by 115 treatment-seeking PGs using principal components analysis. The first of the four factors comprised the full factor loadings of the Beck Depression Inventory (BDI; Beck, Ward, Mendelson, Mock, & Erbaugh, 1961), the Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975), Neuroticism, and the Symptom Checklist-90 (SCL-R-90; Derogatis, Lipman, & Covi, 1973) global severity index, and boredom proneness, and was labeled *Psychological Distress* or neuroticism. This factor was positively associated

with female gender, history of suicidal ideation and suicide attempts, as well as with a family history of psychiatric disorders. The second factor loaded *Sensation Seeking* items, and was positively associated with a history of problematic alcohol use. The third factor, which they labeled *Crime and Liveliness*, had high loadings of items pertaining to criminal activity and behaving/making decisions spontaneously. Finally, the fourth factor included items that represented EPQ psychoticism, impulsivity, and antisocial behavior traits and was labeled the *Impulsive Antisocial* factor. This factor was associated with the earliest onset of gambling and gambling-related difficulties. Overall, Steel and Blaszczynski noted the factorial structure they identified reproduced the structure found by Zimmerman and colleagues (1985).

Lesieur (2001) examined the appropriateness of two or three-cluster solutions to conceptualize the heterogeneity of PGs' self-reported psychopathology and personality traits in a sample of inpatient gamblers. In testing a two-cluster solution, Lesieur found a cluster of PGs with lower gambling severity who were relatively less impulsive, had lower levels of depression and trait anxiety, as well as lower levels of attentional difficulties. This cluster of gamblers was less likely to report using gambling to escape dysphoric mood or to report engaging in illegal activities. The second cluster demonstrated greater levels of gambling-related problems and other forms of psychopathology. Lesieur concluded that this two-cluster solution supported the existence of a *normal* PG and a *severe* PG.

In testing a three-cluster solution, the first cluster was comprised of PGs with low levels of psychopathology including impulsiveness, attention deficit, depression, anxiety, dissociation, and illegal activity, similar to the "normal" gambler identified in the two-cluster solution. The gamblers in the second cluster had moderate levels of impulsiveness,

attention deficit, depression, anxiety, trauma, and dissociation and were deemed moderately-impulsive action seekers. This second cluster also had elevated gambling severity relative to the first cluster, as well as younger age of onset of gambling, higher levels of excitement seeking, and greater narcissistic personality traits than the other two clusters. Finally, the third or *impulsive escape seeker* cluster fell in the severely psychopathological range of impulsiveness, attention deficit, depression, anxiety, trauma, dissociation, and gambling to escape. Lesieur was able to establish the concurrent validity of his clusters by examining hypothesized differences on several gambling severity, gambling type, impulsivity, trauma, psychopathology, substance use, psychosocial, and personality variables. Based on his test of both the two-cluster and the three-cluster solutions, Lesieur concluded that gamblers did not cluster based on specific theorized differences in types of psychopathology and motivation to gamble. Rather, they were classified primarily on the severity of psychopathology.

The emergence of three pathways subtypes (2002 – 2010). Blaszczynski and Nower (2002) noted that, despite the work of previous investigators, an empirically validated theoretical model of PG that integrated relevant biological, psychological, and ecological factors into a coherent conceptual framework to explain the etiology of the disorder was lacking. Emphasizing the relevance of symptoms of depression, substance use, impulsivity, and antisocial behaviors that are often observed in PGs, they suggested that most existing typologies of gamblers have neglected to adequately cluster individuals into homogenous groups based on etiology, psychopathology, and personality. Blaszczynski and Nower postulated the pathways model that attempts to integrate biological, personality, developmental, and ecological factors described in the gambling literature into a concise theoretical framework. Their model suggests there are three major

pathways, each associated with specific vulnerability factors, demographic features, and etiological processes, that lead to the development of PG.

The model proposes that all gamblers, regardless of pathway, gamble in part because of environmental determinants (e.g., availability of gambling), operant and classical conditioning, and cognitive processes resulting in faulty beliefs related to personal skill and probability. Each of these factors has been confirmed by recent studies (Gerstein et al., 1999; Kassinov & Schare, 2001; Moore & Ohtsuka, 1999; Wulfert, Roland, Hartley, Wang, & Franco, 2005). Blaszczynski and Nower argue that *behaviourally conditioned* gamblers fluctuate between regular/heavy and excessive gambling mainly because of the effects of conditioning, distorted cognitions, and/or a series of bad judgments or poor decision-making rather than because of impaired control or premorbid psychopathological vulnerabilities. Behaviourally conditioned gamblers may abuse alcohol and exhibit elevated levels of depression and/or anxiety in response financial burden imposed by their gambling, but these conditions are not the cause of their gambling. This subtype is associated with the least severe gambling and gambling-related difficulties, and these individuals do not demonstrate signs of major premorbid psychopathology, substance abuse, impulsivity, or antisocial behaviours.

While *emotionally vulnerable* gamblers exhibit identical ecological determinants, conditioning processes, and cognitive schemas about gambling as behaviourally conditioned gamblers, these gamblers also present with premorbid depression and/or anxiety, a history of inadequate coping and problem-solving skills, and negative family background experiences, developmental variables, and life events. The emotionally vulnerable gamblers' gambling is largely motivated by a desire to regulate dysphoric

mood states and/or to meet specific psychological needs. They have elevated levels of psychopathology, particularly depression, anxiety, and alcohol dependence.

Finally, *antisocial impulsivist* PGs are considered the most psychopathological subtype and exhibit substantial psychological disturbance from gambling and are characterized by signs of potential neurological or neurochemical dysfunction. These gamblers are distinguished from emotionally vulnerable gamblers by features of impulsivity, antisocial personality disorder, and attention deficit. Antisocial impulsivist gamblers report a wide range of behavioural difficulties independent of their gambling, including excessive alcohol and polydrug experimentation, suicidality, irritability, low tolerance for boredom, and criminal behaviours. The gambling of antisocial impulsivists commences at an earlier age, reaches very severe levels, and is associated with early entry into gambling-related criminal activities.

Several recent studies provide evidence that suggests the validity of the pathways model subtypes may be strong. For example, building on an earlier study with a smaller sample (Gonzalez-Ibanez, 1994), Gonzalez-Ibanez and colleagues (Gonzalez-Ibanez, Aymami, Jimenez, Domenach, Granero, & Lourido-Ferreira, 2003) cluster analyzed responses from 110 treatment-seeking male PGs on the Symptom Checklist-90-R (SCL-90-R; Derogatis et al., 1973) and the Sensation Seeking Scale (SSS; Zuckerman, 1979). Based on their analysis, PGs were classified into three homogeneous groups. *Cluster 1* was composed of PGs who reported little or no psychopathology and who had low scores on impulsivity and sensation seeking measures. PGs in *Cluster 2* reported relatively high scores on anxiety and depression, coupled with low impulsivity and low sensation seeking. Finally, *Cluster 3* was composed of PGs who reported extreme anxiety, moderate to severe depression, and average scores on impulsivity and sensation seeking

measures. While each cluster is generally consistent with the three pathways groups, Gonzalez-Ibanez and colleagues did not validate these clusters using additional variables.

Ledgerwood and Petry (2006) surveyed the gambling motives of 149 PGs in outpatient treatment. Performing a principal component analysis on a measure of gambling experiences they found that three factors, *escape*, *dissociation*, and *egotism*, adequately described gambling motives. These factors were validated using various measures of psychopathology and personality traits. The escape factor represented gambling as a means to escape from problems and painful feelings, and was associated with a general tendency toward dissociative experiences. The dissociation factor represented experiences of dissociating while gambling. Finally, the egotism factor was characterized by gambling to impress others and was associated with heightened impulsivity. Ledgerwood and Petry noted that their escape factor closely resembled Blaszczynski and Nower's (2002) emotionally vulnerable gambler while their egotism factor paralleled the antisocial impulsivist gambler. They, however, did not measure psychopathology using clinical diagnoses and failed to measure some variables specified by the pathways model (e.g., ADHD, antisocial personality disorder).

Stewart and colleagues (Stewart & Zack, 2008; Stewart, Zack, Collins, Klein, & Fragopoulos, 2008) examined the utility of differentiating PGs based on gambling motives in two separate studies. In one study (Stewart & Zack, 2008) they administered a gambling motives measure to 193 PGs recruited from the community. Three factors extracted were labeled *social* (i.e., gambling for recreational purposes), *coping* (i.e., gambling to decrease negative affect), and *enhancement* (i.e., gambling to enhance positive affect). The coping and enhancement factors predicted the frequency of gambling, and enhancement predicted loss of control over gambling behavior. Stewart

and Zack concluded that coping and enhancement predicted gambling problems, suggesting an association between emotion-regulation motives for gambling and PG.

In their other study (Stewart, Zack, Collins, Klein, & Fragopoulos, 2008) they again examined the utility of subtyping PG according to their primary motives for gambling. In total, 158 community-recruited PGs who drink while gambling were clustered into three distinct subtypes based on responses to the Inventory of Gambling Situations (Turner & Littman-Sharp, 2006). These clusters were validated using an additional gambling motives questionnaire. The first cluster obtained positive scores on the Positive Gambling Situations factor and negative scores on the Negative Gambling Situations factor and was labeled *enhancement* gamblers. These individuals gambled solely for positive reinforcement (i.e., to increase positive emotions and excitement). The second cluster obtained positive scores on both Positive and Negative Gambling Situations factors, especially elevated on the latter factor, and was labeled *coping* gamblers because these gamblers were mainly driven by negative reinforcement. That is, they gambled to relieve worry and other unpleasant emotions. The third cluster obtained low scores on both positive and negative factors and was referred to as *low emotion regulation* gamblers because they did not report gambling for reasons related to the direct modulation of affect.

Notably, enhancement gamblers and coping gamblers demonstrated elevated rates of alcohol use problems relative to low emotion regulation gamblers. Stewart and colleagues concluded that this subtyping scheme showed similarities to those previously reported by Lesieur (2001) and Blaszczynski and Nower (2002). A relative strength of their studies is that they are among the first to attempt to validate a subtyping scheme in a

non-treatment seeking sample. However, they failed to measure diagnoses and other variables specified by the pathways model of disordered gambling.

Turner, Jain, Spence, and Zangeheh (2008) studied the extent to which Blaszczynski and Nower's (2002) model could be validated using questionnaires that captured aspects of the three pathways. Using responses from 141 community PGs on a variety of questionnaires that measured impulsivity, depression, anxiety, erroneous beliefs, and early gambling wins, these authors extracted a four component solution that fit well with the hypothesized pathways. Specifically, they found *emotional vulnerability*, *impulsivity*, *erroneous beliefs*, and *experiences of wins* components contributed to predicting PG. While the emotional vulnerability and impulsivity components mapped directly onto Blaszczynski and Nower's proposed emotionally vulnerable and antisocial impulsivist subtypes, respectively, the behaviourally conditioned subtype appeared to be separated into erroneous beliefs and experiences of wins components which reflect the distorted cognitions and conditioning histories that drive this type of gambler. As with previous studies examining the validity of the pathways model, Turner and colleagues did not measure psychiatric diagnoses in PGs.

In a sample of 141 French PGs from the general population, Bonnaire and colleagues (2009) attempted to confirm the validity of the pathways model by dividing gamblers based on the type of gambling in which they engaged. They identified three major subgroups among PGs. The first subgroup included PGs who played active games (e.g., horseracing), and demonstrated elevated sensation seeking and alexithymia scores. The second subgroup included PGs who played passive games (e.g., slot machines), and had low sensation seeking scores but elevated depression scores. Finally, the third subgroup included PGs who played games that involved strategies (e.g., roulette), and had

low sensation seeking, alexithymia, and depression scores. Bonnaire and colleagues noted their gambling subgroups directly corresponded to Blaszczynski and Nower's (2002) antisocial impulsivist, emotionally vulnerable, and behaviourally conditioned PGs, respectively.

Vachon and Bagby (2009) cluster analyzed the personality traits of 90 PGs from the community (which they compared to 138 non-PG controls) to test Blaszczynski and Nower's (2002) model of gambling. According to the authors, the best fitting model identified three PG clusters that were each characterized by a unique profile. These three clusters were validated by comparing them on various measures of psychopathology. The first cluster of PGs, which was labeled *simple* PGs, was described by personality trait scores near the normative mean and was distinguished by the relative absence of comorbid psychopathology. The second cluster of PGs, which was labeled *hedonic* PGs, was characterized by a tendency to seek excitement and pleasure, to be careless, and to act with minimal forethought. The third cluster of PGs, which was labeled *demoralized* PGs, was characterized by extreme negative affect, impulsivity, distrust, and poor motivation. The demoralized PGs also demonstrated high levels of mood, anxiety, and SUDs relative to simple and hedonic PGs. The authors note their results suggest a conceptualization of PG as an impulse control disorder with each subtype characterized by a differentiated impulsivity-trait profile. While this study is the most comprehensive validation of the pathways model of disordered gambling, some important variables (e.g., gambling motivation, ADHD, childhood maltreatment, illegal behaviours) were not examined.

Only one study to date has examined differential treatment outcomes based on PG subtype. Ledgerwood and Petry (2010) divided 229 PGs, entering a clinical trial for

cognitive behaviour therapy, into subgroups based on their scores on measures of depression, anxiety, and impulsivity. The three groups were based on Blaszczynski and Nower's (2002) three pathways subtypes, and unlike many other studies, the groups were formed based on a specific model rather than using cluster or factor analysis. In examining treatment outcome data, the authors found that behaviourally conditioned gamblers started treatment with less severe gambling problems and were most likely to be asymptomatic or to no longer meet PG criteria at post-treatment and 12-month follow-up. Antisocial impulsivist and emotionally vulnerable PGs improved at a similar rate to behaviourally conditioned gamblers, but continued to report elevated PG symptoms at post-treatment and follow-up. The authors suggested that the pathways model may not be useful for predicting differential recovery for different subtypes, but that, because of their greater gambling problem severity at baseline, antisocial impulsivist and emotionally vulnerable gamblers may need more intensive treatment than behaviourally conditioned PGs.

Overall, these studies provide empirical support for aspects of Blaszczynski and Nower's (2002) pathways model of PPG. While differences are found between studies in the operationalization and measurement of psychopathology and personality, the methods employed to classify gamblers, and the gambling subtyping schemes that were produced, it appears that three relatively distinct subtypes of PGs consistently emerge. These subtypes are differentiated based on psychopathological and personality presentations as well as their motivations for gambling. The first subtype of PG demonstrates elevated levels of depression and/or anxiety and has been referred to as the neurotic (Moran, 1970), depressive or anxious (Graham & Lowenfeld, 1986), recurrently depressed (McCormick, 1987), depression prone (Blaszczynski et al., 1990), escape seeker (Lesieur

& Blume, 1991), psychologically distressed (Steel & Blaszczynski, 1996), emotionally vulnerable (Blaszczynski & Nower, 2002; Ledgerwood & Petry, 2010), escape (Ledgerwood & Petry, 2006), coping (Stewart et al., 2008), emotional vulnerability (Turner et al., 2008), and demoralized (Vachon & Bagby, 2009) gambler. Furthermore, this type of gambler appears to be captured by Gonzalez-Ibanez and colleagues' (Gonzalez-Ibanez, 1994; Gonzalez-Ibanez et al., 2003) second PG cluster as well as Bonnaire and colleagues' (2009) second subgroup of PGs, both of which consist of gamblers reporting increased depression and anxiety. Most of these investigators have suggested that this gambler is largely motivated to gamble to relieve or escape the dysphoric moods they experience.

The second subtype of PG that consistently emerges in the literature shows marked impulsivity and gambles to increase levels of arousal and/or decrease boredom. This type of gambler has been referred to as an impulsive (Moran, 1970), passive-aggressive or emotionally unstable (Graham & Lowenfeld, 1986), chronically understimulated (McCormick, 1987), boredom prone (Blaszczynski et al., 1990), action seeker (Lesieur, 2001; Lesieur & Blume, 1991), impulsive antisocial (Steel & Blaszczynski, 1996), antisocial impulsivist (Blaszczynski & Nower, 2002; Ledgerwood & Petry, 2010), egotistic (Ledgerwood & Petry, 2006), enhancement (Stewart et al., 2008), impulsivity (Turner et al., 2008), and hedonic (Vachon & Bagby, 2009) gambler. Furthermore, this type of gambler appears to be captured by Gonzalez and colleagues' (Gonzalez-Ibanez, 1994; Gonzalez-Ibanez et al., 2003) third PG cluster as well as Bonnaire and colleagues' (2009) first subgroup of PGs, both of which consist of gamblers reporting relatively elevated levels of impulsivity or sensation seeking. In addition,

Zimmerman and colleagues' (1985) psychopathic and impulsive factors represent aspects of this type of gambler.

Some studies have also presented a third subtype of PG. This type of gambler does not exhibit serious signs of psychopathology or maladaptive personality traits, and is reported to gamble largely due to external factors (e.g., social pressure) and/or behavioural conditioning. The third subtype of gambler has been referred to as a subcultural (Moran, 1970), normal (Lesieur, 2001), behaviourally conditioned (Blaszczynski & Nower, 2002; Ledgerwood & Petry, 2010), social (Stewart & Zack, 2008), low emotion regulation (Stewart et al., 2008), and simple (Vachon & Bagby, 2009) gambler. Furthermore, this gambler is captured by Gonzalez-Ibanez and colleagues' (Gonzalez-Ibanez, 1994; Gonzalez-Ibanez et al., 2003) first PG cluster as well as Bonnaire and colleagues' (2009) third subgroup of PGs, both of which are characterized by little psychopathology and low impulsivity or sensation seeking. Turner and colleagues (2008) noted their erroneous beliefs and experiences of wins factors captured this type of gambler as well. Based on the studies published to date there is strong convergent validity for three PG subtypes. Blaszczynski and Nower (2002) present a comprehensive theoretical model of gambling subtypes that appears to capture the distinct types of gamblers consistently reported by most investigators.

It appears that the emotionally vulnerable and antisocial impulsivist gamblers of the pathways model of PPG (Blaszczynski & Nower, 2002) share similarities with the internalizing and externalizing dimensions presented in the internalizing-externalizing model of psychopathology. Unipolar mood and anxiety disorders are noted to co-occur in emotionally vulnerable gamblers, which is expected given elevated comorbidity of internalizing conditions in general and clinical populations. From the perspective of the

internalizing-externalizing model, therefore, emotionally vulnerable gamblers may be considered ‘internalizing’ gamblers. Furthermore, SUDs, antisocial personality disorder, and impulsivity are reported to be characteristic of the antisocial impulsivist gambler, which is expected given the frequent co-occurrence of these externalizing conditions and traits. From the perspective of the internalizing-externalizing model, therefore, antisocial impulsivist gamblers may be considered ‘externalizing’ gamblers. Finally, Blaszczynski and Nower’s (2002) behaviourally conditioned gambler may be seen, through the lens of the internalizing-externalizing model, as a ‘low pathology’ gambler who presents with relatively little internalizing or externalizing psychopathology. In examining the applicability of the internalizing-externalizing model to a heterogeneous sample of individuals with PTSD, Miller and colleagues (2003) discovered a subtype that was ‘low pathology’ relative to the internalizing and externalizing subtypes. The internalizing-externalizing model may provide a useful framework from which to explore differences between pathways model gambling subtypes.

Aims of Current Study

The current study had the following aims:

1. **To explore the validity of the internalizing-externalizing model of psychopathology in individuals with PPG:** Psychiatric disorders (i.e., unipolar mood, anxiety, substance use, and antisocial personality disorders) and higher-order facets of personality (i.e., positive emotionality, negative emotionality, and constraint) were measured. Principle axis factoring (PAF) was used to determine the factors underlying psychopathology, and the factors that were extracted were correlated with personality traits. Given previous literature on the factor structure

of common psychiatric disorders (e.g., Krueger, Caspi, Moffitt, & Silva, 1998) and psychiatric disorders in PTSD samples (e.g., Miller, Fogler, Wolf, Kaloupek, & Keane, 2008), it was hypothesized that a two-factor model of internalizing and externalizing factors would underlie psychopathology in disordered gamblers. The internalizing factor was predicted to load unipolar mood disorder and anxiety disorder criteria, and to be positively correlated with negative emotionality. The externalizing factor was predicted to load SUD, conduct disorder, and antisocial personality disorder criteria, and to be negatively correlated with constraint. The objective of the first aim was to apply the internalizing-externalizing model, developed to account for covariation among broad classes of psychopathology, to PPGs to improve conceptualization of the heterogeneity of comorbid psychopathology seen in these individuals.

- 2. Examining differences in internalizing and externalizing psychopathology, and gambling and other psychosocial variables in subtypes of PPGs:** Using personality traits associated with internalizing and externalizing dimensions of psychopathology and with the pathways model of PPG (i.e., negative emotionality and impulsivity), disordered gamblers were divided into subtypes. The pathways model, which was assumed to be valid given the extensive and consistent empirical literature on gambling subtypes (Milosevic & Ledgerwood, 2010), was used as a guide to group gamblers into subtypes. The three personality-based subtypes were compared on symptoms of internalizing (e.g., mood and anxiety disorders) and externalizing (e.g., SUDs, conduct disorder, antisocial personality disorder) psychopathology, gambling variables (i.e., gambling severity, age of onset of regular gambling, gambling motivation), various dimensions of

impulsivity, childhood maltreatment experiences, and additional psychosocial variables. Based on expectations of the internalizing-externalizing and pathways models, the following predictions about subtype differences were made:

- a. Behaviourally conditioned, or low pathology, gamblers would evidence lower levels of psychopathology, lower ADHD and impulsivity scores, less severe gambling, less enhancement gambling (i.e., gambling to increase positive emotions), less coping gambling (i.e., gambling to reduce or avoid negative emotions), and lower levels of childhood maltreatment relative to the other subtypes.
- b. Emotionally vulnerable, or internalizing, gamblers would evidence higher levels of internalizing psychopathology (i.e., unipolar mood and anxiety disorder) symptoms, and coping gambling relative to behaviourally conditioned gamblers; more and less severe gambling than behaviourally conditioned and antisocial impulsivist gamblers, respectively; higher and lower ADHD and impulsivity scores than behaviourally conditioned and antisocial impulsivist gamblers, respectively; and, higher levels of childhood maltreatment relative to behaviourally conditioned gamblers.
- c. Antisocial impulsivist, or externalizing, gamblers would evidence higher levels of externalizing psychopathology (i.e., SUD, conduct disorder, and antisocial personality disorder) symptoms, a younger age of onset of disordered gambling, more severe gambling, greater levels of enhancement gambling, elevated ADHD and impulsivity levels, and greater levels of illegal activity relative to behaviourally conditioned and emotionally

vulnerable gamblers; and, higher levels of childhood maltreatment relative to behaviourally conditioned gamblers.

CHAPTER III

METHODOLOGY

Sample

One hundred and fifty participants (N = 150) who met criteria for current and/or lifetime problem gambling or PG, based on scores of greater than three on the National Opinion Research Centre DSM-IV Screen for Gambling Problems (NODS; Gerstein et al., 1999) comprise the sample. A sample size of 150 participants was determined based on sample requirements for the factor analytic procedure (Nunnally, 1978; Tabachnick & Fidell, 2001). Tabachnick and Fidell (2001) recommend a minimum of 150 cases for factor analysis, and Nunnally (1978) suggests 10 cases for each item to be factor analyzed. In the current study, 12 items were subjected to factor analysis.

The current study was descriptive in that it sought to describe the personality traits and psychopathology found in PPGs. Accordingly, there were few exclusionary criteria. Exclusionary criteria were current and lifetime NODS scores below three, and an inability to understand and/or read English. Forty-two individuals were screened who did not meet NODS exclusionary criteria, and one individual who met NODS criteria verbally reported he could not read English and he was excluded from participation. The limited exclusionary criteria increased generalizability of findings to the general population of PPGs.

Recruitment

Recruitment of participants began in April 2009 and ended in August 2010, and involved the use of three strategies. Advertisements (see Appendix 5) were placed in two

Windsor, Ontario newspapers to recruit community participants who had current and/or lifetime problem gambling or PG. Community participants were also recruited through advertisements placed on two websites (i.e., Craigslist, Kijiji). Finally, participants were recruited from the University of Windsor undergraduate population through the Department of Psychology's Participant Pool (see Appendix 6). A payment of \$45 CDN, in the form of a gift certificate from a local shopping centre, was given to community gamblers, and three University of Windsor undergraduate (see Appendix 9) Psychology course bonus points were given to students who participated in the study.

Measures

Problem and PG. In the current study participants were designated as PPGs, and deemed eligible for participation, using the National Opinion Research Centre DSM-IV Screen for Gambling Problems (NODS; Gerstein et al., 1999) administered over the telephone. In addition, severity of disordered gambling behaviour was measured using the NODS. The NODS is a self-report measure designed to reflect the 10 DSM-IV diagnostic criteria for PG. The NODS includes 17 questions that evaluate whether an individual has had gambling difficulties during the last 12 months and during his or her lifetime. The 10 diagnostic criteria are scored either 0 (absent) or 1 (present), with the maximum possible score being 10. Scores of 3 or 4 on the NODS indicate problem gambling while scores of 5 or more on the NODS indicate PG. Higher scores on the NODS indicate increased severity of disordered gambling. In the current study, gambling severity scores were hypothesized to differentiate the gambling subtypes. The NODS has demonstrated strong internal consistency, good test-retest reliability, and good construct and concurrent validity, and is considered more conservative than other gambling measures in identifying

PPGs (Gerstein et al., 1999; Hodgins, 2004; Wickwire, Burke, Brown, Parker, & May, 2008).

Detailed information on involvement in specific gambling activities (e.g., type of gambling activity, frequency of gambling, and amount of money spent) was obtained using the Canadian Problem Gambling Index (CPGI; Ferris & Wynne, 2001). Furthermore, all participants were asked about their current and past participation in problem gambling treatment.

Gambling motivation. The Gambling Motives Questionnaire (GMQ) was developed by Stewart and Zack (2007) as a measure of self-reported gambling motives that was modeled after the psychometrically-sound Drinking Motives Questionnaire (DMQ; Cooper et al., 1992). Specifically, the GMQ assesses gamblers' relative frequency of gambling for each of 15 reasons. Relative frequency of gambling is rated on a 4-point scale (1 = almost never/never; 2 = sometimes; 3 = often; 4 = almost always). In the current study, total scores were calculated for the GMQ Social (i.e., gambling to increase social affiliation), Coping (i.e., gambling to reduce or avoid negative emotions), and Enhancement (i.e., gambling to increase positive emotions) subscales, and gambling subtypes were compared on gambling motivations.

The GMQ has shown good internal consistency (Stewart & Zack, 2008), and has demonstrated concurrent validity with another measure of gambling motives (Stewart, Zack, Collins, Klein, & Fragopoulos, 2008). Furthermore, subscales are differentially related to gambling severity (Stewart & Zack, 2008), with Coping and Enhancement gambling predicting elevated disordered gambling relative to Social gambling.

Multidimensional Personality Questionnaire (MPQ; Tellegen, 1982, 2000; Tellegen & Walker, in press) is a 276-item self-report inventory, constructed through an

exploratory factor-analytic process, assessing the emotional-temperamental structure of normal personality. The MPQ is composed of 11 primary trait scales (i.e., Well Being, Social Potency, Achievement, Social Closeness, Stress Reaction, Aggression, Alienation, Control, Harm Avoidance, Traditionalism, and Absorption) that structure around three orthogonal higher-order or broad traits: Positive Emotionality, Negative Emotionality, and Constraint. Positive emotionality refers to individual differences in the capacity to experience positive emotions and tendencies toward active involvement in social and occupational environments. Positive emotionality is represented with subtle definitional variations in other models of personality, such as Extraversion (Costa & McCrae, 1985; Gough, 1987; Eysenck & Eysenck, 1975), Activity (Buss & Plomin, 1975), and Ambition/Sociability (Hogan, 1986). Negative emotionality, in contrast, refers to dispositions toward negative moods and emotions, and a tendency toward adversarial interactions with others. Negative emotionality is synonymous with Neuroticism (Costa & McCrae, 1985; Eysenck & Eysenck, 1975), Emotionality (Buss & Plomin, 1975), and negative Adjustment (Hogan, 1986). The third higher order dimension, constraint, consists of traits related to impulsivity versus behavioural restraint. Constraint has been referred to by other theorists as Psychoticism (Eysenck & Eysenck, 1975), Novelty Seeking (Cloninger, 1987), Impulsivity (Buss & Plomin, 1975), Control (Gough, 1987), and Prudence (Hogan, 1986). In the current study, negative emotionality and constraint raw scores were used to classify participants into gambling subtypes, and the higher-order traits were correlated with the psychopathology factors. The primary MPQ scales have high internal consistencies and 30-day test-retest reliabilities ranging from .82 to .92 (Johnson, Spinath, Krueger, Angleitner, & Reimann, 2008). In addition, evidence for

construct and convergent validity is strong when the MPQ is compared to the MMPI (DiLalla, Gottesman, Carey, & Vogler, 1993; Sellbom & Ben-Porath, 2005).

Unipolar mood, anxiety, substance use, and antisocial behaviour disorders. The Structured Clinical Interview for DSM-IV (SCID; First, Spitzer, Gibbon, & Williams, 1997) is a semi-structured diagnostic interview used to assess current and past DSM-IV-based Axis I (i.e., clinical) and Axis II (i.e., personality) disorders. For the purpose of the current study, the SCID was used to assess current and past mood, anxiety, and SUDs and substance use variables (e.g., onset of onset of substance use, substance abuse treatment history), in addition to conduct disorder and antisocial personality disorder. SCID items directly correspond to DSM-IV diagnostic criteria, and are rated 1 (absent), 2 (subthreshold), or 3 (threshold). Furthermore, diagnoses of Axis I and Axis II disorders are rendered based on an individual meeting a threshold number of diagnostic criteria (e.g., 5 of 9 diagnostic criteria to be diagnosed with major depressive disorder). For the current study, psychiatric disorder criteria total counts were subjected to a principal axis factoring to explore the underlying structure of psychopathology in PPGs. Analyzing the underlying structure of psychopathology by using DSM total symptom counts is a strategy employed in several previous studies (e.g., Krueger, McGue, & Iacono, 2001). Gambling subtypes were also compared on psychiatric disorder criteria counts of disorders suggested by the internalizing-externalizing and pathways models (Blaszczynski & Nower, 2002).

Attention-deficit hyperactivity disorder (ADHD). The Conners' Adult ADHD Rating Scale (CAARS; Conners, Erhardt, & Sparrow, 1999) is a 66-item scale that measures both inattentive and hyperactive-impulsive dimensions of ADHD symptomatology in adults. The frequency and severity of items are assessed on a 4-point

scale (0 = not at all, never; 1 = just a little, once in a while; 2 = pretty much, often; and, 3 = very much, very frequently). Results can be organized into five different combinations to yield a Total Symptoms score, a Total DSM-IV ADHD Symptoms score, an Inattention score, a Hyperactivity/Impulsivity score, and an Index score which assesses features of ADHD in adults that are not diagnostic criteria (e.g., bad temper, underachievement, and procrastination). In the current study, total and subscale CAARS scores were compared among gambling subtypes. The CAARS has been shown to have good internal consistency and inter-rater reliability (Adler et al., 2008), as well as adequate criterion validity (Erhardt, Epstein, Conners, Parker, & Sitarenios, 1999).

Impulsivity. Given the importance of measuring impulsivity as a multi-dimensional construct using different methods of assessment, two self-report measures and one behavioural task were employed to measure various dimensions of impulsivity in the current study. Subscale and total scores on various dimensions of impulsivity were used when comparing gambling subtypes. The Barratt Impulsiveness Scale (BIS-11; Patton et al., 1995) is a 30-item self-report questionnaire comprised of items reflecting three dimensions of impulsivity: Attentional Impulsiveness (i.e., impulsivity resulting from hectic thinking and hasty decisions), Motor Impulsiveness (i.e., impulsivity resulting from fast reactions and restlessness), and Non-Planning Impulsiveness (i.e., impulsivity resulting from a drive for immediate outcomes and a failure to assess long-term consequences). BIS items are rated on a 4-point Likert scale. Adequate reliability has been established for the BIS, with Cronbach's alphas ranging between .79 and .83 for the subscales (Patton et al., 1993). Construct and convergent validity are also strong (Stanford, Mathias, Dougherty, Lake, Anderson, & Patton, 2009).

The UPPS Impulsive Behaviour Scale (UPPS; Whiteside & Lynam, 2001) is a 45-item self-rated inventory designed to measure four distinct personality pathways to impulsive behaviour. The scale was derived through a factor analytic method that included several widely used impulsivity scales. The first dimension of the scale, Urgency, refers to the tendency to experience strong impulses, frequently under conditions of negative emotionality. The second dimension, (lack of) Premeditation, refers to the tendency to think and reflect on the consequences of an act before engaging in the act. The third dimension, (lack of) Perseverance, refers to an individual's ability to remain focused on a task that may be boring or difficult. Finally, Sensation Seeking measures both a tendency to enjoy or pursue activities that are exciting, and an openness to trying new experiences that may or may not be dangerous. Whiteside and Lynam (2001) showed that the UPPS has sound internal consistency, as well as good divergent and external validity. The UPPS subscales have demonstrated Cronbach's alpha coefficients ranging from .77 to .91 for the four dimensions.

The Delayed Discounting of Monetary Rewards (Petry & Casarella, 1999) task is a computer-based behavioural measure of impulsivity that assesses an individual's tendency to discount larger delayed monetary reinforcements in favour smaller, more immediate ones. Individuals are offered a choice between a hypothetical dollar amount delivered immediately (i.e., \$1, \$3.50, \$8.75, \$17.50, \$45, \$60, \$80, \$95, \$150, \$200, \$250, \$300, \$350, \$400, \$450, \$500, \$550, \$600, \$650, \$700, \$750, \$800, \$850, \$920, \$960, \$980, and \$1000) versus \$1000 delivered after an amount of time has passed (i.e., 1 week to 25 years). The Area Under the Curve (AUC) was used as the rate of discounting, with lower AUC values (i.e., less area under the curve) indicative of steeper delay discounting curves and, thus, higher rates of impulsivity. The AUC is calculated by

extending vertical lines from the point at each delay on the curve to the x axis, thus creating several trapezoid-like shapes. The total AUC is calculated using the formula, $(x_2 - x_1)[(y_1 + y_2)]/2$, where each x represents the value of two adjacent delays and each y value represents the participant's subjective value or indifference points at correspondence delays (Myerson, Green, & Warusawitharama, 2001).

Childhood maltreatment. The Childhood Trauma Questionnaire (CTQ; Bernstein et al., 2003) is a 28-item self-report questionnaire that assesses retrospective accounts of child maltreatment. The five subscales include physical abuse, emotional abuse, emotional neglect, sexual abuse, and physical neglect. Each subscale contains five items. Higher scores on each subscale represent greater severity of the type of childhood trauma. The CTQ begins with the phrase, "When I was growing up..." and each item is rated on a 5-point scale (1 = never true to 5 = very often true) based on the frequency with which the statement/events occurred. The CTQ produces both dimensional and categorical levels for each form of trauma. For the current study, childhood maltreatment dimensional total and subscale scores were used when comparing gambling subtypes. Childhood maltreatment is high in PGs (e.g., Petry & Steinberg, 2005), and the pathways model (Blaszczynski & Nower, 2002) suggests emotionally vulnerable gamblers have elevated rates of developmental trauma. Bernstein and Fink (1998) reported mean internal consistency estimates of .92 for the sexual abuse subscale and .80 for the physical abuse subscale across eight samples. Furthermore, test-retest reliabilities have been reported as .80 for physical abuse and .81 for sexual abuse throughout a 1.6 to 5.6 month time period (Bernstein & Fink, 1998). There is also support for the convergent and discriminant validity of the CTQ (Bernstein, Ahluvalia, Pogge, & Handelsman, 1997).

Additional variables. The Addiction Severity Index (ASI; McLelland et al., 1992) measures several potential problems areas reflecting real life domains. It is composed of seven subscales, which measure the severity of medical, employment, alcohol and drug use, legal, family/social, and psychiatric problems. Composite scores, ranging from 0 to 1, take into account subjective ratings from the participant as well as a number of responses to objective questions asked in each section. For the purpose of the current study, ASI employment and legal items were used when gambling subtypes were compared. The reliability and validity of the ASI has been demonstrated in a number of samples and settings (e.g., Kosten et al., 1983; Leonhard et al., 1985; McLelland et al., 1985).

Procedure

Advertisements in local newspapers and on websites asked potential participants if they gambled frequently and were interested in participating in a study on factors associated with gambling. Advertisements also stated that participants could make \$45 CDN for approximately two to two-and-a-half hours of completing questionnaires and interviews. Potential community participants were asked to call the telephone number listed on the advertisement to determine their eligibility to participate in the current study.

Undergraduate participants responded to several gambling-related questions as part of the online Participant Pool at the University of Windsor. Notably, they were asked if they have felt the need to bet more and more money while gambling, or if they have lied to family members as a result of their gambling behaviour. These two screening questions are based on the Lie/Bet Questionnaire (Johnson, Hamer, & Nora, 1988; Johnson, Hamer, Nora, & Tan, 1997; Johnson, Hamer, Nora, Tan, Eistenstein, &

Englehart, 1998), and have been found to be the best predictors of disordered gambling status. Furthermore, the Lie/Bet Questionnaire has been shown to have a sensitivity of 0.99 (i.e., 99% of PGs were appropriately classified) and a specificity of 0.91 (i.e., 91% of non-PGs were appropriately classified). Students who endorsed at least one of these items were emailed and given a telephone number to call to determine their eligibility for the current study if they were interested in participating. Potential undergraduate participants also completed the NODS on the telephone to determine if they met current or past criteria for problem gambling or PG.

All participants meeting NODS criteria for problem or PG in their lifetime met in-person with the principal investigator at the University of Windsor's Problem Gambling Research Group house for approximately two-and-a-half hours. The in-person meeting began with the consenting process (see Appendices 7 and 8). Participants read, along with the investigator, the written informed consent form approved by the University of Windsor Research Ethics Board (REB). The consent form outlined the nature of the study, risks and benefits of participating in the study, as well as the individual's rights as a research participant. The consent form was explained in detail by the investigator, and it was ensured the participant understood the consent form and all of his or her questions were answered prior to agreeing to participate in the study.

Following the consenting process, participants completed the semi-structured diagnostic interview (i.e., the SCID), structured interviews (i.e., CPGI and ASI scales), self-report questionnaires, and computer-based impulsivity task. At the end of the in-person meeting, all participants were asked if they currently were interested in problem gambling treatment and their responses were recorded as a *Yes* or *No*. All participants were then given a referral for problem gambling treatment (see Appendix 11). In addition,

all participants received a referral package of mental health treatment resources in the community (see Appendix 11). Community participants received a \$45 CDN gift certificate for their participation, while undergraduate student participants received three bonus points to be added to one of their undergraduate psychology course final grades.

All procedures received approval from the University of Windsor REB prior to beginning the study.

Data Analysis

Principal axis factoring (PAF) was used to examine the factor structure underlying psychopathology in disordered gamblers. An oblique rotation method (i.e., direct oblimin) was used to allow for correlation between factors. Factors produced by the PAF were correlated with higher-order personality traits (i.e., positive emotionality, negative emotionality, and constraint). These statistical methods were used to test to the first hypothesis that the structure of psychopathology in PPGs consisted of internalizing and externalizing dimensions related to negative emotionality and impulsivity, respectively.

Multivariate analysis of variance (MANOVA) was used to compare gambling subtypes on continuous variables, including demographic, psychopathology, personality, and childhood maltreatment variables. Separate MANOVAs were run for each group of variables (e.g., gambling, psychopathology, personality, etc.). Chi-square analyses were used to compare gambling subtypes on dichotomous variables (e.g., gender). Tukey's post-hoc test was used to determine specific differences between subtypes. These statistical methods were used to test the second hypothesis that gambling subtypes could be differentiated based on internalizing and externalizing psychopathology and gambling and other psychosocial variables.

CHAPTER IV

RESULTS

Descriptive Statistics

One-hundred and fifty ($N = 150$) individuals participated in the study, with 50% of the sample being male (see Appendix 1 for a comparison of male and female participants). Approximately 60% ($N = 91$) of the sample were recruited from newspaper and online advertisements, while approximately 40% ($N = 59$) were recruited from the University of Windsor Psychology Participant Pool (see Appendix 2 for a comparison of community and student participants). The average participant age was 36.29 years ($SD = 15.46$, range 18 to 80 years), and most participants (i.e., 78%, $N = 117$) were Caucasian. In regards to marital status, 46.7% ($N = 70$) reported being single, 28% ($N = 42$) married or in common-law relationship, and 25.3% ($N = 38$) divorced, separated, or widowed. About 40% ($N = 58$) of the sample were currently students, while 24.7% ($N = 37$) reported being employed, 18.7% ($N = 28$) unemployed, 12% ($N = 18$) on disability, and 6% ($N = 9$) retired. Therefore, 36.7% of the sample was neither a student nor employed. The average number of years of formal education in the sample was 13.85 years ($SD = 2.62$, range 8 to 22 years). The median past year income was \$30,000 (range \$0 to \$200,000).

In regards to disordered gambling status, 92% ($N = 138$) of the sample met NODS criteria for PG at some point in their lifetime while 8% ($N = 12$) only met criteria for problem gambling at some point in their lifetime. Specifically, in the *past year* 73.3% ($N = 110$) of the sample met NODS criteria for PG, 15.3% ($N = 23$) met criteria for problem gambling, and 11.3% ($N = 17$) were deemed non-problem gamblers. Furthermore, rates of

PG, problem gambling, and non-problem gambling *prior to the past year* were 74.0% (N = 111), 16.0% (N = 24), and 10.0% (N = 15), respectively (*Note*: all participants who were deemed non-problem gamblers in the past year at least met criteria for problem gambling prior to the past year, and all participants who were deemed non-problem gamblers prior to the past year at least met criteria for problem gambling in the past year). In regards to gambling severity scores, the average number of NODS criteria endorsed was 6.03 ($SD = 2.66$, range 0 to 10) and 6.24 ($SD = 2.86$, range 0 to 10) for the past year and for lifetime, respectively. The average highest NODS score (i.e., the highest score received on the NODS at any point in time) was 7.41 ($SD = 1.87$, range 0 to 10). The average Canadian Problem Gambling Index Short-Form (CPGI-SF) total score for the past year was 10.12 ($SD = 6.34$, range 0 to 24), which meets the cut-off criteria for problem gambling.

The average age of onset of any gambling behaviour was 18.49 years ($SD = 8.07$, range 4 to 57), while the average age of onset of regular gambling (i.e., gambling three or more episodes per week) was 24.76 years ($SD = 12.43$, range 6 to 67 years). In the current study, the initiation of regular gambling was used as a proxy measure for the beginning of problematic gambling behaviour. In regards to types of gambling activities engaged in at least twice per week in the past year, the current sample engaged in the following activities: 20.7% lottery, 18% scratch tickets, 2.7% horse racing, 10.6% bingo, 24.6% casino slot machine, 7.3% casino poker, 4.0% casino blackjack, 4.0% casino roulette, 1.3% casino craps, 9.4% sports lotteries, 4.6% card and board games, 2.7% games of skill, and 12.7% internet gambling (see Figure 1). The average gambler engaged in 6.49 types of gambling activities in the past year ($SD = 3.35$, range 0 to 17). The median maximum amount of money spent on any gambling activity in one day in the past year

was \$400 (range \$0 to \$15000). Over half (i.e., 54%, N = 81) of the current sample reported using alcohol or drugs while gambling within the past year. Twenty-four percent (24%, N = 36) of the sample reported currently being interested in problem gambling treatment, and 16% (N = 24) had some problem gambling treatment in the past. The amount of past treatment was generally quite minimal, however, with most participants who sought treatment (75%, N = 18) having attended one or two Gamblers Anonymous meetings.

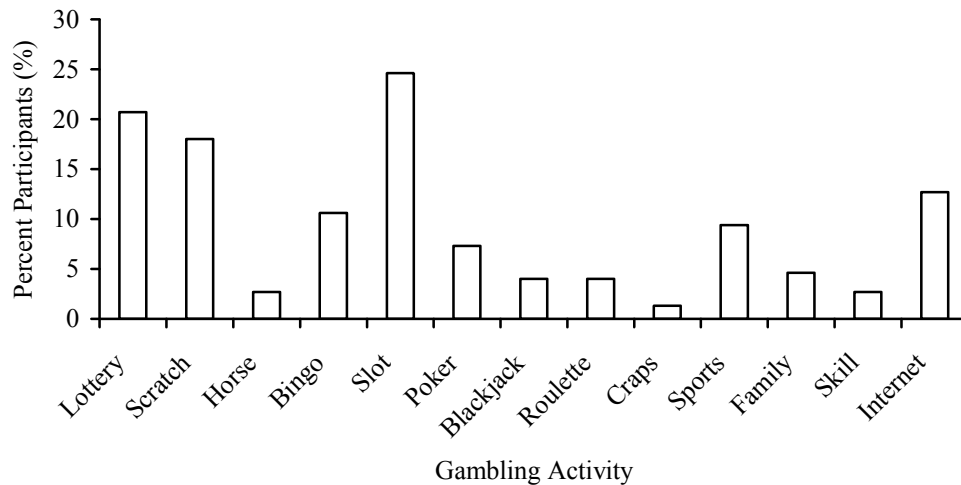


Figure 1: Percentage of participants engaging in gambling activities (at least twice per week in past year) (N = 150)

Hypothesis 1: Factor Structure of Psychopathology

Prior to statistical analyses, data were screened using guidelines set forth by Tabachnick and Fidell (2001). Given that different screening procedures apply for

ungrouped data and for grouped data, and the current study analyzed both ungrouped (i.e., principle axis factoring) and grouped data (i.e., MANOVA), data were screened twice.

Raw data were initially examined for missing values and for accuracy of data entry. Participants' responses to questionnaire items were individually examined by the investigator for missed responses immediately following their completion during the in-person meeting. This was done to ensure that all questionnaire items were answered. When unanswered questions were discovered, participants were asked to complete the items prior to leaving the testing session. No cases or variables were deleted as there were ultimately no missing data.

Accuracy of data entered into the data file was assessed by examining descriptive statistics and graphical representations of variables. For continuous variables, values that were out of the expected range were corrected. For discrete variables, out-of-range numbers were also evaluated, and any data that were entered incorrectly were corrected.

Data were also screened for outliers. Cases that were extreme were first examined to determine if the data were correctly entered. Univariate outliers for dichotomous variables were assessed for using frequency distributions. For continuous variables, outliers were assessed separately for ungrouped data and for grouped data. For ungrouped data, univariate and multivariate outliers were sought among all cases at once. For grouped data, outliers were sought separately within each group. Standardized scores (i.e., z scores in excess of 3.29) and histograms were examined to determine univariate outliers, while Mahalanobis distances' were used to determine multivariate outliers. When an extreme case was deemed to be from the intended sample (but the distribution for the variable in the population had more extreme values than a normal distribution), the case was retained but the value was changed to minimize the impact of the outlier. Raw

scores were assigned to the outlying variables that were either one unit smaller or larger than the next most extreme score in the distribution. Five cases with extremely high z scores were found to be univariate outliers (i.e., yearly income, maximum money spent on gambling in one day, number of lifetime episodes of major depressive disorder, lifetime number of arrests, lifetime number of charges), and the same five cases were identified through Mahalanobis distance as multivariate outliers.

Prior to statistical analyses, all variables were examined for fit between their distributions and the assumptions of multivariate analysis. The assumption of multivariate normality applies differently to ungrouped and grouped data. For analyses when participants were not grouped, this assumption applies to the distributions of the variables (or residuals) themselves; each variable is itself normally distributed and the relationship between pairs of variables, if present, are linear and homoscedastic. For analyses when participants were grouped, the assumption applies to the sample distributions of the means of the variables.

Normality of variables was assessed using graphical (i.e., frequency histograms) and statistical (i.e., z scores) methods. Alpha levels of 0.01 were used to evaluate the significance of skewness and kurtosis given the moderate size of the sample. Notably, SCID disorder criteria count variables were positively skewed based on histograms and skewness z scores (absolute values greater than 1.96 were used because of the moderate sample size).

The assumption of linearity was assessed using bivariate scatterplots between pairs of variables. If both variables were normally distributed and linearly related, the scatterplot was oval-shaped. Given the number of variables in the present study, statistics on skewness were used to screen only pairs that were likely to depart from linearity.

For ungrouped data the assumption of homoscedasticity was assessed using scatterplots. For grouped data, the assumption of homogeneity of variance was assessed using Levene's test of homogeneity of variance because it is typically sensitive to departures from normality. When Levene's test was found to be significant it was concluded the variances between groups was different and the assumption was violated. Violations of homogeneity of variance could have been corrected by transformation of the dependent variable scores, but interpretation is limited to the transformed scores. Therefore, in some cases, untransformed variables were used with a more stringent alpha level (0.01 for severe violations). Homogeneity of variance-covariance matrices was assessed using Box's M.

Given that, for ungrouped data, it is preferable to transform variables to normality unless interpretation is not feasible with transformed scores, data were transformed. After data were transformed, each variable was examined to determine if it was normally or near-normally distributed after the transformation. When variables are skewed to about the same moderate extent, improvement of analysis with transformation was marginal. Several transformations were attempted before the most helpful one was found. Finally, logarithmic transformation was used when necessary in order to reduce extreme skewness and kurtosis. For DSM-IV criteria count variables and amount of money gambled, transformation did not significantly reduce skewness. Therefore, criteria count untransformed data were used.

Principal axis factoring (PAF) was used to examine the factor structure underlying the manifest psychopathology in disordered gamblers. An oblique rotation method (i.e., direct oblimin) was used to allow factors to be correlated. The appropriate number of

factors extracted was determined by examining the scree plot. That is, factors to the left of the point of inflexion, i.e., where the slope of the line changes dramatically, were retained. In order to determine if there were data points clustered together near the point of inflexion, multiple factor analyses were run with two-, three-, and four-factor solutions manually specified. Following rotation, the item loading tables of the different solutions were compared, and the solution with the “cleanest” factor structure (i.e., item loadings above 0.3, no or few cross loadings, no factors with fewer than three items) was determined to have the best fit to the data (Tabachnick & Fidell, 2001). Kaiser-Meyer-Olkin (KMO) measure of sample adequacy, Bartlett’s test of sphericity, and the anti-image correlation matrix were also examined. According to Hutchenson and Sofroniou (1999), KMO values between 0.5 and 0.7 are mediocre, values between 0.7 and 0.8 are good, values between 0.8 and 0.9 are great, and values above 0.9 are superb. Items showing anti-image correlation matrix values above 0.5 were included in the factor analysis, while values below 0.5 were excluded from the analysis (Field, 2009).

DSM-IV disorder criteria total counts for disorders with symptoms that were relatively common in the sample were included in the PAF. These variables are continuous. When less than 15% of the sample exhibited at least one criterion for a disorder, the criteria count variable for the disorder was excluded from the PAF. Using this strategy, the following disorder criteria counts were included in the PAF: current major depressive disorder, past major depressive disorder, current dysthymic disorder, lifetime social anxiety disorder, lifetime specific phobia, lifetime posttraumatic stress disorder, current generalized anxiety disorder, lifetime alcohol abuse, lifetime alcohol dependence, lifetime cannabis abuse, lifetime cannabis dependence, past conduct disorder, and lifetime antisocial personality disorder (See Figure 2). However, given that

the specific phobia variable produced an anti-image correlation value below 0.5 it was excluded from the analysis. The following disorder criteria counts were also excluded from the PAF (including percentages of the sample meeting at least one criterion for the disorder): current panic disorder (2%), past panic disorder (10.7%), lifetime sedative abuse (3.3%) and lifetime sedative dependence (8.7%), lifetime stimulant abuse (8%) and life stimulant dependence (8%), lifetime opioid abuse (13.3%) and lifetime opioid dependence (13.3%), lifetime cocaine abuse (10.7%) and lifetime cocaine dependence (12%), and lifetime hallucinogen abuse (9.3%) and lifetime hallucinogen dependence (6.7%). The prevalences of DSM-IV-TR diagnoses in the sample are included in Figure 3.

A PAF was conducted on the 12 items with oblique rotation (direct oblimin). The Kaiser-Meyer-Olkin measure verified the sampling adequacy for the analysis, KMO = 0.68, and all KMO values for individual items were above the acceptable limit of 0.50. Bartlett's test of sphericity $\chi^2(66) = 721.36, p < 0.001$, indicated that correlations between items were sufficiently large for PAF. An initial analysis was run to obtain eigenvalues for each factor in the data. Four factors had eigenvalues over Kaiser's criterion of one and in combination explained 63.48% of the variance. The scree plot (see Appendix 3) was ambiguous and showed inflexions that might justify retaining two, three, or four factors. Additional factor analyses were run with two-, three-, and four-factor solutions manually specified (see Appendices 4 and 5 for pattern and structure matrices for three- and four-factor solutions). Following rotation, the pattern and structure matrices of the different solutions were compared, and the solution with the "cleanest"

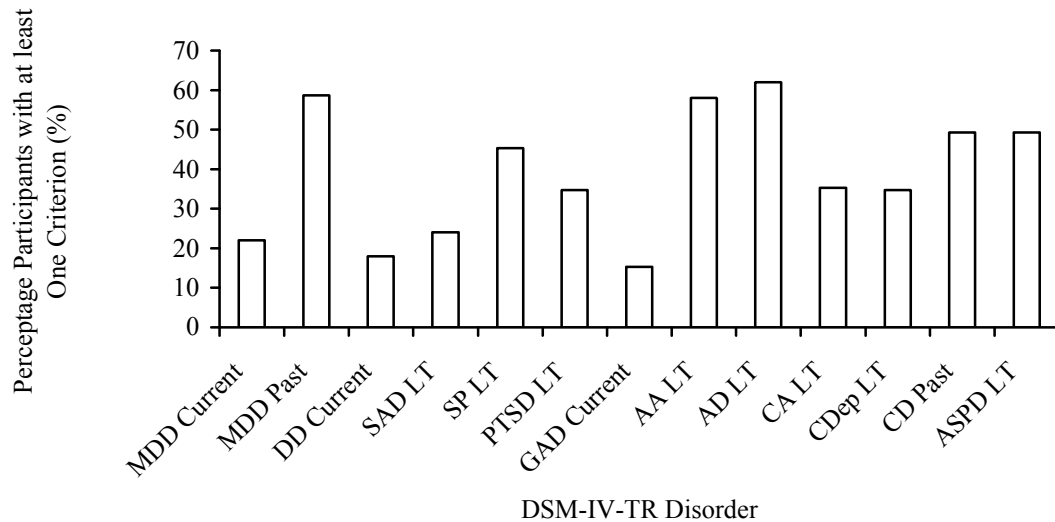


Figure 2: Percentage of participants with at least one DSM-IV-TR disorder criteria (N = 150)

AA = DSM-IV-TR alcohol abuse; AD = DSM-IV-TR alcohol dependence; ASPD = DSM-IV-TR antisocial personality disorder; CA = DSM-IV-TR cannabis abuse; CD = DSM-IV-TR cannabis dependence; DD = DSM-IV-TR dysthymic disorder; GAD = DSM-IV-TR generalized anxiety disorder; LT = lifetime; MDD = DSM-IV-TR major depressive disorder; SAD = DSM-IV-R social anxiety disorder; SP = DSM-IV-TR specific phobia

factor structure (i.e., item loadings above 0.3, no or few cross loadings, no factors with fewer than three items) was determined to have the best fit to the data (Tabachnick & Fidell, 2001). The solution that best fit the data was a two-factor solution, which explained 47.47% of the variance. Tables 1 and 2 contain the factor loadings (pattern and structure matrices) for the rotated two-factor solution. Factor 1 explained 31.23% of total variance, and consisted of total criteria counts for lifetime alcohol abuse, lifetime alcohol dependence, lifetime cannabis abuse, lifetime cannabis dependence, past conduct

disorder, and lifetime antisocial personality disorder. Given this factor contained loadings of externalizing disorder criteria total counts it was labeled *Externalizing*. Factor 2

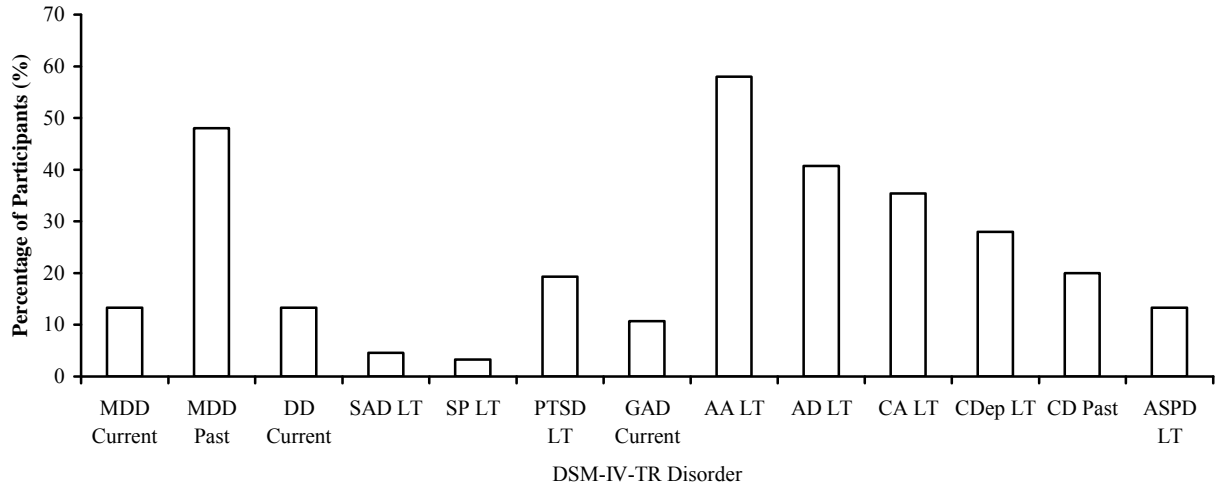


Figure 3: Prevalence rates of DSM-IV-TR disorders (N = 150)

AA = DSM-IV-TR alcohol abuse; AD = DSM-IV-TR alcohol dependence; ASPD = DSM-IV-TR antisocial personality disorder; CA = DSM-IV-TR cannabis abuse; CD = DSM-IV-TR cannabis dependence; DD = DSM-IV-TR dysthymic disorder; GAD = DSM-IV-TR generalized anxiety disorder; LT = lifetime; MDD = DSM-IV-TR major depressive disorder; SAD = DSM-IV-R social anxiety disorder; SP = DSM-IV-TR specific phobia

explained an additional 16.24% of variance, and consisted of total criteria counts for current major depressive disorder, past major depressive disorder, current dysthymic disorder, and current generalized anxiety disorder. Given this factor contained loadings of internalizing disorder criteria total counts it was labeled *Internalizing*. Total criteria counts for social anxiety disorder and posttraumatic stress disorder did not load highly on either factor. The Externalizing and Internalizing factors were positively correlated with each other ($r = 0.196, p = 0.02$).

Bivariate correlational analyses between the two factors and the higher-order, MPQ personality traits positive emotionality, negative emotionality, and constraint were conducted (see Table 3). Positive emotionality was significantly negatively correlated with the Internalizing factor ($r = -0.41, p < 0.01$), but not significantly correlated with the Externalizing factor. Negative emotionality was significantly positively associated with the Externalizing factor ($r = 0.24, p < 0.01$) and the Internalizing factor ($r = 0.39, p < 0.01$). Finally, CON was significantly negatively associated with the Externalizing factor ($r = -0.32, p < 0.01$), but not significantly correlated with the Internalizing factor.

Table 1

Pattern Matrix for Two-Factor Structure of Psychopathology (N = 150)

	Factor Loadings	
	1	2
Alcohol abuse (lifetime)	0.692	0.140
Alcohol dependence (lifetime)	0.654	0.293
Cannabis abuse (lifetime)	0.826	-0.080
Cannabis dependence (lifetime)	0.799	-0.080
Conduct disorder (past)	0.463	0.030
Antisocial personality disorder (lifetime)	0.742	0.223
Major depressive disorder	-0.049	0.743

(current)		
Major depressive disorder	0.010	0.475
(past)		
Dysthymic disorder	0.050	0.639
(current)		
Social anxiety disorder	0.090	-0.060
(lifetime)		
Posttraumatic stress disorder (lifetime)	0.050	0.240
Generalized anxiety disorder (current)	-0.010	0.377
Eigenvalue	3.75	1.95
% of variance (pre-rotation)	31.23	16.24

Table 2

Structure Matrix for Two-Factor Structure of Psychopathology (N = 150)

	Factor Loadings	
	1	2
Alcohol abuse (lifetime)	0.711	0.237
Alcohol dependence (lifetime)	0.695	0.285

Cannabis abuse (lifetime)	0.814	0.030
Cannabis dependence (lifetime)	0.788	0.030
Conduct disorder (past)	0.467	0.090
Antisocial personality disorder (lifetime)	0.773	0.236
Major depressive disorder (current)	0.060	0.736
Major depressive disorder (past)	0.080	0.476
Dysthymic disorder (current)	0.143	0.646
Social anxiety disorder (lifetime)	0.080	-0.050
Posttraumatic stress disorder (lifetime)	0.080	0.247
Generalized anxiety disorder (current)	0.040	0.376
Eigenvalue	3.75	1.95
% of variance (pre-rotation)	31.23	16.24

Table 3

Correlations between Psychopathology Factors and Multidimensional Personality Questionnaire (MPQ) Personality Traits (N = 150)

Variable	Positive Emotionality	Negative Emotionality	Constraint
Externalizing Factor	-0.07	0.24*	-0.32*
Internalizing Factor	-0.41*	0.39*	-0.12
Positive Emotionality	1.00	-0.12	0.13
Negative Emotionality	-	1.00	-0.15
Constraint	-	-	1.00

*p < 0.01

Hypothesis 2: Subtypes of PPGs

Participants were divided into gambling subtypes using a strategy similar to that used by Ledgerwood and Petry (2010). Raw scores on negative emotionality and constraint were used to classify participants into one of the three, personality-based subtypes. These personality traits were chosen to classify gamblers given their association with both the internalizing-externalizing model and the pathways model. Furthermore, negative emotionality and impulsivity underlie the internalizing and externalizing forms of psychopathology expected to differentiate the gambling subtypes. Participants were classified as high on negative emotionality if their score was greater than one standard

deviation higher than the mean of the normative sample (Tellegen, in press; negative emotionality $M = 30.2$, $SD = 15.43$). Participants who scored lower than one standard deviation above the normative mean on negative emotionality (and were therefore average or below average with respect to negative emotionality) were assigned to the behaviourally conditioned, or low pathology, subtype. Participants were characterized as high on impulsivity (higher impulsivity reflected by lower constraint scores) if their constraint score was one standard deviation or more below the average of the normative sample (Tellegen, in press; constraint $M = 59.16$, $SD = 15.19$). Participants who scored greater than one standard deviation above the normative mean were placed in the low to average impulsivity group. Among the participants who scored high on negative emotionality, those who scored relatively lower on the impulsivity measure were assigned to the emotionally vulnerable, or internalizing, subtype, and those who scored high on impulsivity were assigned to the antisocial impulsivist, or externalizing, subtype. This resulted in 65 participants being categorized as behaviourally conditioned, 55 participants as emotionally vulnerable, and 30 participants as antisocial impulsivist. The subtypes received the following mean scores on positive emotionality, negative emotionality, and constraint, respectively: behaviourally conditioned 42.09 ($SD = 14.79$), 30.55 ($SD = 9.13$), and 50.66 ($SD = 14.81$); emotionally vulnerable 39.00 ($SD = 19.06$), 62.27 ($SD = 11.98$), and 56.27 ($SD = 8.07$); antisocial impulsivist 33.53 ($SD = 20.37$), 66.53 ($SD = 14.88$), and 32.17 ($SD = 8.31$). No group differences in positive emotionality were found. Behaviourally conditioned gamblers scored significantly lower than both emotionally vulnerable and antisocial impulsivist gamblers on negative emotionality, while there were no differences between emotionally vulnerable and antisocial impulsivist gamblers on negative emotionality. Antisocial impulsivist gamblers scored significantly lower on

constraint than behaviourally conditioned and emotionally vulnerable gamblers, while emotionally vulnerable gamblers significantly scored higher than behaviourally conditioned gamblers on constraint (see Table 4).

Table 4

Subtype Comparisons on Multidimensional Personality Questionnaire (MPQ) Personality Traits

Variable	Behaviourally Conditioned (N = 65)	Emotionally Vulnerable (N = 55)	Antisocial Impulsivist (N = 30)	F and p values
Positive Emotionality	42.09 (14.79)	39.00 (19.06)	33.53 (20.37)	$F(2, 147) = 2.43, p = 0.09$
Negative Emotionality	30.55 (9.13) ^a	62.27 (11.98)	66.53 (14.88)	$F(2, 147) = 154.56, p < 0.01$
Constraint	50.66 (14.81) ^b	56.27 (8.07)	32.17 (8.31) ^c	$F(2, 147) = 43.55, p < 0.01$

^aBehaviourally Conditioned < Emotionally Vulnerable & Antisocial Impulsivist

^bBehaviourally Conditioned < Emotionally Vulnerable

^cAntisocial Impulsivist < Behaviourally Conditioned & Emotionally Vulnerable

Comparing Gambling Subtypes

Multivariate analysis of variance (MANOVA) was used to compare gambling subtypes on continuous variables, including demographic, psychiatric, substance use,

personality, and childhood maltreatment variables. Separate MANOVAs were run for each group of variables (e.g., gambling, psychiatric, personality, etc.). Chi-square analyses were used to compare gambling subtypes on dichotomous variables. Given that homogeneity of variance assumptions were violated, and sample sizes were not equal, MANOVA was not considered robust in the current analyses. Tukey's post-hoc test was used to determine specific differences between subtypes. Analyses were conducted using transformed and non-transformed data and no differences in results were found; therefore, non-transformed data analyses will be presented to facilitate interpretation of results.

In regards to demographic characteristics (see Table 5), gambling subtypes did not differ in age or in gender. Behaviourally conditioned gamblers, however, reported higher levels of formal education than both emotionally vulnerable and antisocial impulsivist gamblers. Furthermore, the subtypes of gamblers did not differ in terms of recruitment source (i.e., percentage recruited from the community versus from the student population).

Gambling subtypes were compared on 11 continuous gambling-related variables, with a significance level alpha equal to 0.05 (see Table 5). There was an overall significant multivariate effect of gambling variables by gambling subtype ($V = 0.35$, $F = 2.23$, $p < 0.01$). In regards to NODS scores there was a significant difference on past year gambling severity, with emotionally vulnerable and antisocial impulsivist gamblers scoring higher than behaviourally conditioned gamblers. In addition, there was a significant difference on highest NODS score, with antisocial impulsivist gamblers scoring higher than behaviourally conditioned gamblers. There were no differences between groups, however, in lifetime NODS severity. Furthermore, emotionally vulnerable and antisocial impulsivist gamblers scored higher on past year gambling

severity than behaviourally conditioned gamblers on the CPGI-SF. While there were no significant differences in Social or Enhancement gambling motives, emotionally vulnerable and antisocial impulsivist gamblers self-reported gambling for Coping reasons more frequently than behaviourally conditioned gamblers. There were no group differences in age at first gambling, maximum money spent per day on gambling in the past year, or number of gambling activities engaged in during the past year. Antisocial impulsivist gamblers began gambling regularly at a younger age than both behaviourally conditioned and emotionally vulnerable gamblers.

Table 5

Subtype Comparisons on Demographic and Gambling-Related Variables

	Behaviourally Conditioned (N = 65)	Emotionally Vulnerable (N = 55)	Antisocial Impulsivist (N = 30)	F, chi-square, and p values
Age (years)	38.20 (16.05)	35.87 (15.55)	32.90 (13.76)	$F(2, 147) = 1.24, p = 0.29$
Sex (% male)	49.20	49.10	53.30	$\chi^2(2) = 0.17, p = 0.92$
Education (years)	14.62 (2.73) ^a	13.41 (2.30)	13.00 (2.55)	$F(2, 147) = 5.45, p < 0.01$
Recruitment source (% community)	52.3	70.9	60.0	$\chi^2(2) = 4.33, p = 0.12$

NODS past year	5.11 (2.72) ^b	6.73 (2.34)	6.77 (2.53)	$F(2, 147) = 7.57, p < 0.01$
NODS lifetime	6.05 (2.75)	5.96 (3.04)	7.17 (2.64)	$F(2, 147) = 2.01, p = 0.14$
NODS highest	6.89 (1.89) ^c	7.65 (1.76)	8.10 (1.77)	$F(2, 147) = 5.30, p < 0.01$
CPGI-SF	8.21 (5.99) ^b	11.51 (5.98)	11.70 (6.83)	$F(2, 147) = 5.49, p < 0.01$
GMQ coping	10.22 (3.93) ^b	11.98 (4.45)	12.90 (4.47)	$F(2, 147) = 4.95, p < 0.01$
GMQ enhancement	13.85 (4.07)	14.65 (3.69)	15.33 (4.05)	$F(2, 147) = 1.60, p = 0.21$
GMQ social	9.88 (3.72)	10.55 (3.26)	11.23 (3.41)	$F(2, 147) = 1.85, p = 0.16$
Age first gambling (years)	18.66 (8.08)	19.65 (8.99)	15.97 (5.56)	$F(2, 147) = 2.08, p = 0.13$
Age first regular gambling (years)	25.97 (13.83)	26.07 (12.73)	19.73 (6.15) ^d	$F(2, 147) = 3.15, p = 0.046$
Maximum past year, one day spending (dollars)	1015.31 (2590.75)	600.64 (807.45)	1027.73 (2739.64)	$F(2, 147) = 0.66, p = 0.52$
Gambling	5.91 (3.49)	6.73 (3.03)	7.30 (3.49)	$F(2, 147) =$

activities (past
year)

2.02, $p = 0.14$

CPGI SF = Canadian Problem Gambling Index Short-Form; GMQ = Gambling Motives Questionnaire;

NODS = National Opinion Research Centre DSM-IV Screen for Gambling Problems

^aBehaviourally Conditioned > Emotionally Vulnerable & Antisocial Impulsivist, $p < 0.01$

^bBehaviourally Conditioned < Emotionally Vulnerable & Antisocial Impulsivist, $p < 0.01$

^cBehaviourally Conditioned < Antisocial Impulsivist, $p < 0.01$

^dAntisocial Impulsivist < Behaviourally Conditioned & Emotionally Vulnerable, $p = 0.046$

Substance Use Variables

Gambling subtypes were compared on seven continuous substance use variables, with a significance level alpha equal to 0.05 (see Table 6). There was an overall significant multivariate effect of substance use variables by gambling subtype ($V = 0.17$, $F = 1.83$, $p = 0.035$). There was a significant group difference in total lifetime alcohol abuse criteria, with antisocial impulsivist gamblers meeting more criteria than behaviourally conditioned and emotionally vulnerable gamblers. Antisocial impulsivist gamblers had significantly more lifetime alcohol dependence criteria than behaviourally conditioned and emotionally vulnerable gamblers. Furthermore, antisocial impulsivist gamblers had significantly more lifetime cannabis abuse criteria than behaviourally conditioned gamblers, as well as more lifetime cannabis dependence criteria than behaviourally conditioned gamblers.

Antisocial impulsivist gamblers used significantly more drug classes in their lifetimes than behaviourally conditioned and emotionally vulnerable gamblers. Antisocial impulsivist gamblers had significantly more drug abuse diagnoses in their lifetimes than

behaviourally conditioned gamblers, and more drug dependence diagnoses in their lifetimes than behaviourally conditioned gamblers.

Table 6

Subtype Comparisons on Substance Use Variables

	Behaviourally Conditioned (N = 65)	Emotionally Vulnerable (N = 55)	Antisocial Impulsivist (N = 30)	F and p values
Alcohol abuse (lifetime)	1.00 (1.08)	1.13 (1.38)	1.90 (1.54) ^a	$F(2, 147) = 5.25, p < 0.01$
Alcohol dependence (lifetime)	1.74 (2.07)	2.24 (2.51)	3.63 (2.58) ^a	$F(2, 147) = 6.74, p < 0.01$
Cannabis abuse (lifetime)	0.46 (0.92) ^b	0.71 (0.94)	1.07 (1.28)	$F(2, 147) = 3.75, p = 0.03$
Cannabis dependence (lifetime)	0.78 (1.60) ^b	1.42 (2.04)	2.17 (2.38)	$F(2, 147) = 5.40, p < 0.01$
Drug classes used (lifetime)	1.85 (1.76)	2.56 (2.04)	3.77 (1.48) ^a	$F(2, 147) = 11.48, p < 0.01$
Drug abuse disorders (lifetime)	0.46 (1.02) ^b	0.89 (1.29)	1.43 (1.50)	$F(2, 147) = 6.63, p < 0.01$

Drug dependence disorders (lifetime)	0.40 (0.83) ^b	0.71 (0.96)	1.17 (1.26)	$F(2, 147) = 6.46, p < 0.01$
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^aAntisocial Impulsivist > Behaviourally Conditioned & Emotionally Vulnerable

^bBehaviourally Conditioned < Antisocial Impulsivist

Psychiatric Variables

Gambling subtypes were compared on nine continuous psychiatric variables, with a significance level alpha equal to 0.05 (see Table 7). There was an overall significant multivariate effect of psychiatric variables by gambling subtype ($V = 0.22, F = 1.88, p = 0.02$). There were no differences between groups in total number of current major depressive disorder criteria or lifetime number of major depressive disorder episodes, although antisocial impulsivist gamblers had a higher number of past major depressive disorder criteria than behaviourally conditioned and emotionally vulnerable gamblers. Emotionally vulnerable gamblers had greater total number of current dysthymic disorder criteria than behaviourally conditioned gamblers. No group differences in total lifetime criteria for social anxiety disorder or generalized anxiety disorder were found. Antisocial impulsivist gamblers demonstrated a greater total number of lifetime posttraumatic stress disorder criteria than behaviourally conditioned gamblers. Behaviourally conditioned gamblers exhibited lower internalizing psychopathology scores than the other subtypes. Finally, antisocial impulsivist gamblers had a greater number of lifetime psychiatric treatment episodes than both behaviourally conditioned and emotionally vulnerable gamblers.

Table 7

Subtype Comparisons on Psychiatric Variables

	Behaviourally Conditioned (N = 65)	Emotionally Vulnerable (N = 55)	Antisocial Impulsivist (N = 30)	F and p values
Major depressive disorder (current)	0.68 (1.76)	1.36 (2.61)	1.63 (2.72)	$F(2, 147) = 2.25, p = 0.11$
Major depressive disorder (past)	3.47 (3.56)	3.49 (3.58)	5.33 (3.54) ^a	$F(2, 147) = 3.23, p = 0.04$
Major depressive disorder episodes (lifetime)	4.58 (12.17)	4.20 (8.40)	6.37 (10.20)	$F(2, 147) = 0.44, p = 0.65$
Dysthymic disorder (current)	0.46 (1.63) ^b	1.53 (2.68)	1.60 (2.79)	$F(2, 147) = 4.12, p = 0.02$
Social anxiety disorder (lifetime)	0.86 (1.49)	0.67 (1.47)	0.70 (1.58)	$F(2, 147) = 0.27, p = 0.77$

Posttraumatic stress disorder (lifetime)	2.17 (4.63) ^c	4.07 (6.22)	5.43 (6.98)	$F(2, 147) = 3.70, p = 0.03$
Generalized anxiety disorder (current)	0.69 (2.15)	1.04 (2.49)	1.50 (3.06)	$F(2, 147) = 1.12, p = 0.33$
Internalizing factor score	-0.24 (0.60) ^d	0.08 (0.98)	0.37 (0.96)	$F(2, 147) = 6.06, p < 0.01$
Psychiatric treatments (lifetime)	0.89 (1.17)	1.11 (1.49)	2.07 (2.68) ^a	$F(2, 147) = 5.15, p < 0.01$

^aAntisocial Impulsivist > Behaviourally Conditioned & Emotionally Vulnerable

^bBehaviourally Conditioned < Emotionally Vulnerable

^cBehaviourally Conditioned < Antisocial Impulsivist

^dBehaviourally Conditioned < Emotionally Vulnerable & Antisocial Impulsivist

Antisocial and ADHD-related Variables

Gambling subtypes were compared on eight continuous antisocial and ADHD-related difficulties, with a significance level alpha equal to 0.05 (see Table 8). There was an overall significant multivariate effect of antisocial and ADHD-related variables by gambling subtype ($V = 0.23, F = 2.74, p < 0.01$). Antisocial impulsivist gamblers met more conduct disorder criteria in their childhoods and more antisocial personality disorder criteria, and had higher externalizing factor scores than behaviourally conditioned and emotionally vulnerable gamblers. While there were no group differences

in lifetime number of legal charges, antisocial impulsivist gamblers had significantly more lifetime arrests than both behaviourally conditioned and emotionally vulnerable gamblers. While no differences were found in self-reported hyperactive-impulsive symptoms, antisocial impulsivist and emotionally vulnerable gamblers had higher levels of self-reported inattentive symptoms and total ADHD scores than behaviourally conditioned gamblers.

Table 8

Comparing Subtypes on Antisocial and ADHD-Related Variables

	Behaviourally Conditioned (N = 65)	Emotionally Vulnerable (N = 55)	Antisocial Impulsivist (N = 30)	F and p values
Conduct disorder (past)	0.75 (1.57)	1.42 (1.56)	2.37 (2.70) ^a	$F(2, 147) = 7.97, p < 0.01$
Antisocial personality disorder (lifetime)	0.70 (1.34)	1.24 (1.61) ^b	2.30 (1.74) ^a	$F(2, 147) = 11.62, p < 0.01$
Externalizing factor score	-0.27 (0.82)	0.01 (0.93)	0.58 (0.98) ^a	$F(2, 147) = 9.45, p < 0.01$
Charges (lifetime)	2.17 (3.14)	1.98 (3.27)	4.43 (10.89)	$F(2, 147) = 2.11, p = 0.12$
Arrests (lifetime)	0.22 (0.72)	0.96 (3.08)	3.33 (4.89) ^a	$F(2, 147) = 11.97, p < 0.01$

CAARS	8.22 (4.62) ^c	10.40 (4.94)	11.57 (6.32)	$F(2, 147) =$
Inattentive				5.25, $p < 0.01$
CAARS	9.64 (4.92)	10.82 (5.12)	12.30 (4.45)	$F(2, 147) =$
Hyperactive- Impulsive				3.09, $p = 0.05$
CAARS ADHD	17.86 (8.60) ^c	21.22 (9.05)	23.87 (9.76)	$F(2, 147) =$
Total				5.02, $p < 0.01$

ADHD = Attention Deficit/Hyperactivity Disorder; CAARS = Conners' Adult ADHD Rating

Scale

^aAntisocial Impulsivist > Behaviourally Conditioned & Emotionally Vulnerable

^bEmotionally Vulnerable > Behaviourally Conditioned

^cBehaviourally Conditioned < Emotionally Vulnerable & Antisocial Impulsivist

General Personality Traits and Impulsivity Traits

Gambling subtypes were compared on 13 continuous personality and impulsivity variables, with a significance level alpha equal to 0.05 (see Table 10). There was an overall significant multivariate effect of personality and impulsivity variables by gambling subtype ($V = 1.04$, $F = 12.47$, $p < 0.01$). Behaviourally conditioned gamblers scored higher on MPQ Well-Being, and lower on MPQ Stress Reaction, MPQ Aggression, and MPQ Alienation than both emotionally vulnerable and antisocial impulsivist gamblers. Antisocial impulsivist gamblers scored significantly lower than both behaviourally conditioned and emotionally vulnerable gamblers on MPQ Harm Avoidance.

In regards to impulsivity (see Table 9 for bivariate correlations between impulsivity variables), antisocial impulsivist gamblers scored significantly higher on all dimensions of BIS impulsivity than both behaviourally conditioned and emotionally vulnerable gamblers. Behaviourally conditioned gamblers scored lower on UPPS Urgency than both emotionally vulnerable and antisocial impulsivist gamblers, and antisocial impulsivist gamblers scored higher on UPPS (Lack of) Perseverance than behaviourally conditioned gamblers. Furthermore, antisocial impulsivist gamblers scored higher on UPPS (Lack of) Planning than both behaviourally conditioned and emotionally vulnerable gamblers. Finally, antisocial impulsivist gamblers scored higher than emotionally vulnerable gamblers on UPPS Sensation Seeking (see Table 10).

Antisocial impulsivist gamblers had lower AUC values than behaviourally conditioned gamblers.

Childhood Maltreatment Variables

Gambling subtypes were compared on six continuous childhood maltreatment variables, with significance level alpha equal to 0.05 (see Table 11). There was no overall significant multivariate effect of childhood maltreatment variables by gambling subtype ($V = 0.11$, $F = 1.60$, $p = 0.11$). There were no group differences on self-reported Sexual Abuse, Emotional Abuse, and Emotional Neglect. Antisocial impulsivist gamblers had higher self-reported rates of Physical Abuse than behaviourally conditioned gamblers, higher self-reported rates of Physical Neglect than emotionally vulnerable gamblers, and higher rates of Total Abuse than behaviourally conditioned gamblers.

Table 9

Correlations between Impulsivity Variables (N = 150)

	MPQ CON	BIS Attentional	BIS Motor	BIS Non- Planning	UPPS Urgency	UPPS (Lack of) Perseverance	UPPS (Lack of) Planning	UPPS Sensation Seeking	Delayed Discounting
MPQ CON	1.00	-0.45*	-0.38*	-0.50*	-0.18*	-0.35*	-0.65*	-0.47*	0.20*
BIS Attentional	-	1.00	0.50*	0.57*	0.50*	0.44*	0.53*	0.04	-0.25*
BIS Motor	-	-	1.00	0.45*	0.36*	0.17*	0.39*	0.16	-0.30*
BIS Non- Planning	-	-	-	1.00	0.44*	0.48*	0.66*	-0.14	-0.33*
UPPS Urgency	-	-	-	-	1.00	0.34*	0.33*	-0.04	-0.26*
UPPS (Lack of) Perseverance	-	-	-	-	-	1.00	0.45*	-0.19*	-0.01
UPPS (Lack of) Planning	-	-	-	-	-	-	1.00	0.07	-0.28*

of) Planning

UPPS	-	-	-	-	-	-	-	-	1.00	0.08
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Sensation

Seeking

Delayed	-	-	-	-	-	-	-	-	-	-
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Discounting

BIS = Barratt Impulsiveness Scale; CON = Constraint; MPQ = Multidimensional Personality Questionnaire

*p < 0.05

Table 10

Subtype Comparisons on General Personality and Impulsivity Traits

	Behaviourally Conditioned (N = 65)	Emotionally Vulnerable (N = 55)	Antisocial Impulsivist (N = 30)	F and p values
MPQ Well-Being	16.74 (5.33) ^a	12.80 (6.50)	11.03 (7.16)	$F(2, 147) = 10.90, p < 0.01$
MPQ Stress Reaction	9.57 (6.18) ^b	15.93 (4.78)	16.90 (5.18)	$F(2, 147) = 27.59, p < 0.01$
MPQ Aggression	3.26 (2.55) ^b	8.35 (3.86)	9.87 (3.94)	$F(2, 147) = 53.18, p < 0.01$
MPQ Alienation	3.52 (2.98) ^b	10.89 (3.99)	11.80 (4.60)	$F(2, 147) = 78.94, p < 0.01$
MPQ Harm Avoidance	17.60 (5.56)	18.22 (3.77)	11.13 (4.01) ^c	$F(2, 147) = 25.30, p < 0.01$
BIS Attentional	16.62 (3.94)	18.05 (3.76)	20.67 (4.66) ^d	$F(2, 147) = 10.40, p < 0.01$
BIS Motor	24.38 (4.47)	25.58 (4.54)	28.07 (4.53) ^d	$F(2, 147) = 6.85, p < 0.01$
BIS Non-Planning	25.54 (5.60)	26.45 (5.01)	30.53 (5.22) ^d	$F(2, 147) = 9.35, p < 0.01$
UPPS Urgency	5.94 (3.67) ^b	9.13 (2.83)	9.90 (2.07)	$F(2, 147) =$

				23.50, $p < 0.01$
UPPS (Lack of)	2.65 (2.63) ^e	3.38 (2.72)	4.70 (3.02)	$F(2, 147) =$
Perseverance				5.77, $p < 0.01$
UPPS (Lack of)	3.62 (3.57)	2.51 (2.78)	5.9 (3.29) ^d	$F(2, 147) =$
Planning				10.60, $p < 0.01$
UPPS	6.92 (3.65)	6.69 (3.38) ^f	8.63 (3.10)	$F(2, 147) =$
Sensation-				3.40, $p = 0.04$
Seeking				
Delayed	0.30 (0.26) ^e	0.26 (0.25)	0.16 (0.25)	$F(2, 147) =$
Discounting				3.06, $p = 0.048$
(AUC)				

BIS = Barratt Impulsiveness Scale; MPQ = Multidimensional Personality Questionnaire

^aBehaviourally Conditioned > Emotionally Vulnerable & Antisocial Impulsivist

^bBehaviourally Conditioned < Emotionally Vulnerable & Antisocial Impulsivist

^cAntisocial Impulsivist < Behaviourally Conditioned & Emotionally Vulnerable

^dAntisocial Impulsivist > Behaviourally Conditioned & Emotionally Vulnerable

^eBehaviourally Conditioned > Antisocial Impulsivist

^fEmotionally Vulnerable < Antisocial Impulsivist

Table 11

Subtype Comparisons on Childhood Maltreatment Variables

Behaviourally Conditioned	Emotionally Vulnerable	Antisocial Impulsivist	F and p values
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	(N = 65)	(N = 55)	(N = 30)	
CTQ Physical Abuse	8.66 (4.41) ^a	9.27 (4.19)	11.50 (5.76)	$F(2, 147) = 3.92, p = 0.02$
CTQ Sexual Abuse	7.35 (5.08)	8.09 (5.58)	10.07 (7.50)	$F(2, 147) = 2.24, p = 0.11$
CTQ Emotional Abuse	10.63 (5.17)	12.07 (5.62)	12.93 (7.16)	$F(2, 147) = 1.90, p = 0.15$
CTQ Physical Neglect	7.97 (4.22)	7.47 (2.61) ^b	9.73 (4.65)	$F(2, 147) = 3.54, p = 0.03$
CTQ Emotional Neglect	10.89 (4.90)	11.31 (5.11)	12.47 (5.03)	$F(2, 147) = 1.02, p = 0.36$
CTQ Total	45.51 (18.47) ^a	48.22 (17.48)	56.70 (23.63)	$F(2, 147) = 3.50, p = 0.03$

CTQ = Childhood Trauma Questionnaire

^aBehaviourally Conditioned < Antisocial Impulsivist

^bEmotionally Vulnerable < Antisocial Impulsivist

CHAPTER V

DISCUSSION

Hypothesis 1: Factor Structure of Psychopathology

This is the first study to examine the factor structure underlying psychopathology in PPGs as it relates to the internalizing-externalizing model. In this regard, current findings are consistent with the internalizing-externalizing model in producing internalizing and externalizing dimensions of psychopathology. Current results advance understanding of the patterns of psychiatric comorbidity in disordered gamblers as well as the personality substrates that underlie the co-occurrence of internalizing and externalizing forms of psychopathology. Results can facilitate the improved assessment and treatment of comorbid psychopathology in PPGs.

The presence of an externalizing psychopathology factor in disordered gamblers is in line with previous literature on the frequent co-occurrence of SUDs, conduct disorder, and antisocial personality disorder in the general population (Armstrong & Costello, 2002; Waldman & Slutske, 2000). Externalizing disorders also occur frequently in PPGs and these conditions are often comorbid (e.g., Petry, Stinson, & Grant, 2005; Pietrzak & Petry, 2005). While some authors have hypothesized the comorbidity of externalizing disorders in gamblers can be explained by an underlying, externalizing dimension (Slutske et al., 1998, 2001, 2005), no previous research has directly examined this possibility. Current results confirm that a latent, externalizing dimension underlies SUDs and antisocial behaviour disorders in PPGs.

The externalizing factor accounts for most of the variance in psychopathology in the current sample. This suggests the comorbid psychopathology exhibited by gamblers is primarily externalizing in nature and its manifestation is at least partially explained by a latent externalizing dimension. The predominance of externalizing disorders in the current sample is consistent with research concluding that SUDs are the most common co-occurring conditions in PGs (Petry, Stinson, & Grant, 2005; Wareham & Potenza, 2010). Clinicians assessing PPGs are advised to comprehensively evaluate co-occurring externalizing conditions in these individuals, and should recognize the presence of one externalizing disorder strongly suggests the occurrence of another. Treatment of concurrent externalizing disorders in gamblers is critically important given the association between externalizing behaviours and elevated gambling severity (Ibanez et al., 2001), early onset gambling and gambling-related illegal activity (Pietrzak & Petry, 2006), suicide attempts (Ciarrocchi, 1987; Kausch, 2003), and increased impulsivity (McCormick et al., 1989). The management of externalizing disorders in gamblers would benefit from integrated treatments that are effective for and target *all* of these conditions (e.g., behavioural therapy), and should address externalizing disorders simultaneously given their underlying similarity.

In the current study, the externalizing dimension of psychopathology was negatively correlated with the personality trait constraint at a moderate level. In the general literature the association between SUDs, antisocial behaviour disorders, and impulsivity-related constructs such as constraint is strong (Howard, Kivlahan, & Walker, 1997; Krueger, Caspi, Moffitt, Silva, & McGee, 1996; McGue, Slutske, & Iacono, 1999; McGue, Slutske, Taylor, & Iacono, 1997; Patrick & Zempolich, 1998; Sher & Trull,

1994; Verona & Parker, 2000; Watson & Clark, 1993), as is the association between the externalizing dimension of psychopathology and impulsivity (Krueger, McGue, & Iacono, 2002). Furthermore, individuals with PG and concurrent SUDs and/or with antisocial behaviour disorders are often found to be highly impulsive (Blaszczynski, Steel, & McConaghy, 1997; Petry & Casarella, 1999). Slutske and colleagues (2000, 2001, 2005) have noted the overlap between PG, SUDs, and antisocial personality disorder may be explained in part by a latent externalizing factor associated with impulsivity. Based on current results it appears that impulsivity is the personality substrate for externalizing disorders in gamblers and partially explains the high rates of co-occurrence between these forms of psychopathology in gamblers. Future research on the etiology of externalizing disorders in disordered gamblers should examine various dimensions of impulsivity as factors that explain the development of concurrent externalizing conditions. Appropriately assessing and managing impulsivity in PGs is critical as impulsivity is associated with increased gambling severity (Loxton, Nguyen, Casey, & Dawe, 2008; Vitaro, Arsenault, & Tremblay, 1997) and non-response to treatment (Gonzalez-Ibanez, Mora, Gutierrez-Maldonado, Ariza, & Lourido-Ferreira, 2005). Clinicians should target maladaptive impulsivity both with behavioural and pharmacological treatments as a strategy to manage concurrent externalizing conditions in disordered gamblers. Focusing on reducing impulsivity as a treatment target may facilitate the integration of treatments for externalizing conditions in gamblers and allow management of these disorders to be concurrent rather than independent.

The presence of an internalizing factor in disordered gamblers, consisting of unipolar mood disorders and generalized anxiety disorder, is also consistent with most

previous research on the structure of psychiatric disorders in the general population (Krueger, 1999). Research documents the covariation of unipolar mood and anxiety disorders (Maser & Cloninger, 1990; Merikangas et al., 1996; Mineka, Watson, & Clark, 1998). While unipolar mood disorders occur at relatively elevated rates in disordered gamblers (Kim, Grant, Eckert, Faris, & Hartman, 2006; Petry, Stinson, & Grant, 2005), rates of anxiety disorders and the co-occurrence of unipolar mood and anxiety disorders in PPGs have not been extensively studied. Given the genetic overlap between PG and major depressive disorder, Potenza and colleagues (2005) noted a common, internalizing factor may explain the relationship between PG and internalizing disorders. The current study is the first to examine this possibility, and results suggest a latent, internalizing dimension underlies unipolar mood disorders and generalized anxiety disorder in PPGs.

The internalizing factor accounted for relatively less variance in psychopathology in disordered gamblers than the externalizing factor. Comorbid psychopathology in PPGs, while predominantly externalizing, is therefore also significantly internalizing in nature. This is line with previous research suggesting lifetime unipolar mood and anxiety disorders occur at lower rates than SUDs in PGs (e.g., Petry, Stinson, & Grant, 2005). Researchers studying comorbid psychopathology in gamblers are advised to continue to examine the prevalence of unipolar mood disorders and generalized anxiety disorder and the gambling-related correlates of these conditions. While clinicians are advised to focus more attention on evaluating co-occurring externalizing disorders in PPGs, internalizing conditions should also be adequately assessed. Clinicians should recognize that unipolar mood disorders and generalized anxiety disorder are significantly correlated in gamblers, and assess for generalized anxiety disorder when unipolar mood disorders are present and

vice versa. The treatment of co-occurring unipolar mood disorders in PGs is critical given the association between these conditions and increased gambling severity (Becona et al., 1995; Moodie & Finnigan, 2006), increased risk for uncontrolled gambling following treatment (Blaszczynski, McConaghy, & Frankova, 1991), and decreased rates of abstinence (Hodgins, Peden, & Cassidy, 2005). The management of co-occurring internalizing disorders in gamblers should involve treatments that are effective for *all* of these conditions (e.g., selective serotonin reuptake inhibitors, cognitive behavioural therapy), and clinicians should address internalizing disorders simultaneously given their underlying similarity.

Social anxiety disorder and posttraumatic stress disorder did not load heavily on the internalizing factor as predicted. Social anxiety disorder is a condition characterized by fears that are situation-specific, i.e., fears arising only when the individual is exposed to social situations. Individuals with social anxiety disorder, then, likely do not experience anxiety that is pervasive across time and situations, as do individuals with generalized anxiety disorder, for example. Social anxiety disorder likely represents a more fear-based condition, and thus may not load on an internalizing factor comprised of disorders (i.e., unipolar mood disorders, generalized anxiety disorder) that are not fear-based. Previous research suggests the anxiety disorders may not be a homogenous group of conditions, and that generalized anxiety disorder is more highly related to major depressive disorder than to other anxiety disorders (Kendler, Walters, Neale, Kessler, Heath, & Eaves, 1995). Furthermore, in the current sample, most individuals who met criteria for social anxiety disorder did so only because of excessive fears related to public speaking, suggesting they do not experience generalized anxiety across situations.

Previous research suggests posttraumatic stress disorder symptoms can be separated into fear, dysphoria, and anxious misery/distress-related factors (Forbes et al., 2011). Notably, the anxious misery/distress factor is related to unipolar depression and generalized anxiety disorder. Given that posttraumatic stress disorder symptoms can be represented by a three-factor structure, the *total* criteria count variable entered into the factor analysis in the current study may obscure the specific symptoms of the disorder that are related to the internalizing factor (which is similar to the anxious misery/distress factor of posttraumatic stress disorder symptoms). This may explain why posttraumatic stress disorder did not load highly on the internalizing factor in the current study.

The internalizing factor was positively correlated with negative emotionality at a moderate level. That is, the internalizing factor was associated with an increased tendency toward negative moods and emotions. In the general literature, the association between both unipolar mood and anxiety disorders and the negative emotionality personality construct is strong (Trull & Sher, 1994; Watson, Clark, & Carey, 1988; Widiger & Trull, 1992), as is the association between the internalizing dimension of psychopathology and negative emotionality (Fanous et al., 2002; Jang & Livesley, 1999; Kendler et al., 1993; Markon, Krueger, Bouchard, & Gottesman, 2002; Mineka, Watson, & Clark, 1998; Roberts & Kendler, 1999). PGs report elevated levels of neuroticism compared to controls (Blanco, Ibanez, Blanco-Jerez, Baca-Garcia, & Saiz-Ruiz, 2001; Blaszczynski, Buhrich, & McConaghy, 1985; Blaszczynski, Steel, & McConaghy, 1997; Blaszczynski, Wilson, & McConaghy, 1986; Graham & Lowenfeld, 1986; Roy et al., 1989). Current results confirm that negative emotionality is a personality substrate of unipolar mood

disorders and generalized anxiety disorder in PPGs and likely partially explains the co-occurrence of these conditions.

Researchers studying the etiology of internalizing disorders in PPGs should continue to examine factors that explain the development of the co-occurrence of these conditions (e.g., genetics factors related to negative emotionality, childhood maltreatment experiences related to increased negative emotionality). This will advance knowledge on the formation of comorbid internalizing conditions in gamblers. Clinicians should target increased negative emotionality both with behavioural and pharmacological treatments as a method to manage unipolar mood disorders and generalized anxiety disorder in gamblers. Focusing on negative emotionality as a treatment target may facilitate the integration of treatments for internalizing conditions and allow management of these conditions to be concurrent rather than sequential. Appropriately addressing negative emotionality in treatment is important given its association with PG severity (McCormick, 1993), treatment failure (Echeburua, Fernandez-Montalvo, & Baez, 2001), uncontrolled gambling following treatment (Blaszczynski, McConaghy, & Frankova, 1991), and earlier relapse following treatment (Daughters, Lejuez, Strong, Brown, Breen, & Lesieur, 2005).

Overall, current results confirm the internalizing-externalizing model of psychopathology has utility in conceptualizing the comorbid psychopathology that frequently occurs in PPGs. The model organizes various forms of psychopathology into externalizing and internalizing dimensions, can explain common personality factors that inform understanding of the etiology of these conditions, and may predict the effectiveness of treatments for externalizing disorders and for internalizing disorders.

Hypothesis 2: Subtypes of PPGs

The majority of gamblers in the current study were categorized as behaviourally conditioned, or low pathology, gamblers. In previous studies examining pathways model subtypes, the behaviourally conditioned gambler has variably been found to be the most (e.g., Stewart et al., 2008) or the least (Ledgerwood & Petry, 2010) common subtype. This difference between studies may result from dissimilarities in sample recruitment strategies. Behaviourally conditioned gamblers may be the most common subtype in samples of community-recruited gamblers, and the least common subtype in samples of treatment-seeking gamblers. It is possible that behaviourally conditioned gamblers are less represented in treatment samples because they experience less comorbid psychopathology and/or gambling-related difficulties, which may make them less distressed and less likely to seek treatment. The current sample also included some problem gamblers who present as less severe and, therefore, may be more likely to be categorized as behaviourally conditioned.

Behaviourally conditioned gamblers did not differ from emotionally vulnerable and antisocial impulsivist gamblers in age, gender, or recruitment source. Therefore, any differences between gambling subtypes are unlikely to be explained by demographic factors. A previous study found that gambling subtypes differed by gender (Ledgerwood & Petry, 2010), while another study did not (Vachon & Bagby, 2009). Female PGs who seek treatment may experience more internalizing symptoms, and as a consequence may sometimes be more likely to be categorized as emotionally vulnerable gamblers than female gamblers in the community. Behaviourally conditioned gamblers self-reported higher levels of formal education than the other two subtypes; this is consistent with

previous research on the pathways model (Vachon & Bagby, 2009; Ledgerwood & Petry, 2010). Reduced levels of impulsivity in behaviourally conditioned gamblers relative to emotionally vulnerable and antisocial impulsivist gamblers is one possible explanation of this relationship, given that increased impulsivity has been associated with decreased educational attainment (Fink & McCown, 1993). Furthermore, differences in how studies have categorized gambling subtypes may explain discrepant findings across studies.

As predicted by the pathways model, behaviourally conditioned gamblers demonstrated less severe gambling relative to the other subtypes. Decreased gambling severity in behaviourally conditioned gamblers has been seen in previous community and treatment-seeking gambling samples (e.g., Vachon & Bagby, 2009). Behaviourally conditioned gamblers were less likely to report gambling to reduce or avoid negative emotions than emotionally vulnerable and antisocial impulsivist gamblers, but did not self-report a decreased likelihood to gamble to increase positive emotions as predicted. No previous studies on pathways subtypes have examined motives for gambling, and current results reveal behaviourally conditioned gamblers do not use gambling as a strategy to regulate negative emotions. Using gambling to regulate negative feelings has been associated with increased gambling problems (Stewart et al., 2008), which further confirms that behaviourally conditioned gamblers have less severe gambling problems.

In regards to concurrent psychiatric and substance use disorders, behaviourally conditioned gamblers demonstrated significantly less externalizing psychopathology than antisocial impulsivist gamblers, less past major depressive disorder and lifetime posttraumatic stress disorder criteria than antisocial impulsivist gamblers, and fewer current dysthymic disorder criteria than emotionally vulnerable gamblers. Behaviourally conditioned gamblers, therefore, have relatively less comorbid internalizing and

externalizing psychopathology. This confirms this subtype should be considered a ‘low pathology’ group of gamblers that is neither prone to the internalization or externalization of distress. Previous studies that generated a gambling subtype similar to the behaviourally conditioned gambler have concluded these gamblers have reduced levels of psychopathology (see Milosevic & Ledgerwood, 2010 for a review). Mental disorders play a less significant role in the etiology, assessment, and management of behaviourally conditioned gamblers, and future research should investigate how non-psychopathological factors are related to gambling difficulties and treatment responsiveness in these gamblers.

Behaviourally conditioned gamblers are less impulsive on many dimensions of impulsivity than the other gambling subtypes. Decreased impulsivity may suggest behaviourally conditioned gamblers will be more responsive to PG treatment, given the association between elevated impulsivity and non-responsiveness to treatment (Gonzalez-Ibanez, Mora, Gutierrez-Maldonado, Ariza, & Lourido-Ferreira, 2005). Much of the previous literature on impulsivity in gamblers has not taken into account the possible existence of gambling subtypes; current results suggest the importance of examining impulsivity in gamblers by subtype. For example, the lack of an association between sensation seeking and PG in previous research, despite the belief that gamblers are characteristically sensation seeking, was resolved in the current study by examining subtypes differences.

Overall, characteristics of behaviourally conditioned gamblers in the current study are generally consistent with the description of the behaviourally conditioned gambler of the pathways model. Current results provide additional support for the validity of a BC gambler (see Milosevic & Ledgerwood, 2010, for a review). Factors reported to play a

role in development of PG in the general gambling literature, such as impulsivity, negative emotionality, ADHD, and antisocial behaviour disorders are relatively less relevant to behaviourally conditioned gamblers. Future etiological research should focus on how cognitive factors (e.g., gambling beliefs) are related to the development and maintenance of gambling disorders in these individuals. In addition, clinicians should tailor treatments to address the distorted thoughts and beliefs these gamblers have about gambling, and focus less attention on managing concurrent impulsivity or psychiatric disorders in an effort to reduce gambling problems.

The second largest subtype in the current study was the emotionally vulnerable, or internalizing, gambler. Previous studies using both community and treatment-seeking samples have found the same result (Ledgerwood & Petry, 2010; Stewart et al., 2008). While these gamblers demonstrated more severe gambling relative to behaviourally conditioned gamblers as expected, they did not have less severe gambling than antisocial impulsivist gamblers as predicted. Behaviourally conditioned and antisocial impulsivist gamblers have been shown to have similar levels of gambling severity in another study using community gamblers (Vachon & Bagby, 2009). However, emotionally vulnerable gamblers had less severe gambling (using one gambling measure) than the antisocial impulsivist in a previous treatment-seeking sample (Ledgerwood & Petry, 2010). Gambling severity differences by subtype across studies may be explained by sample recruitment source or by the method used to classify gamblers.

Emotionally vulnerable gamblers reported using gambling to cope with negative emotions more frequently than behaviourally conditioned gamblers, and their gambling motives were no different than antisocial impulsivist gamblers. Regulating negative emotions through excessive gambling is consistent with the pathways model description

of emotionally vulnerable gamblers; the current study is the first to support this hypothesis. Gambling behaviour motivated by a desire to reduce or avoid negative internal states may contribute to dependence on the activity (Stewart et al., 2008), and may partially explain the etiology and maintenance of PG in emotionally vulnerable gamblers.

As predicted, emotionally vulnerable gamblers met fewer lifetime criteria for alcohol abuse and dependence compared to antisocial impulsivist gamblers. Guided by the internalizing-externalizing and pathways models, the psychopathology in these individuals was expected to be primarily internalizing in nature. Given the association between comorbid alcohol-related disorders and gambling relapse after recent quitting (Hodgins & el-Guebaly, 2010), emotionally vulnerable gamblers may be able to sustain gambling abstinence for longer than antisocial impulsivist gamblers. While major depressive disorder and anxiety disorder criteria were not relatively increased in emotionally vulnerable gamblers, dysthymic disorder criteria were elevated in these gamblers relative to behaviourally conditioned gamblers. This suggests that only one form of internalizing psychopathology, in the absence of externalizing conditions, characterizes emotionally vulnerable gamblers. The relatively low rates of anxiety disorders in the current sample may have prevented differences between the gambling subtypes to emerge in statistical analyses. It is possible, however, that internalizing forms of psychopathology may not be uniquely relevant to the etiology and maintenance of their disordered gambling.

Emotionally vulnerable gamblers reported relatively lower levels of many dimensions of impulsivity, further suggesting they might have an increased ability to remain abstinent from problematic gambling following treatment. Furthermore,

impulsivity likely plays less of a role in the development of emotionally vulnerable gamblers' disordered gambling. Finally, emotionally vulnerable gamblers were not distinguished from other gambling subtypes based on childhood maltreatment experiences. While it has been suggested that these gamblers have greater histories of childhood adversity (Blaszczynski & Nower, 2002; McCormick, 1993), current results reveal abuse and neglect may not play a uniquely important role in the etiology of PG for emotionally vulnerable gamblers as predicted. The origins of their tendency to use gambling to relieve negative emotions may not necessarily lie in experiences of childhood adversity. Rates of childhood maltreatment were high in all three gambling subtypes relative to the general population (see Petry & Steinberg, 2005), which may have prevented differences between groups from emerging.

Overall, characteristics of emotionally vulnerable gamblers in the current study are consistent in some ways and inconsistent in other ways with the description of the prototypical emotionally vulnerable gambler of the pathways model. Notably, these gamblers do not present with more internalizing disorder symptoms than behaviourally conditioned gamblers. Previous studies of the pathways model have also discovered inconsistencies in the emotionally vulnerable subtype relative to the model (e.g., Ledgerwood & Petry, 2010; Lesieur, 2001), which suggests this type of gambler may not look exactly as Blaszczynski and Nower (2002) envisioned it.

Antisocial impulsivist, or externalizing, gamblers were the smallest group of gamblers in the current study. In a previous study of treatment-seeking gamblers, antisocial impulsivist gamblers were the most highly represented group (Ledgerwood & Petry, 2010) which preliminarily suggests these gamblers are more prevalent in treatment samples than in community samples. The prominence of antisocial impulsivist gamblers

in treatment may result from a number of factors, including their elevated gambling severity, impulsivity and impulsivity-related difficulties, SUDs, or a combination of these factors. In regards to gambling severity, antisocial impulsivist gamblers demonstrated more severe gambling than behaviourally conditioned gamblers but not emotionally vulnerable gamblers. In previous research on the pathways model, antisocial impulsivist gamblers were found to have equivalent levels of gambling severity to behaviourally conditioned and emotionally vulnerable gamblers in a sample of community gamblers (Vachon & Bagby, 2009) but elevated gambling severity relative to emotionally vulnerable gamblers in a sample of PGs in treatment (Ledgerwood & Petry, 2010). Inconsistencies in gambling severity across these studies and the current study may be explained by sample recruitment sources.

As expected, antisocial impulsivist gamblers reported an earlier onset of regular gambling than the other gambling subtypes. Onset of regular gambling was used as a proxy measure of onset of gambling-related difficulties in the current study. This result is unique within the gambling subtyping literature. Antisocial impulsivist gamblers may develop gambling-related difficulties at an earlier age due to their elevated impulsivity. Antisocial impulsivist gamblers did not self-report increased gambling to enhance positive emotions as predicted. That is, these gamblers were not characteristically prone to gamble because of the increase of positive feelings associated with gambling experiences. Given that these gamblers self-reported various motivations for gambling, the etiology and maintenance of their disordered gambling may not be informed by their reasons for gambling.

Relative to the other gambling subtypes, antisocial impulsivist gamblers presented with an increased level of substance-related disorders. Given their SUD comorbidities,

antisocial impulsivist may have a decreased response to traditional PG treatment and likely will require treatment that concurrently addresses their alcohol and drug problems. As predicted, antisocial impulsivist gamblers met more criteria for conduct disorder and antisocial personality disorder, and had more lifetime arrests than the other gambling subtypes. These findings are consistent with previous research on antisocial personality disorder in PG which finds that PGs with antisocial personality disorder have elevated gambling-related illegal activities (Pietrzak & Petry, 2006). Given their co-occurring SUDs and antisocial personality disorder, and increased ADHD symptoms, the gambling of antisocial impulsivist gamblers may be a function of their general tendency toward externalizing behaviours. In line with this conclusion, antisocial impulsivist gamblers were more impulsive than the other gambling subtypes, supporting the view that antisocial impulsivist gamblers can be conceptualized as externalizing gamblers. This is consistent with previous research on PGs with antisocial personality disorder, which suggests they experience elevated impulsivity (Pietrzak & Petry, 2006). Given their elevated impulsivity, antisocial impulsivist gamblers will likely experience more difficulties in PG treatment and their impulsivity needs to be adequately managed.

Antisocial impulsivist gamblers met more lifetime major depressive disorder criteria than behaviourally conditioned and emotionally vulnerable gamblers and more lifetime posttraumatic stress disorder criteria than behaviourally conditioned gamblers. Research on antisocial personality disorder and impulsivity in PGs suggests that antisocial and impulsive gamblers report higher levels of psychological distress (Blaszczynski et al., 1997; Blaszczynski & Steel, 1998; Pietrzak & Petry, 2006), and specifically increased depression and anxiety (Pietrzak & Petry, 2006; Steel & Blaszczynski, 1998). Current findings regarding increased internalizing psychopathology

in antisocial impulsivist gamblers may relate to the manner in which gamblers were categorized. That is, antisocial impulsivist gamblers were classified by increased levels of negative emotionality (in combination with increased impulsivity). Given the association between negative emotionality and major depressive disorder and posttraumatic stress disorder, current results may deviate from expectations of the pathways model as a function of the classification strategy used.

Overall, characteristics of antisocial impulsivist gamblers in the current study are generally consistent with the description of the antisocial impulsivist gambler of the pathways model. Results provide additional empirical support for the validity of the antisocial impulsivist gambler (see Milosevic & Ledgerwood, 2010, for a review). Impulsivity and a tendency toward the externalization of distress are factors likely relevant to the etiology of gambling problems in antisocial impulsivist gamblers. These factors may relate to early onset gambling difficulties and more severe gambling in these individuals.

Current results are generally in line with expectations of the pathways model (Blaszczynski & Nower, 2002). Behaviourally conditioned and antisocial impulsivist gamblers appear to be distinguishable from one another based on psychopathological, personality, gambling and other psychosocial variables to a greater degree than emotionally vulnerable gamblers are distinguishable from the other subtypes. Therefore, the validity of the behaviourally conditioned and antisocial impulsivist subtypes may be stronger than the validity of the emotionally vulnerable gambler subtype. Furthermore, from the perspective of the internalizing-externalizing model, current results support the existence of a low pathology gambling subtype and an externalizing gambling subtype which are relatively distinct from each other. A primarily internalizing subtype was not

supported by the current findings. While the underlying *structure* of psychopathology consists of externalizing and internalizing dimensions associated with the personality traits of impulsivity and negative emotionality, the classification of *individuals* with PPG by personality traits supports only an externalizing-impulsive group and not an internalizing group. These findings highlight the predominance of externalizing disorders and traits in PGs, and suggest the internalizing-externalizing model of psychopathology may provide a framework for understanding at least one type of gambler (i.e., the antisocial impulsivist gambler).

Limitations of Current Study

There are several limitations in the current study.

Sample. The total size of the current sample is relatively small. When conducting a factor analytic procedure, small samples can present problems related to several forms of sampling error. This may have limited the extent to which current data are representative of the larger population of gamblers, and may have generated factors that cannot be replicated. Nonetheless, some authors (Tabachnick & Fidell, 2001) suggest a sample size of 150 is the minimum acceptable size when factor analytic methods are employed. Future research examining the factor structure of psychopathology in PPGs should employ larger sample sizes. Furthermore, the small sample size limited the number of participants classified into each disordered gambling subtype. This is most evident in that only 30 participants were classified as antisocial impulsivist, or externalizing, gamblers. Small sample size may have limited the ability to detect additional group differences between gambling subtypes.

Cross-sectional design. All but one of the existing studies on subtyping PGs (Ledgerwood & Petry, 2010), including the present study, employed a cross-sectional research design (e.g., Ledgerwood & Petry, 2006; Steel & Blaszczynski, 1996; Stewart et al., 2008; Zimmerman et al., 1986). This research design limits knowledge about the stability of gambling subtypes over time, and limits our ability to evaluate whether elements related to these subtypes have a role in the development of PG. In addition, the ability to predict various gambling-related outcomes based on subtype (e.g., the antisocial impulsivist subtype predicting the course of disordered gambling) is compromised by this research design. Future research should employ longitudinal designs that measure psychopathology, personality, and PG across time to understand the degree of temporal consistency of the subtypes and how factors interact to predict subtype outcomes.

Measures. The majority of measures employed in the current study, both the interviews and questionnaires, rely heavily on participant self-report. Self-report results may be limited by personal biases, over- or under-reporting of psychological difficulties, lack of insight into one's behaviour, and difficulties accurately remembering historical information. Accuracy of data, therefore, may be affected by self-report. While self-report methods are difficult to avoid, future research can supplement self-report data with clinical file information, reports by individuals who know participants (e.g., family members), and biological and behavioural measures.

Data. Some of the data, particularly the psychiatric disorder criteria, had extremely non-normal distributions. Using data with non-normal distributions violated assumptions of statistical analyses and subsequently made these analyses less robust. Attempts were made to normalize data but multiple strategies were unsuccessful. Results

of the current study, particularly results involving mental disorder criteria, should be interpreted with caution. Using self-report questionnaires of psychological symptoms with Likert scales will likely produce data with normal distributions; future researchers should use both diagnostic interview and self-report questionnaire data when assessing psychopathology in gambling subtypes.

Pathways model. Some relevant aspects of the pathways model were not measured in the current study. Particularly, beliefs about gambling, gambling expectancies, and gambling learning experiences, which are reported by Blaszczynski and Nower (2002) to have etiological significance for all subtypes of disordered gamblers, were excluded. The lack of data on beliefs and cognitions limits understanding of how gambling subtypes may differ in these ways. Researchers can build upon the current study by including measures that assess these constructs.

Strengths of Current Study

Despite the aforementioned limitations, the current study has a number of strengths.

Sample. The current sample consists of an equal number of male and female PPGs, who range in age from 18 to 80 years old. Most research on psychopathology in disordered gamblers and on gambling subtypes has relied on predominantly male and primarily younger samples (e.g., Bonnaire et al., 2009; Stewart et al., 2008). This may limit the external validity of results. In addition, the current sample was recruited from multiple sources in the community, and consisted only of community-recruited gamblers. Much of the previous literature on gambling subtypes has utilized treatment-seeking

samples (e.g., Gonzalez-Ibanez et al., 2003; Ledgerwood & Petry, 2006). The generalizability of current findings to the general population of individuals with gambling disorders is strengthened given the diverse nature of the sample. Furthermore, current findings build upon the emerging subtyping research that has used community-based samples with relatively equivalent numbers of male and female gamblers (e.g., Turner et al., 2008; Vachon & Bagby, 2009).

Measures. Psychopathology was measured using a structured diagnostic interview, which is generally considered the most valid method of assessing mental disorders. Only two previous studies on gambling subtypes (Ledgerwood & Petry, 2010; Vachon & Bagby, 2009) have employed a diagnostic interview to determine psychiatric disorders in PGs. Questionnaires of psychiatric symptoms rely on participant self-report and may result in misinterpretation of items and/or inappropriate endorsement of mental disorder criteria. Given the potential bias inherent in self-report, the use of clinician-derived diagnoses increases the validity of the current findings.

Impulsivity was conceptualized as a multi-dimensional construct, and measured using both self-report and behavioural measures. The importance of multidimensional and multi-assessment measurement of impulsivity in disordered gamblers has been highlighted by previous researchers (e.g., Ledgerwood et al., 2009). The current study is in line with recommendations on the measurement of impulsivity by previous authors. Furthermore, this was the first study on gambling subtypes to employ a behavioural measure of impulsivity. This allowed for the most thorough and detailed understanding of the multiple dimensions of impulsivity in gambling subtypes.

Internalizing-externalizing model. As noted, this is the first study to empirically explore the utility of the internalizing-externalizing model of psychopathology in disordered gamblers. Given the lack of a model that conceptualizes the relationships between mental disorders in PPGs, the internalizing-externalizing model can improve our understanding of the patterns of comorbid psychopathology in gamblers by providing a useful heuristic for organizing findings. Current findings can further knowledge on the etiology, assessment, and treatment of comorbid forms of psychopathology in PPGs.

Pathways model. Variables such as gambling motivations, ADHD, and childhood maltreatment, while explicitly stated to be relevant to the pathways model, have not been measured in previous subtyping studies (e.g., Turner et al., 2008; Vachon & Bagby, 2009). Given the suggestion by Milosevic and Ledgerwood (2010) that the pathways model be used as a guiding model in studies on gambling subtypes, the current study provides additional evidence to support the model.

Future Directions

Given the elevated prevalence rates of psychiatric and substance-related conditions in PPGs, as well as our limited understanding on how co-occurring mental disorders are related to the etiology, maintenance, and treatment of PG, future research should continue to study the relationship between mental disorders and disordered gambling status. To advance our knowledge of the role of psychopathology in gamblers, the internalizing-externalizing model can serve as a useful heuristic in organizing disparate forms of mental disorders into simple dimensions comprised of disorder symptoms that cohere together. The specific relationships between externalizing disorders

with each other, and internalizing disorders with each other, is evident in the general population and, based on current results, in individuals with gambling disorders. Future work examining the factor structure of psychopathology in gamblers may test the validity of competing models (e.g., two-, three-, or four-factor models) using confirmatory statistical methods to confirm the validity of the internalizing-externalizing model over other possible models. In addition, research should explore the relationship between psychopathology factors and gambling variables to understand how the factors may relate to the development, maintenance, and treatment of disordered gambling. The factor structure of psychopathology in treatment-seeking gamblers should be explored to determine if a similar structure applies in these gamblers. Finally, comparing the structure of mental disorders in female versus male PPGs may reveal unique underlying factors explaining manifest psychopathology by gender.

Blaszczynski and Nower (2002) present a comprehensive theoretical model of gambling subtypes that appears to capture the distinct types of gamblers consistently reported by most investigators (see Milosevic & Ledgerwood, 2010, for a review). While evidence is beginning to emerge to validate aspects of the pathways model (e.g., Ledgerwood & Petry, 2006; Stewart et al., 2008; Turner et al., 2008; Vachon & Bagby, 2009), no empirical work has directly validated the *complete* model. Doing so would take into full account the various psychopathological, personality, motivational, and etiological variables explicitly specified by Blaszczynski and Nower. Given that the behaviourally conditioned, emotionally vulnerable, and antisocial impulsivist gambling subtypes presented in the pathways model appear to be consistent with many published subtyping studies, the pathways model may be adopted as a conceptual framework upon which further theoretical and empirical investigation on gambling subtypes is grounded. It

is suggested, however, that future work on PG subtypes explicitly and consistently operationalize all aspects of Blaszczynski and Nower's theory. Studies that examine the order of onset of PG in relation to psychopathology and maladaptive personality traits should be conducted. This will allow for validation of the pre-morbid vulnerabilities in each subtype of pathological gambler. Despite the appeal of this proposed subtyping scheme, it does not seem to have been routinely adopted for classifying gamblers in clinical practice (Stewart et al., 2008). Future research should investigate the differential association between gambling subtypes and types of treatment and treatment outcomes.

Blaszczynski and Nower (2002) note the importance of identifying clinically distinct subtypes of gamblers that exhibit similar phenomenological features but, at the same time, are distinct with respect to key variables that are of etiological relevance and that determine approaches to management and prognosis of the disorder. Advances in the understanding and treatment of disordered gambling are dependent on the development of a comprehensive explanatory model of gambling, which integrates knowledge from theory, research, and practice (Shaffer & Gambino, 1989). Furthermore, given that the etiology and pathophysiology of PG is not fully known, subtyping gamblers may prove productive as it can reduce the complexity of the phenomenon, facilitate the discovery of causal mechanisms, generate treatment measures, and develop alternative approaches to prevention. The natural course, morbidity, and prognosis of the disorder may vary by subtype. Gambling subtypes may be differentiated by biological variables associated with them. Finally, treatment may vary in effectiveness among subtypes, and treatment techniques may be developed that appropriately address individual differences in clinical presentation.

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APPENDIX 1

Comparison by Gender

	Male (N = 75)	Female (N = 75)	F and <i>p</i> values
Age (years)	33.67 (14.19)	38.91 (16.31)	F (1, 149) = 4.41, <i>p</i> = 0.04
Education (years)	13.87 (2.47)	13.83 (2.77)	F (1, 149) = 0.01, <i>p</i> = 0.91
NODS past year	5.95 (2.57)	6.12 (2.76)	F (1, 149) = 0.16, <i>p</i> = 0.69
NODS lifetime	6.61 (2.54)	5.87 (3.12)	F (1, 149) = 2.59, <i>p</i> = 0.11
NODS highest	7.36 (1.98)	7.47 (1.76)	F (1, 149) = 0.12 <i>p</i> = 0.73
CPGI-SF	9.72 (6.34)	10.52 (6.37)	F (1, 149) = 0.60, <i>p</i> = 0.44
GMQ Coping	10.25 (3.95)	12.55 (4.45)	F (1, 149) = 11.16, <i>p</i> < 0.01
GMQ Enhancement	15.20 (3.58)	13.68 (4.17)	F (1, 149) = 5.74, <i>p</i> = 0.02
GMQ Social	10.79 (3.43)	9.93 (3.57)	F (1, 149) = 2.23, <i>p</i> = 0.14

Appendix 1 (continued)

Age first gambling (years)	16.59 (6.20)	20.93 (9.24)	F (1, 149) = 8.74, $p < 0.01$
Age first regular gambling (years)	22.05 (9.90)	27.47 (14.09)	F (1, 149) = 7.41 $p < 0.01$
Maximum past year, one day spending (dollars)	1269.53 (2930.17)	461.96 (623.10)	F (1, 149) = 5.45, $p = 0.02$
Gambling activities (past year)	7.69 (3.47)	5.28 (2.76)	F (1, 149) = 22.18, $p < 0.01$
Alcohol abuse (lifetime)	1.56 (1.39)	0.89 (1.18)	F (1, 149) = 10.04, $p < 0.01$
Alcohol dependence (lifetime)	2.52 (2.32)	2.08 (2.54)	F (1, 149) = 1.23, $p = 0.27$
Cannabis abuse (lifetime)	0.80 (1.15)	0.55 (0.87)	F (1, 149) = 2.30, $p = 0.13$
Cannabis dependence (lifetime)	1.57 (2.19)	1.01 (1.74)	F (1, 149) = 2.99, $p = 0.09$

Appendix 1 (continued)

Drug classes used (lifetime)	2.56 (1.98)	2.43 (1.92)	F (1, 149) = 0.18, <i>p</i> = 0.68
Drug abuse disorders (lifetime)	0.92 (1.37)	0.71 (1.16)	F (1, 149) = 1.06, <i>p</i> = 0.31
Drug dependence disorders (lifetime)	0.72 (1.10)	0.61 (0.91)	F (1, 149) = 0.42, <i>p</i> = 0.52
Major depressive disorder (current)	0.67 (1.80)	1.57 (2.69)	F (1, 149) = 5.89, <i>p</i> = 0.02
Major depressive disorder (past)	3.17 (3.55)	4.53 (3.58)	F (1, 149) = 5.45, <i>p</i> = 0.02
Major depressive disorder episodes (lifetime)	2.61 (6.79)	6.99 (12.87)	F (1, 149) = 6.77, <i>p</i> = 0.01

Appendix 1 (continued)

Dysthymic disorder (current)	0.80 (2.10)	1.36 (2.57)	F (1, 149) = 2.13, <i>p</i> = 0.15
Social anxiety disorder (lifetime)	0.67 (1.44)	0.85 (1.55)	F (1, 149) = 0.59, <i>p</i> = 0.45
Posttraumatic stress disorder (lifetime)	1.96 (4.47)	5.08 (6.65)	F (1, 149) = 11.36, <i>p</i> < 0.01
Generalized anxiety disorder (current)	0.97 (2.46)	0.99 (2.51)	F (1, 149) = 0.01, <i>p</i> = 0.97
Psychiatric treatments (lifetime)	0.41 (0.68)	2.00 (2.07)	F (1, 149) = 39.90, <i>p</i> < 0.01
Conduct disorder (past)	1.44 (1.90)	1.20 (1.97)	F (1, 149) = 0.58, <i>p</i> = 0.45
Antisocial personality disorder (lifetime)	1.51 (1.70)	0.91 (1.52)	F (1, 149) = 5.22, <i>p</i> = 0.02
Charges (lifetime)	3.61 (7.62)	1.49 (2.06)	F (1, 149) = 5.41, <i>p</i> = 0.02

Appendix 1 (continued)

Arrests (lifetime)	1.39 (3.55)	0.84 (2.58)	F (1, 149) = 1.16, <i>p</i> = 0.28
CAARS Inattentive	9.53 (5.92)	9.84 (4.53)	F (1, 149) = 0.13, <i>p</i> = 0.72
CAARS Hyperactive- Impulsive	10.91 (5.29)	10.31 (4.65)	F (1, 149) = 0.54, <i>p</i> = 0.46
CAARS ADHD Total	20.44 (10.44)	20.15 (7.94)	F (1, 149) = 0.04, <i>p</i> = 0.85
MPQ Well-Being	15.48 (6.07)	12.83 (6.79)	F (1, 149) = 6.36, <i>p</i> = 0.01
MPQ Stress Reaction	11.13 (6.22)	15.60 (5.84)	F (1, 149) = 20.58, <i>p</i> < 0.01
MPQ Aggression	7.25 (4.29)	5.64 (4.38)	F (1, 149) = 5.19, <i>p</i> = 0.02
MPQ Alienation	7.65 (5.17)	8.11 (5.52)	F (1, 149) = 0.27, <i>p</i> = 0.60
MPQ Harm Avoidance	15.12 (5.18)	17.95 (5.23)	F (1, 149) = 11.04, <i>p</i> < 0.01
MPQ Positive Emotionality	42.97 (16.78)	35.52 (18.10)	F (1, 149) = 6.84, <i>p</i> = 0.01

Appendix 1 (continued)

MPQ Negative Emotionality	48.29 (19.22)	50.47 (21.19)	F (1, 149) = 0.44, $p = 0.51$
MPQ Constraint	47.29 (13.06)	50.75 (15.63)	F (1, 149) = 2.16, $p = 0.14$
BIS Attentional	17.44 (4.06)	18.47 (4.45)	F (1, 149) = 2.18, $p = 0.14$
BIS Motor	25.65 (4.98)	25.47 (4.39)	F (1, 149) = 0.06, $p = 0.81$
BIS Non-Planning	26.27 (5.21)	27.48 (5.94)	F (1, 149) = 1.77, $p = 0.19$
UPPS Urgency	7.55 (3.72)	8.25 (3.34)	F (1, 149) = 1.50, $p = 0.22$
UPPS (Lack of) Perseverance	3.07 (2.86)	3.59 (2.79)	F (1, 149) = 1.27, $p = 0.26$
UPPS (Lack of) Planning	3.52 (3.29)	3.81 (3.62)	F (1, 149) = 0.27, $p = 0.60$
UPPS Sensation- Seeking	8.49 (2.74)	5.87 (3.70)	F (1, 149) = 24.37, $p < 0.01$
Delayed Discounting (AUC)	0.28 (0.28)	0.24 (0.24)	F (1, 149) = 0.80, $p = 0.37$

Appendix 1 (continued)

CTQ Physical Abuse	8.61 (3.93)	10.29 (5.29)	F (1, 149) = 4.87, <i>p</i> = 0.03
CTQ Sexual Abuse	6.12 (2.44)	10.21 (7.39)	F (1, 149) = 20.73, <i>p</i> < 0.01
CTQ Emotional Abuse	9.12 (4.18)	14.12 (6.14)	F (1, 149) = 33.93, <i>p</i> < 0.01
CTQ Physical Neglect	7.20 (3.11)	9.08 (4.33)	F (1, 149) = 9.33, <i>p</i> < 0.01
CTQ Emotional Neglect	9.55 (4.13)	13.17 (5.16)	F (1, 149) = 22.56, <i>p</i> < 0.01
CTQ Total Abuse	40.60 (12.79)	56.88 (21.77)	F (1, 149) = 31.18, <i>p</i> < 0.01

ADHD = Attention-Deficit/Hyperactivity Disorder; BIS = Barratt Impulsiveness Scale; CAARS = Conners' Adult ADHD Rating Scale; CPGI-SF = Canadian Problem Gambling Index Short Form; CTQ = Childhood Trauma Questionnaire; GMQ = Gambling Motives Questionnaire; MPQ = Multidimensional Personality Questionnaire; NODS = National Opinion Research Centre DSM-IV Screen for Gambling Problems; UPPS = UPPS Impulsive Behaviour Scale

APPENDIX 2

Comparison by Recruitment Source

	Community (N = 91)	Student (N = 59)	F and <i>p</i> values
Age (years)	43.34 (15.27)	25.41(7.32)	F (1, 149) = 70.69, <i>p</i> < 0.01
Education (years)	12.73 (2.39)	15.58 (1.94)	F (1, 149) = 58.75, <i>p</i> < 0.01
NODS past year	6.79 (2.46)	4.86 (2.54)	F (1, 149) = 21.38, <i>p</i> < 0.01
NODS lifetime	6.98 (2.65)	5.10 (2.82)	F (1, 149) = 17.09, <i>p</i> < 0.01
NODS highest	7.96 (1.98)	6.58 (1.85)	F (1, 149) = 22.30, <i>p</i> < 0.01
CPGI-SF	11.69 (6.61)	7.69 (5.07)	F (1, 149) = 15.60, <i>p</i> < 0.01
GMQ Coping	12.51 (4.46)	9.69 (3.57)	F (1, 149) = 16.55, <i>p</i> < 0.01
GMQ Enhancement	14.76 (4.01)	13.95 (3.83)	F (1, 149) = 1.51, <i>p</i> = 0.22

Appendix 2 (continued)

GMQ Social	10.33 (3.67)	10.41 (3.30)	F (1, 149) = 0.02, <i>p</i> = 0.90
Age first gambling (years)	19.53 (9.44)	16.88 (4.99)	F (1, 149) = 3.92, <i>p</i> = 0.05
Age first regular gambling (years)	27.76 (14.68)	20.14 (5.20)	F (1, 149) = 14.69, <i>p</i> < 0.01
Maximum past year, one day spending (dollars)	837.82 (1680.33)	908.81 (2736.24)	F (1, 149) = 0.04, <i>p</i> = 0.84
Gambling activities (past year)	6.42 (3.32)	6.59 (3.43)	F (1, 149) = 0.10, <i>p</i> = 0.76
Alcohol abuse (lifetime)	1.31 (1.39)	1.10 (1.23)	F (1, 149) = 0.86, <i>p</i> = 0.36
Alcohol dependence (lifetime)	2.64 (2.62)	1.78 (2.02)	F (1, 149) = 4.56, <i>p</i> = 0.03
Cannabis abuse (lifetime)	0.73 (1.05)	0.59 (0.98)	F (1, 149) = 0.59, <i>p</i> = 0.44

Appendix 2 (continued)

Cannabis dependence (lifetime)	1.32 (1.93)	1.25 (2.11)	F (1, 149) = 0.04, $p = 0.85$
Drug classes used (lifetime)	2.86 (2.00)	1.93 (1.73)	F (1, 149) = 8.51, $p < 0.01$
Drug abuse disorders (lifetime)	0.95 (1.41)	0.61 (1.00)	F (1, 149) = 2.51, $p = 0.12$
Drug dependence disorders (lifetime)	0.80 (1.09)	0.46 (0.84)	F (1, 149) = 4.28, $p = 0.04$
Major depressive disorder (current)	1.32 (2.49)	0.81 (2.02)	F (1, 149) = 1.70, $p = 0.20$
Major depressive disorder (past)	4.22 (3.61)	3.29 (3.59)	F (1, 149) = 2.39, $p = 0.12$

Appendix 2 (continued)

Major depressive disorder episodes (lifetime)	5.64 (10.94)	3.51 (9.70)	F (1, 149) = 1.48, <i>p</i> = 0.23
Dysthymic disorder (current)	1.55 (2.66)	0.36 (1.55)	F (1, 149) = 9.70, <i>p</i> < 0.01
Social anxiety disorder (lifetime)	0.62 (1.27)	0.98 (1.77)	F (1, 149) = 2.19, <i>p</i> = 0.14
Posttraumatic stress disorder (lifetime)	4.10 (6.29)	2.63 (5.05)	F (1, 149) = 2.28, <i>p</i> = 0.13
Generalized anxiety disorder (current)	1.12 (2.62)	0.76 (2.25)	F (1, 149) = 0.75, <i>p</i> = 0.39
Psychiatric treatments (lifetime)	1.44 (1.92)	0.85 (1.32)	F (1, 149) = 4.30, <i>p</i> = 0.04
Conduct disorder (past)	1.37 (2.05)	1.24 (1.75)	F (1, 149) = 0.18, <i>p</i> = 0.67

Appendix 2 (continued)

Antisocial personality disorder (lifetime)	1.56 (1.73)	0.66 (1.29)	F (1, 149) = 11.66, $p < 0.01$
Charges (lifetime)	2.63 (3.55)	2.44 (7.92)	F (1, 149) = 0.04, $p = 0.85$
Arrests (lifetime)	1.53 (3.58)	0.47 (2.07)	F (1, 149) = 4.20, $p = 0.04$
CAARS Inattentive	9.42 (5.29)	10.10 (5.23)	F (1, 149) = 0.60, $p = 0.44$
CAARS Hyperactive- Impulsive	10.11 (5.48)	11.37 (4.00)	F (1, 149) = 2.33, $p = 0.13$
CAARS ADHD Total	19.53 (9.80)	21.47 (8.26)	F (1, 149) = 1.59, $p = 0.21$
MPQ Well-Being	12.93 (6.72)	16.03 (5.88)	F (1, 149) = 8.40, $p < 0.01$
MPQ Stress Reaction	13.62 (6.36)	12.98 (6.54)	F (1, 149) = 0.35, $p = 0.56$

Appendix 2 (continued)

MPQ Aggression	6.52 (4.49)	6.34 (4.28)	F (1, 149) = 0.06, <i>p</i> = 0.81
MPQ Alienation	9.23 (5.65)	5.80 (4.03)	F (1, 149) = 16.38, <i>p</i> < 0.01
MPQ Harm Avoidance	17.07 (4.82)	15.71 (6.10)	F (1, 149) = 2.29, <i>p</i> = 0.13
MPQ Positive Emotionality	35.11 (17.50)	45.63 (16.41)	F (1, 149) = 13.57, <i>p</i> < 0.01
MPQ Negative Emotionality	52.11 (21.45)	45.17 (17.30)	F (1, 149) = 4.34, <i>p</i> = 0.04
MPQ Constraint	49.32 (13.20)	48.56 (16.32)	F (1, 149) = 0.10, <i>p</i> = 0.76
BIS Attentional	18.23 (4.27)	17.53 (4.28)	F (1, 149) = 0.97, <i>p</i> = 0.33
BIS Motor	26.04 (4.77)	24.81 (4.67)	F (1, 149) = 2.50, <i>p</i> = 0.12
BIS Non-Planning	27.93 (5.60)	25.24 (5.24)	F (1, 149) = 8.73, <i>p</i> < 0.01
UPPS Urgency	8.51 (3.26)	6.97 (3.77)	F (1, 149) = 7.03, <i>p</i> < 0.01

Appendix 2 (continued)

UPPS (Lack of) Perseverance	3.62 (2.80)	2.88 (2.84)	F (1, 149) = 2.43, $p = 0.12$
UPPS (Lack of) Planning	3.96 (3.42)	3.22 (3.48)	F(1, 149) = 1.64, $p = 0.20$
UPPS Sensation- Seeking	6.49 (3.36)	8.24 (3.49)	F (1, 149) = 9.35, $p < 0.01$
Delayed Discounting (AUC)	0.24 (0.28)	0.29 (0.23)	F (1, 149) = 1.39, $p = 0.24$
CTQ Physical Abuse	10.03 (5.18)	8.56 (3.78)	F (1, 149) = 3.55, $p = 0.06$
CTQ Sexual Abuse	9.20 (6.56)	6.58 (4.14)	F (1, 149) = 7.48, $p < 0.01$
CTQ Emotional Abuse	12.49 (6.11)	10.27 (5.08)	F (1, 149) = 5.40, $p = 0.02$
CTQ Physical Neglect	8.96 (4.36)	6.88 (2.53)	F (1, 149) = 10.96, $p < 0.01$
CTQ Emotional Neglect	12.22 (4.86)	10.03 (4.97)	F (1, 149) = 7.11, $p < 0.01$

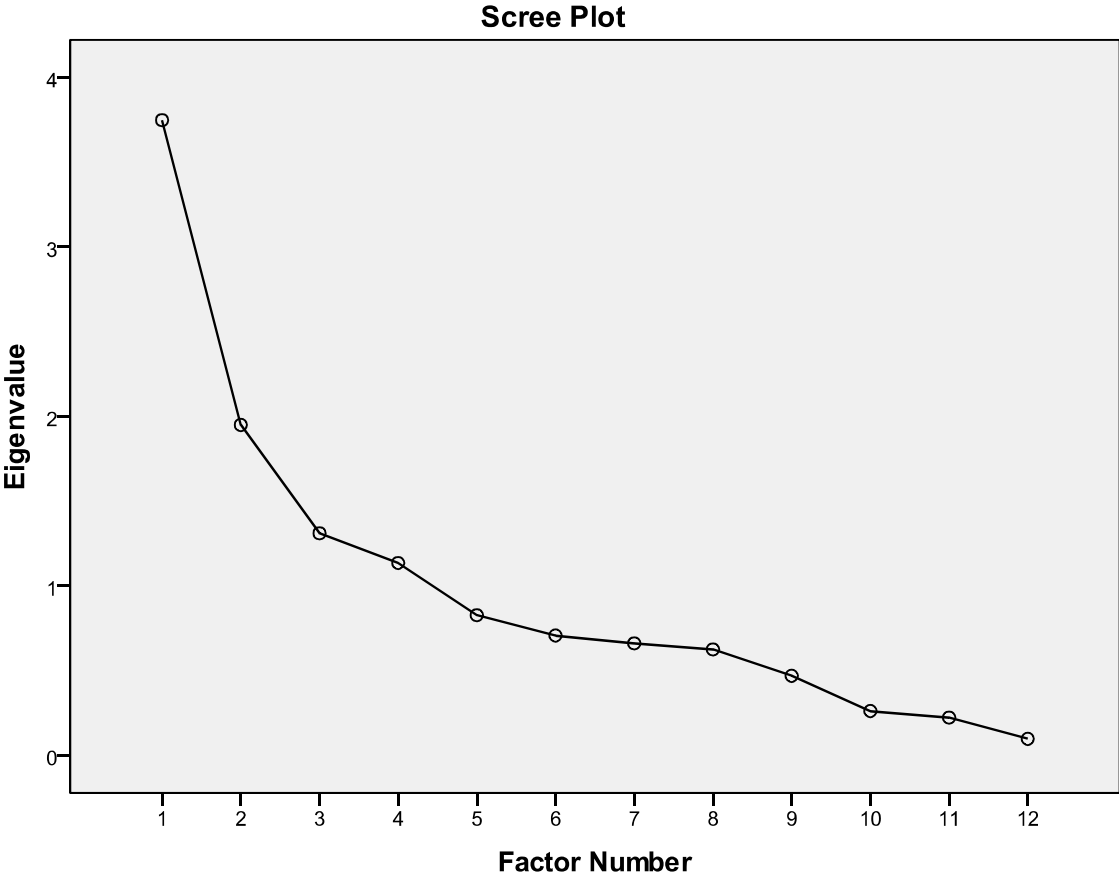
Appendix 2 (continued)

CTQ Total Abuse	52.90 (21.49)	42.32 (14.12)	F (1, 149) = 11.16, $p < 0.01$
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ADHD = Attention-Deficit/Hyperactivity Disorder; BIS = Barratt Impulsiveness Scale; CAARS = Conners' Adult ADHD Rating Scale; CPGI-SF = Canadian Problem Gambling Index Short Form; CTQ = Childhood Trauma Questionnaire; GMQ = Gambling Motives Questionnaire; MPQ = Multidimensional Personality Questionnaire; NODS = National Opinion Research Centre DSM-IV Screen for Gambling Problems; UPPS = UPPS Impulsive Behaviour Scale

APPENDIX 3

Scree Plot



APPENDIX 4

Pattern and Structure Matrices for Three-Factor Structure of Psychopathology

Pattern Matrix for Three-Factor Structure of Psychopathology

	Factor Loadings		
	1	2	3
Major depressive disorder (current)	0.110	0.679	-0.210
Major depressive disorder (past)	-0.040	0.556	0.120
Dysthymic disorder (current)	0.135	0.605	-0.090
Social anxiety disorder (lifetime)	-0.010	-0.010	0.187
Posttraumatic stress disorder (lifetime)	-0.110	0.364	0.295
Generalized anxiety disorder (current)	0.020	0.381	-0.020
Alcohol abuse (lifetime)	0.858	-0.030	-0.120
Alcohol dependence (lifetime)	0.774	0.160	-0.070
Cannabis abuse (lifetime)	0.572	-0.060	0.603

Appendix 4 (continued)

Cannabis dependence (lifetime)	0.521	-0.040	0.731
Conduct disorder (past)	0.427	-0.010	0.114
Antisocial personality disorder (lifetime)	0.811	0.090	-0.001
Eigenvalue	3.75	1.95	1.31
% of variance	31.23	16.24	10.91

APPENDIX 4 (continued)

Pattern and Structure Matrices for Three-Factor Structure of Psychopathology

Structure Matrix for Three-Factor Structure of Psychopathology

	Factor Loadings		
	1	2	3
Major depressive disorder (current)	0.187	0.685	-0.155
Major depressive disorder (past)	0.070	0.555	0.141
Dysthymic disorder (current)	0.224	0.623	-0.030
Social anxiety disorder (lifetime)	0.015	-0.002	0.184
Posttraumatic stress disorder (lifetime)	-0.001	0.362	0.298
Generalized anxiety disorder (current)	0.080	0.383	0.002
Alcohol abuse (lifetime)	0.834	0.112	0.020
Alcohol dependence (lifetime)	0.790	0.288	0.060
Cannabis abuse (lifetime)	0.659	0.070	0.692

Appendix 4 (continued)

Cannabis dependence (lifetime)	0.633	0.090	0.813
Conduct disorder (past)	0.443	0.065	0.182
Antisocial personality disorder (lifetime)	0.827	0.233	0.135
Eigenvalue	3.75	1.95	1.31
% of variance	31.23	16.24	10.91

APPENDIX 5

Pattern and Structure Matrices for Four-Factor Structure of Psychopathology

Pattern Matrix for Four-Factor Structure of Psychopathology

	Factor Loadings			
	1	2	3	4
Major depressive disorder (current)	0.140	0.663	-0.210	-0.050
Major depressive disorder (lifetime)	-0.050	0.552	0.090	0.030
Dysthymic disorder (current)	0.050	0.637	-0.102	-0.215
Social anxiety disorder (lifetime)	0.030	-0.010	0.214	0.188
Posttraumatic stress disorder (lifetime)	-0.172	0.369	0.252	0.008
Generalized anxiety disorder (current)	0.148	0.381	-0.010	0.225
Alcohol abuse (lifetime)	0.846	-0.050	0.098	-0.055
Alcohol dependence (lifetime)	0.840	0.140	0.111	0.042

Appendix 5 (continued)

Cannabis abuse (lifetime)	0.238	-0.030	0.745	-0.239
Cannabis dependence (lifetime)	0.186	-0.006	0.876	-0.179
Conduct disorder (past)	0.429	0.010	0.183	-0.389
Antisocial personality disorder (lifetime)	0.788	0.117	0.144	-0.377
Eigenvalue	3.75	1.95	1.31	1.13
% of variance	31.23	16.24	10.91	9.45

APPENDIX 5 (continued)

Pattern and Structure Matrices for Four-Factor Structure of Psychopathology

Structure Matrix for Four-Factor Structure of Psychopathology

	Factor Loadings			
	1	2	3	4
Major depressive disorder (current)	0.221	0.665	-0.109	-0.085
Major depressive disorder (lifetime)	0.061	0.554	0.136	0.025
Dysthymic disorder (current)	0.182	0.637	-0.015	-0.231
Social anxiety disorder (lifetime)	0.031	-0.009	0.212	0.175
Posttraumatic stress disorder (lifetime)	-0.056	0.367	0.254	0.033
Generalized anxiety disorder (current)	0.160	0.402	0.057	0.188
Alcohol abuse (lifetime)	0.871	0.103	0.281	-0.246
Alcohol dependence (lifetime)	0.879	0.294	0.310	-0.151

Appendix 5 (continued)

Cannabis abuse (lifetime)	0.451	0.099	0.802	-0.315
Cannabis dependence (lifetime)	0.417	0.123	0.922	-0.248
Conduct disorder (past)	0.431	0.055	0.222	-0.375
Antisocial personality disorder (lifetime)	0.694	0.228	0.288	-0.388
Eigenvalue	3.75	1.95	1.31	1.13
% of variance	31.23	16.24	10.91	9.45

APPENDIX 6

Community Advertisement



DO YOU GAMBLE FREQUENTLY?

Women and men 18 years or older who gamble frequently are invited to participate in a study exploring psychological factors associated with gambling.

Participation is voluntary and confidential.
Participants will be compensated up to \$45 for 2.5 to 3 hours of their time.

This study is being conducted by Aleks Milosevic, M.A., a clinical psychology doctoral student at the University of Windsor, and his research advisor, Dr. G. Ron Frisch, Ph.D. The research study has received ethics clearance from the University of Windsor Research Ethics Board.

Please call University of Windsor's Problem Gambling Research Group at (519) 253-3000 Ext. 3946, or email milosev@uwindsor.ca for further information about this study.

APPENDIX 7

Participant Pool Advertisement

Individuals who gamble frequently often have different emotional and behavioural symptoms, personality traits, and childhood experiences. The purpose of this study will be to test the idea that various types of gamblers exist which differ on psychological symptoms, personality traits, and childhood experiences. If you volunteer to participate in this study, we will ask you to do the following things. First, you will complete an interview about psychological symptoms and behaviours, and an interview about gambling behaviour. Second, you will complete several paper-and-pencil questionnaires about personality traits and childhood experiences. Lastly, you will complete a brief computer task that measures an aspect of impulse control.

APPENDIX 8

Community Population Consent Form



CONSENT TO PARTICIPATE IN RESEARCH

Title of Study: Personality, behaviour, and childhood experiences: A typology of gamblers (Community population)

You are asked to participate in a research study conducted by Aleksandar Milosevic, M.A. (Doctoral student in Clinical Psychology), under the advisorship of Dr. G. Ron Frisch, Ph.D. (Professor Emeritus) from the Department of Psychology at the University of Windsor. Results of the research study will contribute to Mr. Milosevic's doctoral dissertation. The research study is being sponsored by a fellowship from the Ontario Problem Gambling Research Centre (OPGRC).

If you have any questions or concerns about the research, please feel to contact Dr. G. Ron Frisch, Ph.D. at (519) 253-3000, ext. 3355.

PURPOSE OF THE STUDY

The purpose of the study is to understand the relationship between personality, behaviour, and childhood experiences in individuals who gamble frequently. We are investigating the possibility that different types of gamblers exist that can be differentiated based on personality traits, psychiatric symptoms, and childhood experiences. Individuals from the Windsor community who have gambled frequently are being asked to participate. We estimate that about 150 participants will be recruited at this site.

PROCEDURES

If you volunteer to participate in this study, we would ask you to do the following things: First, you will discuss and sign this informed consent form that describes the study. During the session that follows the consenting procedure we will ask you to participate in two interviews. The first interview will ask you questions about current and past emotional symptoms that you may have experienced. In addition, this interview will ask you questions about alcohol and drug use, risky behaviours, and treatments you have received. The second interview will ask you specific questions about your gambling

behaviour, including types of gambling engaged in as well as amount of money spent on gambling. Following completion of the interviews, you will complete several paper-and-pencil questionnaires about your personality traits, psychological and behavioural symptoms, and childhood traumatic experiences. The questionnaire about childhood traumatic experiences will ask you if you have experienced physical, sexual, and/or emotional abuse and/or physical and emotional neglect during your childhood. Finally, you will complete a brief computer-based task that measures an aspect of impulse control. The total time you will spend is approximately 2.5 to 3 hours on one day.

POTENTIAL RISKS AND DISCOMFORTS

By taking part in this study, you may experience the following risks:

Emotional risk: You may become uncomfortable answering questions about gambling, psychological problems, alcohol and drug use, and childhood traumatic experiences. If you become uncomfortable with any part of the interviews or questionnaires, you may skip the question or take a break.

Social risk: The information you provide may become available to people who are not involved in the research study. Every effort will be made to protect your confidentiality. Your research record will be labelled with a code number. A master key, which links your name and code number, will be maintained in a separate and secure location. You will not be identified in any presentation or publication based on the results of the research study.

The following information must be released to the appropriate authorities if at any time during the study there is concern that:

- Current child abuse or elder abuse has possibly occurred.
- You are deemed a threat to yourself or others.

Although we do not ask explicitly about the above information in questionnaires, it is possible that some of this information may be elicited by semi-structured interview questions.

POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

You may not benefit from participation in this study. In some cases, participants may benefit from a thorough assessment of their gambling and psychological difficulties. You will receive referrals for gambling and psychological treatment services in the Windsor community that you may contact should you desire to do so.

Studying types of gamblers can aid in the development of assessment and treatment strategies that address individual differences. If it is shown that types of gamblers differ on personality traits, psychiatric symptoms, and childhood traumatic experiences, assessment tools can be developed to differentiate gambling subtypes in order to allow

treatment providers to adequately address the unique psychological factors that underlie specific gamblers' gambling difficulties.

PAYMENT FOR PARTICIPATION

For taking part in this research study, you will be paid for your time and inconvenience. If you are not able to complete all of the testing, we will compensate you \$10 in Devonshire Mall money when you end the testing session. If you complete all of the testing, you will receive \$45 in Devonshire Mall money for your time at the end of the session.

CONFIDENTIALITY

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission. You will be identified in the research records by a code number. A master file that links your name to the code number will be locked in a file cabinet at the Problem Gambling Research Group. Your interview data and questionnaires will be locked in file cabinets, and will not be stored with any identifying information (e.g., name, phone number, social insurance number, etc.). The data will be retained for a period of 5 years, at which time it will be shredded.

When the results of this research are published or discussed in conferences, no information will be included that would reveal your identity.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may also refuse to answer any questions you do not want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so. This might occur if the investigator deems that you are experiencing a significant level of emotional distress that might interfere with completion of interviews and/or questionnaires.

FEEDBACK OF THE RESULTS OF THIS STUDY TO THE SUBJECTS

Research findings will be made available to study participants once the study is completed. Should you be interested in learning about the study findings, feel free to visit uwindsor.ca/reb for posted study results in mid-2010.

SUBSEQUENT USE OF DATA

The data will be used in subsequent studies. Once again, information that reveals your identity will not be released.

RIGHTS OF RESEARCH SUBJECTS

You may withdraw your consent at any time and discontinue participation without penalty. If you have questions regarding your rights as a research subject, contact: Research Ethics Coordinator, University of Windsor, Windsor, Ontario, N9B 3P4; Telephone: 519-253-3000, ext. 3948; e-mail: ethics@uwindsor.ca

SIGNATURE OF RESEARCH SUBJECT/LEGAL REPRESENTATIVE

I understand the information provided for the study *Personality, Behaviour, and Childhood Experiences: A Typology of Gamblers* as described herein. My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a copy of this form.

Name of Subject

Signature of Subject

Date

SIGNATURE OF INVESTIGATOR

These are the terms under which I will conduct research.

Signature of Investigator
Revised February 2008

Date

APPENDIX 9

Undergraduate Population Consent Form



CONSENT TO PARTICIPATE IN RESEARCH

Title of Study: Personality, behaviour, and childhood experiences: A typology of gamblers (Undergraduate population)

You are asked to participate in a research study conducted by Aleksandar Milosevic, M.A. (Doctoral student in Clinical Psychology), under the advisorship of Dr. G. Ron Frisch, Ph.D. (Professor Emeritus) from the Department of Psychology at the University of Windsor. Results of the research study will contribute to Mr. Milosevic's doctoral dissertation. The research study is being sponsored by a fellowship from the Ontario Problem Gambling Research Centre (OPGRC).

If you have any questions or concerns about the research, please feel to contact Dr. G. Ron Frisch, Ph.D. at (519) 253-3000, ext. 3355.

PURPOSE OF THE STUDY

The purpose of the study is to understand the relationship between personality, behaviour, and childhood experiences in individuals who gamble frequently. We are investigating the possibility that different types of gamblers exist that can be differentiated based on personality traits, psychiatric symptoms, and childhood experiences. Individuals from the Windsor community who have gambled frequently are being asked to participate. We estimate that about 150 participants will be recruited at this site.

PROCEDURES

If you volunteer to participate in this study, we would ask you to do the following things:

First, you will discuss and sign this informed consent form that describes the study. During the session that follows the consenting procedure we will ask you to participate in two interviews. The first interview will ask you questions about current and past emotional symptoms that you may have experienced. In addition, this interview will ask you questions about alcohol and drug use, risky behaviours, and treatments you have received. The second interview will ask you specific questions about your gambling

behaviour, including types of gambling engaged in as well as amount of money spent on gambling. Following completion of the interviews, you will complete several paper-and-pencil questionnaires about your personality traits, psychological and behavioural symptoms, and childhood traumatic experiences. The questionnaire about childhood traumatic experiences will ask you if you experienced physical, sexual, and/or emotional abuse and/or physical and emotional neglect during your childhood. Finally, you will complete a brief computer-based task that measures an aspect of impulse control. The total time you will spend is approximately 2.5 to 3 hours on one day.

POTENTIAL RISKS AND DISCOMFORTS

By taking part in this study, you may experience the following risks:

Emotional risk: You may become uncomfortable answering questions about gambling, psychological problems, alcohol and drug use, and childhood traumatic experiences. If you become uncomfortable with any part of the interviews or questionnaires, you may skip the question or take a break.

Social risk: The information you provide may become available to people who are not involved in the research. Every effort will be made to protect your confidentiality. Your research record will be labelled with a code number. A master key, which links your name and code number will be maintained in a separate and secure location. You will not be identified in any presentation or publication based on the results of the research study.

The following information must be released to the appropriate authorities if at any time during the study there is concern that:

Current child abuse or elder abuse has possibly occurred.
You are deemed a threat to yourself or others.

Although we do not ask explicitly about the above information in questionnaires, it is possible that some of this information may be elicited by semi-structured interview questions.

POTENTIAL BENEFITS TO SUBJECTS AND/OR TO SOCIETY

You may not benefit from participation in this study. In some cases, participants may benefit from a thorough assessment of their gambling and psychological difficulties. You will receive referrals for gambling and psychological treatment services in the Windsor community that you may contact should you desire to do so.

Studying types of gamblers can aid in the development of assessment and treatment strategies that address individual differences. If it is shown that types of gamblers differ on personality traits, psychiatric symptoms, and childhood traumatic experiences, assessment tools can be developed to differentiate gambling subtypes in order to allow treatment providers to adequately address the unique psychological factors that underlie specific gamblers' gambling difficulties.

PAYMENT FOR PARTICIPATION

There is no payment for participating in this research study. However, you may be eligible to receive three bonus points to be added to a University of Windsor undergraduate Psychology course mark in which you are currently registered if you complete all testing. If you are not able to complete all the testing, you may be eligible to receive one bonus point to be added to a University of Windsor undergraduate Psychology course mark in which you are currently registered.

CONFIDENTIALITY

Any information that is obtained in connection with this study and that can be identified with you will remain confidential and will be disclosed only with your permission. You will be identified in the research records by a code number. A master file that links your name to the code number will be locked in a file cabinet at the Problem Gambling Research Group. Your interview data and questionnaires will be locked in file cabinets, and will not be stored with any identifying information (e.g., name, phone number, social insurance number, etc.). The data will be retained for a period of 5 years, at which time it will be shredded.

When the results of this research are published or discussed in conferences, no information will be included that would reveal your identity.

PARTICIPATION AND WITHDRAWAL

You can choose whether to be in this study or not. If you volunteer to be in this study, you may withdraw at any time without consequences of any kind. You may also refuse to answer any questions you do not want to answer and still remain in the study. The investigator may withdraw you from this research if circumstances arise which warrant doing so. This might occur if the investigator deems that you are experiencing a significant level of emotional distress that might interfere with completion of interviews and/or questionnaires.

FEEDBACK OF THE RESULTS OF THIS STUDY TO THE SUBJECTS

Research findings will be made available to study participants once the study is completed. Should you be interested in learning about the study findings, feel free to visit uwindsor.ca/reb for posted study results in mid-2010.

SUBSEQUENT USE OF DATA

The data will be used in subsequent studies. Once again, information that reveals your identity will not be released.

RIGHTS OF RESEARCH SUBJECTS

You may withdraw your consent at any time and discontinue participation without penalty. If you have questions regarding your rights as a research subject, contact: Research Ethics Coordinator, University of Windsor, Windsor, Ontario, N9B 3P4; Telephone: 519-253-3000, ext. 3948; e-mail: ethics@uwindsor.ca

SIGNATURE OF RESEARCH SUBJECT/LEGAL REPRESENTATIVE

I understand the information provided for the study *Personality, Behaviour, and Childhood Experiences: A Typology of Gamblers* as described herein. My questions have been answered to my satisfaction, and I agree to participate in this study. I have been given a copy of this form.

Name of Subject

Signature of Subject

Date

SIGNATURE OF INVESTIGATOR

These are the terms under which I will conduct research.

Signature of Investigator
Revised February 2008

Date

APPENDIX 10

Compensation Receipt Form

Compensation received from Aleksandar Milosevic for participant in the research study titled:

Personality, behaviour, and childhood experiences:
A typology of gamblers

Recipient: _____

Compensation Amount: \$ _____ CDN

Date of Compensation: _____

I received \$ _____ CDN in Devonshire Mall money for my participation in the above-mentioned study.

Recipient Signature: _____

APPENDIX 11

Treatment Referral Form

Mental Health Treatment Services in Windsor/Essex County

Crisis Lines

Community Crisis Centre	(519) 973-4435
Distress Line	(519) 256-5000
Sexual Assault Crisis Centre	(519) 253-9667
Sexual Assault Treatment Centre	(519) 255-2234
Victim Services	1-888-732-6228

General Mental Health

Canadian Mental Health Association Windsor-Essex County Branch	(519) 255-7440
Mental Health Service Information Ontario	1-866-531-2600
Ontario Psychological Association	1-800-268-0069
Teen Health Centre	(519) 253-8481
Windsor Regional Hospital Community Psychogeriatric Outreach - Mental Health Program for Older Adults	(519) 257-5105
Windsor Regional Hospital Inpatient Mental Health Care	(519) 254-5577, ext. 75186

Depression & Anxiety

Windsor Regional Hospital Mood and Anxiety Clinic	(519) 257-5125
Windsor Mood Disorders Self-Help Group	(519) 979-5089

Substance Abuse & Gambling

Brentwood Recovery Home	(519) 253-2441
Drouillard Road Clinic	(519) 977-9772
House of Sophrosyne – Recovery Programs for Women	(519) 252-2711
Windsor Gamblers Anonymous	(519) 971-5215
Windsor Regional Hospital Addiction Assessment and Outpatient Service	(519) 257-5220
Windsor Regional Hospital Concurrent Disorder Treatment Service	(519) 257-5125
Windsor Regional Hospital Problem Gambling Services	(519) 254-2112
Windsor Regional Hospital Withdrawal Management Residential Service	(519) 257-5225

VITA AUCTORIS

Aleksandar Milosevic was born in 1980 in Windsor, Ontario. He graduated from W. C. Kennedy Collegiate Institute in 1999. From there he went on to the University of Windsor where he obtained a B.Sc. in General Science in 2002, a B.A. Honours in Psychology in 2004, and a M.A. in Clinical Psychology in 2007. He is currently a candidate for a Ph.D. degree in Clinical Psychology at the University of Windsor. He will complete requirements for his doctoral degree, and will begin a psychologist position at the Royal Ottawa Health Care Group, Integrated Forensic Program in Brockville, Ontario, in September 2011.