Running title: Disordered Gambling

Disordered Gambling: Etiology, Trajectory and Clinical Considerations

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Abstract

Gambling-related research has advanced rapidly during the past 20 years. As a result of expanding interest toward pathological gambling (PG), stakeholders (e.g., clinicians, regulators, and policy makers) have a better understanding of excessive gambling, including its etiology (e.g., neurobiological/neurogenetic, psychological, and sociological factors) and trajectory (e.g., initiation, course, and adaptation to gambling exposure). In this article, we will examine these advances in PG-related research and then consider some of the clinical implications of these advances. We will consider the DSM-V Impulse Control Work Group's recently proposed changes to the DSM criteria for PG. We also will review how clinicians can more accurately and efficiently diagnose clients seeking help for gambling-related problems by utilizing brief screens. Finally, we consider the importance of future research that can identify behavioral markers for PG. We suggest that identifying these markers will allow clinicians to make earlier diagnoses, suggest targeted treatments, and advance secondary prevention efforts.

Key words: pathological gambling, epidemiology, brief screening, behavioral markers

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Glossary

- Pathological gambling (PG): "…persistent and recurrent maladaptive gambling behavior… that disrupts personal, family or vocational pursuits" (American Psychiatric Association 1994, p. 615).
- Sub-clinical pathological gambling (i.e., problem gambling): gambling behavior that does not meet the criteria for pathological gambling but results in some adverse consequence (National Research Council 1999).
- Disordered gambling: a term used to describe the range of gambling problems, which includes pathological and sub-clinical gambling (Shaffer et al 1997).
- Epidemiology: the study of the distribution and determinants of health-related states or events in specified populations (Last 1983).
- Shadow syndrome: a cluster of sub-clinical signs and symptoms related to an abnormal underlying condition (Ratey & Johnson 1997).
- "Cocktail" treatment approach: the use of multiple and integrated treatments (e.g., a plan that integrates medication, group counseling, financial education, and family therapy).
- Nosology: area of scientific specialization that deals with the classification of diseases.

Acronyms

- Pathological gambling: PG
- Pathological gamblers: PGs
- American Psychiatric Association: APA
- Diagnostic and Statistical Manual of Mental Disorders: DSM
- Brief bio-social gambling screen: BBGS
- Positive predictive value: PPV
- National Epidemiological Survey on Alcohol and Related Conditions: NESARC

Central points

1. Short History of Gambling Research and Nosology
2. Factors that Influence the Etiology of Gambling Disorders
    a. Neurobiological/Neurogenetic, Psychological and Sociological Factors
3. The Trajectory of Gambling Disorders
    a. Exposure and Adaptation
    b. Games People Play
4. Clinical Considerations of Treating Gambling Disorders
    a. Diagnostic and Treatment Considerations

Brief annotations

Boudreau A, LaBrie RA, Shaffer HJ. 2009. Towards DSM-V: "Shadow syndrome" symptom patterns among pathological gamblers. *Addiction Research & Theory* 17:406-19:

- Examines sub-clinical signs and symptoms associated with PG.

Gebauer L, LaBrie RA, Shaffer HJ. 2010. Optimizing DSM-IV classification accuracy: a brief bio-social screen for detecting current gambling disorders among gamblers in the general household population. *Canadian Journal of Psychiatry* 55:82-90:

- Discusses a 3 item brief screen for PG that classifies cases more accurately than the 10 DSM-IV-TR items.

Kessler RC, Hwang I, LaBrie RA, Petukhova M, Sampson N, Shaffer HJ. 2008. DSM-IV pathological gambling in the National Comorbidity Survey Replication. *Psychological Medicine* 38:1351-60:

- Study examining the prevalence of PG, related co-morbidity and age of onset among a large, nationally representative sample.

LaPlante DA, Nelson SE, LaBrie RA, Shaffer HJ. 2008a. Stability and progression of disordered gambling: Lessons from longitudinal studies. *Canadian Journal of Psychiatry* 53:52-60:

- Review of longitudinal studies that casts doubt about the intractability and progressiveness of PG.

LaPlante DA, Shaffer HJ. 2007. Understanding the influence of gambling opportunities: Expanding exposure models to include adaptation. *American Journal of Orthopsychiatry* 77:616-23:

- Review that examines the gambling-related risks associated with increased exposure to gambling opportunities and shows how adaptation attenuates the influence of exposure.

Petry NM, Stinson FS, Grant BF. 2005. Comorbidity of DSM-IV pathological gambling and other psychiatric disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry* 66:564-74:

- Study examining the prevalence of PG and PG-related co-morbidity among a large, nationally representative sample.

Shaffer HJ, LaPlante DA, LaBrie RA, Kidman R, Donato A, Stanton M. 2004c. Toward a syndrome model of addiction: Multiple expressions, common etiology. *Harvard Review of Psychiatry* 12:367-74:

- Discusses an etiological model of addiction that postulates that there are shared risk factors that influence different manifestations of addiction (i.e., behavioral and chemical).

Slutske WS. 2006. Natural recovery and treatment-seeking in pathological gambling: Results of two U.S. national surveys. *American Journal of Psychiatry* 163:297-302:

- Examination of PG-related treatment and recovery, using two nationally representative samples, that casts doubt about the assumptions that PG is chronic and persisting.

"Gambling has a long past, but a short history" (Nathan 2001).

Introduction

Gambling is an activity in which something of value is risked on the outcome of an event when the probability of winning or losing is less than certain (Korn & Shaffer 1999). The American Psychiatric Association currently classifies pathological gambling (PG) as an impulse control disorder and defines it as "persistent and recurrent maladaptive gambling behavior that disrupts personal, family or vocational pursuits" (1994, p. 615). The National Research Council (1999) defines problem (i.e., sub-clinical) gambling as gambling behavior that does not meet the criteria for PG but results in harmful effects to gamblers, their families, significant others, friends, co-workers, and others. Disordered gambling is a term used to describe the full range of gambling problems, which includes pathological and sub-clinical gambling (Shaffer et al 1997).

As with most addictive disorders, the vast majority of those who eventually experience disordered gambling do not develop addiction after their initial gambling experiences. Similar to other expressions of addiction (e.g., excessive psychoactive substance use), among those who have experienced a gambling disorder, gambling escalated – sometimes rapidly and more often slowly – as players develop neuroadaptation (i.e., tolerance and withdrawal). In addition to physical signs and symptoms, disordered gamblers often experience negative psychosocial consequences (e.g., debt, shame, guilt, depression, loss of control; Shaffer et al 2004c). Individuals who continue to gamble despite these adverse consequences, lose control over their gambling, and crave opportunities to gamble likely are experiencing a clinical disorder: PG.

PG is a more complex and unstable disorder (LaPlante et al 2008a) than originally and traditionally thought (e.g., American Psychiatric Association 2000; Gamblers Anonymous 2010; National Council on Problem Gambling 2010). For example, the National Council on Problem Gambling reflects the long-held view of PG as a progressive disorder when they describe PG as "…a progressive addiction characterized by increasing preoccupation with gambling (and) a need to bet more money more frequently… in spite of mounting, serious, negative consequences (2010)." This view represents the current Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) description of PG (American Psychiatric Association 2000). However, longitudinal studies of PG indicate that this disorder is not necessarily progressive or stable (e.g., Jacques & Ladouceur 2006; LaBrie et al 2007b; Slutske et al 2003).

The purpose of this article is to review the history, etiology, and trajectory of disordered gambling as well as some of the primary clinical (e.g., diagnosis and treatment) and public health issues (e.g., epidemiology) that derive from the emerging research focusing on gambling disorders. How we conceptualize and understand the etiology and trajectory of gambling-related disorders has important implications for the diagnosis and treatment of these problems. As we will discuss, commonly held conventional beliefs influence how stakeholders (e.g., clinicians, public health workers, public policy makers) think about gambling-related issues (e.g., exposure to gambling opportunities, the games that gamblers play). To illustrate, clinicians who consider intemperate gambling as a moral issue are likely to view piety as a treatment; clinicians who view excessive gambling as a disease are more likely to employ treatments that include medical interventions compared with clinicians who view unrestrained gambling as a result of poor judgment.

Short History of Gambling Research and Nosology

Gambling has been part of the human experience since the beginning of recorded history. Although gambling has a long history, gambling research is a relatively new activity. The American Psychiatric Association (APA) first introduced PG to the diagnostic nomenclature in 1980 (American Psychiatric Association 1980). This event stimulated a new interest in gambling studies in general and the study of PG in particular (Eber & Shaffer 2000; Shaffer et al in press-b; Shaffer et al 2006). An examination of scientific and scholarly gambling citations between 1903 and 2003 shows that a disproportionate number of gambling-related articles were published during the latter part of the twentieth century and the first decade of the 21st century; nearly all (97%) were published after 1963, and approximately one third of these were published between 1998 and 2003 (Shaffer et al 2006).

Transition from Gambling to Gambling Disorders

Gambling research has become increasingly focused on the association between psychopathology and excessive gambling; the reference to psychopathology became more prevalent among gambling citations during the period between 1999 and 2003 than among articles published during the past century (Shaffer et al 2006). As a result of this shift, Shaffer et al (2006) suggested that gambling research is bifurcating between (1) studies about gambling, and (2) studies about PG. As we noted earlier, this shift was accompanied by a dramatic increase

in the knowledge-base concerning PG during the latter part of the 20th century and into the first decade of the 21st century. As knowledge about gambling advanced, new research stimulated epidemiological challenges to the conventional wisdoms about gambling. Most notably, there has been a new understanding about (1) the relationships among gambling exposure, adaptation to this exposure, and disordered gambling; and (2) the influence of games, gambling involvement, and disordered gambling. Next, we will briefly describe these challenges and then discuss them in more detail later in the section focusing on trajectory.

Exposure and Adaptation

Observers traditionally have presumed that exposure to gambling opportunities was sufficient to stimulate the development of gambling disorders (e.g., Kindt 1994; Lorenz 1998; Volberg 2000). However, recent empirical research indicates that individuals adapt relatively quickly after exposure to gambling opportunities and the prevalence of PG only increases during the short term – as a novelty effect – after the introduction of new gambling opportunities (LaPlante & Shaffer 2007). One way to illustrate adaptation to gambling exposure is to consider the prevalence rate for PG before and after the introduction of new gambling opportunities. More specifically, the lifetime prevalence rate of PG in the US observed during the mid-1970's (0.7%; Commission on the Review of the National Policy Toward Gambling 1976; Kallick et al 1979) is consistent with the US lifetime rates reported recently (0.4-0.6%; Kessler et al 2008; Petry et al 2005). Thus, contrary to predictions derived from the exposure model, the prevalence of PG has remained stable or been influenced by adaptation during the past 35 years despite an unprecedented increase in opportunities and access to gambling (e.g., lotteries, casinos, internet gambling) throughout this period.

Gambling Involvement

If exposure and opportunity to gamble are not the primary driving forces behind the development and maintenance of PG, what other factors might influence the incidence and prevalence of this disorder? Some researchers have reported an association between specific gambling activities and disordered gambling (e.g., Productivity Commission 1999; Smith & Wynne 2004), often implicating gambling machines as especially "addictive" because of their ubiquity or their capacity to deliver wins according to a strict variable ratio schedule of reinforcement. However, as we will discuss in more detail subsequently in our trajectory section, new research shows that

gambling involvement (i.e., the number of games in which one participates) is a better predictor of PG than participation in a particular game (e.g., LaPlante et al 2009b; Welte et al 2009). This new research has important consequences for advancing clinical aspects of PG treatment, such as diagnosis and treatment. By knowing that gambling involvement evidences a stronger association with PG than the games played, researchers have a more sophisticated understanding of the disorder; this perspective encourages clinicians to focus assessment and treatment upon the full range of patient gambling activities rather than just the individual games that people play.

Just as new research can impact clinical practice, gambling-related research methods are evolving. As we describe more fully below, new technology has provided the opportunity to advance investigative methods; the implications of these changes include the ability and opportunity to gather more reliable information about gambling-related behavior.

A Paradigm Shift in Gambling Research

In addition to the previously mentioned dramatic increase in the number of gambling publications, there also has been a pronounced shift in the research methods that scientists utilize to study gambling. Until recently, the majority of empirical gambling-related research relied upon opinion, self-report and anecdote as the evidence base for understanding disordered gambling behavior (Shaffer et al in press-b). The downside of relying on such data is that it is vulnerable to biases, both unintentional (e.g., failing to recall past gambling behavior accurately) and intentional (e.g., reporting information inaccurately to manage interviewer impressions). Simply put, people do not always say what they do or do what they say. As a result of the limitations that are associated with self-report methods, the findings of this body of gambling research should be interpreted with caution.

With the emergence of Internet gambling, empirical gambling research has evolved. There is a growing body of research focused on actual, instead of self-reported, gambling behavior (Shaffer et al in press-b). By studying actual gambling behavior, investigators avoid the limitations and biases commonly associated with self-report. This strategy provides a more direct and perhaps more valid indicator of gambling patterns, decisions under conditions of uncertainty, and the impact of winning or losing than has been available from self-report. Gambling involves so many factors (e.g., odds, patterns of risk taking, patterns of wins and losses) that players rarely can recall their play patterns with the precision necessary for research needs. For example, in one

of the first studies of actual gambling behavior, Xuan and Shaffer (2009) observed that self-identified problem gamblers became more conservative as they approached the end of their gambling – a finding that would not have been expected from the self-report literature, which would have predicted more liberal risk taking (e.g., "chasing"). The shift from investigative reliance on self-report to actual gambling behavior first occurred among those interested in Internet gambling; but, more recently, lottery research also has integrated data derived from actual gambling behavior (LaPlante et al in press). As gambling research evolved, so did the diagnosis of PG. Next, we will discuss PG and its integration into the nosology of the American Psychiatric Association (APA).

Intemperate Gambling Becomes a Psychiatric Disorder

The APA first integrated the criteria for PG into the third edition of its Diagnostic and Statistical Manual of Mental Disorders (DSM; American Psychiatric Association 1980). However, along with the acceptance of PG as a psychiatric disorder came evidence of diagnostic and social ambivalence. For example, consider the following cautionary DSM note:

> "…[I]nclusion here, for clinical and research purposes, of a diagnostic category such as Pathological Gambling or Pedophilia does not imply that the condition meets legal or other non-medical criteria for what constitutes mental disease, mental disorder, or mental disability. The clinical and scientific considerations involved in categorization of these conditions as mental disorders may not be wholly relevant to legal judgments, for example, that take into account such issues as individual responsibility, disability discrimination and competency" (American Psychiatric Association 1994, p. xxvii).

It is interesting to note that PG and pedophilia are the only disorders that the DSM includes in this disclaimer. Because this cautionary note diminishes the exculpatory value of PG and pedophilia within legal settings, it implies that the authors of the DSM-III were uncertain about the construct validity of these disorders. Their ambivalence is evidenced by the notion that these conditions might not be "wholly relevant to legal judgments" permitting that these disorders might be partially relevant. The ambivalence both reflects and encourages a skeptical view of PG and its origins.

Currently, the DSM-IV-TR defines PG as a "…persistent and recurrent maladaptive gambling behavior… that disrupts personal, family or vocational pursuits" (American Psychiatric

Association 2000, p. 671). Individuals who concurrently experience five or more of the following ten criteria meet the diagnostic threshold for PG (American Psychiatric Association 2000):

1. Preoccupation with gambling;
2. Needing to gamble with increasing amounts of money in order to achieve the desired excitement;
3. Repeated unsuccessful efforts to control, cut back, or stop gambling;
4. Restless or irritable when attempting to cut down or stop gambling;
5. Gambling as a way of escaping from problems;
6. After losing money gambling, often returning another day to get even ("chasing" one's losses);
7. Lying to family members, a therapist, or others to conceal the extent of involvement with gambling;
8. Committing illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling[3];
9. Jeopardizing or losing a significant relationship, job, or educational or career opportunity because of gambling;
10. Relying on others to provide money to relieve a desperate financial situation caused by gambling.

As this article goes to press, the APA is revising DSM-IV by proposing major changes for the DSM-V PG criteria, including no longer classifying PG as an impulse control disorder (American Psychiatric Association 2010). If these proposed changes occur, there will be important clinical ramifications (e.g., changes in the number of PG criteria and corresponding screens). We will discuss these proposed changes and potential clinical ramifications in more detail in our clinical considerations sections.

Factors that Influence the Etiology of Gambling Disorders

There are few etiological models of addiction in general and of PG in particular. Despite the paucity of theory, research suggests that the etiology of gambling disorders is complex and multi-factorial. A recent etiological model, the syndrome model of addiction (Shaffer et al 2004c), postulates that there are shared neurobiological, psychological and social risk factors that influence the development and maintenance of different manifestations of addiction. These risk factors are similar for both substance-based (e.g., cocaine dependence) and activity-based (e.g., disordered gambling) expressions of addiction. The syndrome model further states that addiction develops into its specific manifestation as a result of exposure to and desirable subjective

[3] The APA currently is proposing that this criterion be deleted from its forthcoming DSM-V.

experiences associated with objects of addiction. Once the expression(s) of the addiction syndrome emerge, manifestations of the syndrome simultaneously reflect both the specific object of addiction (e.g., cirrhosis with alcohol addiction, debt with gambling addiction) and general characteristics of addiction (e.g., tolerance, withdrawal).

The syndrome model of addiction has parallels to our understanding of another syndrome: AIDS. Before AIDS was identified as a consequence of HIV infection, clinicians did not recognize rare diseases, now known to be associated with AIDS, as opportunistic infections of an underlying immune deficiency syndrome. Similarly, for both behavioral and substance-related addiction "…neurobiological research suggests that addictive disorders might not be independent: each outwardly unique addiction disorder might be a distinctive expression of the same underlying addiction syndrome…. Our analysis of the extant literature reveals that the specific objects of addiction play a less central role in the development of addiction than previously thought, and it identifies the need for a more comprehensive philosophy of addiction" (Shaffer et al 2004c, p. 367).

As researchers identify and clarify the interactive roles that neurobiological, psychological and social factors play in the etiology, maintenance and recovery processes associated with PG, it is important to consider the interactive nature of these factors. These risk factors will combine to influence the emergence, development and maintenance of addiction. For example, some people with a genetic load that predisposes them to addiction (e.g., Kendler et al 2003) might not develop PG because they are not exposed to gambling or are more attracted by competing interests (e.g., religion, work) that diminish the likelihood of gambling. Alternatively, people with resilient psychological features might not develop disordered gambling despite being heavily exposed to gambling opportunities; gamblers compromised by mental disorders might develop disordered gambling despite having *less* exposure to gambling opportunities. Finally, the social setting influences both the neurobiology and the psychology of gambling. To illustrate, research indicates that psychological risk factors (e.g., impulsivity and delinquency) and sociological risk factors (e.g. family, peer groups and poverty) are common across behavioral and chemical expressions of addictions (Shaffer et al 2004c). Finally, it is important to note that risk factors for addiction – both individually and interactively – pose potentially recursive risks that can modify the original risk matrix associated with the etiology of addiction.

In the following section, we will discuss the interactive (1) neurobiological/neurogenetic, (2) psychological, and (3) sociological influences that can impact the etiology, persistence, and impact of gambling-related problems. Zinberg (1984) was the first to recognize the importance of understanding the interactive biological (drug), psychological (set), and social influences (setting) that determine the subjective effects of intoxicant use. Observers have compared gambling to the use of intoxicants (Shaffer et al 2004c). Similar to ingesting stimulants, there is evidence that gambling is associated with autonomic arousal including elevated blood pressure, heart rate, and mood (e.g., Krueger et al 2005; Ladouceur et al 2003; Meyer et al 2000; Meyer et al 2004; Moodie & Finnigan 2005; Yucha et al 2007). Consistent with this understanding, Shaffer and Korn (2002) extended Zinberg's original ideas about the effects of intoxicant use by suggesting that three interactive factors – the game played, the player's set (i.e., psychological expectations about the game or games played), and the social setting within which people gamble (i.e., the physical, cultural, and interpersonal aspects of the gambling setting) – influence the experience of gambling.

Neurobiological and Neurogenetic Factors

There is emerging scientific evidence indicating that gambling problems might be associated with neurobiological and neurogenetic influences. When an interaction with a potential object of addiction (e.g., alcohol or gambling) produce desirable feelings, repeated exposure can lead to changes in the brain's reward circuitry; these changes likely reflect shifts in the release of dopamine, serotonin and other neurotransmitters (Shaffer et al 2004c). There is evidence that specific neurotransmitters relate to various expressions of addiction, including PG. In the case of PG, there is evidence of associations between PG and a variety of neurotransmitters (e.g., noradrenaline, serotonin, glutamate, dopamine and endorphins; Potenza 2008). Specifically, researchers have found increased noradrenergic measures among PGs (Meyer et al 2000; Meyer et al 2004). In addition, serotonergic drugs (Grant & Potenza 2006; Hollander et al 2000), opioid antagonists (Grant et al 2006; Kim et al 2001), and glutamatergic agents (Grant et al 2007) have been found to positively impact some disordered gamblers.

An illustration of the role of neurotransmitters in PG is the emergence of PG among patients with no apparent history of PG after they receive dopamine agonist treatment for Parkinson's disease (Bostwick et al 2009; Dodd et al 2005; Driver-Dunckley et al 2003; Singh et al 2007; Voon et al

2007; Weintraub et al 2006) and restless leg syndrome (Driver-Dunckley et al 2007; Tippmann-Peickert et al 2007). Such findings are not unique to PG. Compared to their counterparts with deficient levels of dopamine, research indicates that individuals treated with dopamine agonists have increased risk for developing various expressions of addiction, such as compulsive sexual behaviors (Bostwick et al 2009; Klos et al 2005; Pontone et al 2006; Singh et al 2007; Weintraub et al 2006), compulsive eating (Klos et al 2005; Nirenberg & Waters 2005), and compulsive shopping or spending (Klos et al 2005; Pontone et al 2006; Voon et al 2006; Weintraub et al 2006). Research that observed different brain activity characteristics among PGs compared with non-PGs (Potenza 2008) provides additional support for a neurobiological link to PG. Specifically, when viewing tapes of other people in gambling-related situations, PGs showed decreased activity in regions of the brain implicated in impulse regulation (e.g., the ventromedial prefrontal cortex) compared to non-PGs (Potenza et al 2003a; Potenza et al 2003b).

There is evidence that genetics play an important role in the general risk for addiction (Kendler et al 2003), and gambling addiction in particular. For example, studies show that 50-60% of the variation in risk for PG is accounted for by genetics (Lobo & Kennedy 2009). Research also shows that gambling problems tend to run in families (Black et al 2006; Gambino et al 1993). For example, immediate family members of pathological gamblers are more susceptible to PG than individuals without immediate PG family members (Black et al 2006). Finally, studies among twins found that PG was higher among co-twins of individuals with PG than co-twins of individuals without PG (Slutske et al 2001; Slutske et al 2009; Xian et al 2007). In sum, genetic influences might not determine the development of a specific expression of addiction; however, genetics does influence the risk of addiction in general. Among people who experience PG, there are important neurobiological differences compared to their counterparts without gambling problems. Next, we briefly will discuss the potential impact of another neurogenetic factor – gender – on PG.

Gender and Gambling Disorders

Although the evidence suggests that gender is a neurogenetic risk for PG (e.g., Johansson et al 2009; Kessler et al 2008; Petry et al 2005), there also is evidence that gender is not necessarily a moderator of gambling behavior and disordered gambling (e.g., LaPlante et al 2006; Nelson et al 2006). Research with treatment seekers reveals that gender primarily influences the age of onset

for gambling and its trajectory (e.g., Hing & Breen 2001; Ladd & Petry 2002; Potenza et al 2001; Tavares et al 2003; Tavares et al 2001). More specifically, women tend to begin gambling later than men, but women develop gambling problems more rapidly than men. Women also seek treatment sooner than men. Beyond these differences, gender minimally contributes to game preference or the prevalence, development and maintenance of PG beyond that of specific psychosocial factors (e.g., demographics, gambling history, gambling behavior and consequences, criminality, and comorbidity; LaPlante et al 2006; Nelson et al 2006). These findings suggest that gender is less informative than specific demographic, economic, and health-related factors (e.g., age, gambling background, chemical addictions, socio-economic status, family responsibilities) for understanding gambling and problem gambling behavior.

Psychological Factors

In this section, we will consider the association between co-occurring clinical and sub-clinical (i.e., "shadow syndromes") disorders and PG, and the role of expectations and gambling. PGs are significantly more likely to have mental disorders and/or substance use disorders compared to those without gambling problems. Overall, research indicates that individuals with such psychiatric disorders are approximately 17 times more likely to develop PG than those without such disorders (Kessler et al 2008). Specifically, PGs are 5.5 times more likely than non-PGs to have had a substance abuse disorder (Kessler et al 2008); 75% of PGs have had an alcohol disorder, 38% have had a drug use disorder and 60% have had nicotine dependence (Petry et al 2005). Concerning mental health disorders, PGs are 4 times more likely than non-PGs to experience a mood disorder in their lifetime and 3 times more likely to have had an anxiety disorder (Kessler et al 2008); 50% of PGs have had a mood disorder, 41% have had an anxiety disorder and 61% of PGs have had a personality disorder (Petry et al 2005).

In addition to being vulnerable to psychiatric disorders, research also indicates that PGs are more likely to experience sub-clinical patterns (i.e., shadow syndromes; Boudreau et al 2009). Shadow syndromes are sub-clinical clusters of signs and symptoms. As features of human personality and character, everyone has characteristic shadows (Ratey & Johnson 1997); however, these typically go unnoticed or undetected by observers and often are thought of as a person's personality style (Shapiro 1965). In the case of PG-related shadow syndromes, research indicates that there is a likelihood that individuals with a PG diagnosis experience characteristic reliable

and identifiable sub-clinical symptom patterns from symptom clusters associated with psychiatric disorders other than PG (e.g., dysthemia, anxiety and specific phobias; Boudreau et al 2009).

Research indicates that, apart from clinical disorders and shadow syndromes, there are other psychological factors that also influence PG. For example, studies have found that the expectations that gamblers have about the games they play also impact gambling (e.g., Ladouceur et al 2003; Pantalon et al 2008). Individuals who gamble because of an expectation of excitement are more likely to have large gambling wins and losses, more frequent and varied gambling, and more symptoms of pathological gambling (Pantalon et al 2008). These expectations and the development of gambling-related problems also are associated with impulsivity (e.g., Blanco et al 2009; Petry 2001; Vitaro et al 1999).

Sociological Factors

Just as there are neurobiological and psychological factors that influence the etiology of gambling disorders, social factors interact to influence the emergence of intemperate gambling. Of the three interactive factors that influence the effect of intoxicant use (i.e., the drug, the set, the setting), Zinberg and Shaffer (1990) noted that the social setting is the least understood of these three factors and there is inadequate attention paid to the social context during social policy considerations, clinical interventions, and scientific research.

To illustrate the potential role of sociological factors different populations (e.g., adolescents, college students, general population, adults in treatment and prisons) tend to prefer different gambling activities (Shaffer et al 1997; 1999a). Because gambling is illegal for adolescents, they prefer gambling activities that are socially accessible and do not require authorization (e.g., games of skill, non-casino card betting, sports betting; Shaffer et al 1997; Welte et al 2009) compared to their adult gambling counterparts.

In addition, a number of aspects about an individual's social setting impact gambling behavior (Shaffer & Korn 2002; Shaffer et al 2004c). For instance, researchers have found an association between gambling problems, poor parental supervision (Vitaro et al 2001), and delinquency (Johansson et al 2009; Vitaro et al 2001). Research also has shown that individuals who are widowed, separated or divorced (Petry et al 2005), and individuals who begin gambling at a young age (Kessler et al 2008) are more susceptible to gambling problems than others. Further,

there are correlations between gambling behavior and socio-economic status (SES): studies have found that individuals with a lower SES are at increased risk for gambling problems (Welte et al 2008).

Markers for Risk and Protective Factors

PG-related research is nascent and there is much more to be learned about factors that influence the etiology of PG. The identification of risk and protective factors for the development of gambling problems has stimulated interest to distinguish behavioral markers that will predict the emergence of gambling disorders. Prospective studies of actual gambling behavior have emerged (Shaffer et al in press-b); this circumstance provides a more accurate picture of gambling and how gambling problems develop than does cross-sectional self-report based research. This advance provides the opportunity to identify behavioral markers that might identify and/or influence the emergence of disordered gambling (LaBrie & Shaffer in press). For example, researchers have suggested that the following types of gambling involvement are precursors to the development of gambling problems: betting intensity (i.e., average number of bets per day) and gambling frequency (i.e., number of gambling days; Braverman & Shaffer 2010; LaBrie et al 2008; LaBrie & Shaffer in press); gambling trajectory or tolerance (i.e., the tendency to increase the amount of wagered money; LaPlante et al 2008b; Xuan & Shaffer 2009); and gambling variability (i.e., deviating from a uniform, consistent gambling pattern; Braverman & Shaffer 2010; LaBrie et al 2008; LaBrie et al 2007a; LaPlante et al 2009a). We will explore the pursuit of behavioral markers in more detail later during our discussion concerning clinical recommendations.

The Trajectory of Gambling Disorders

Now that research provides a better understanding of the factors that can influence the etiology of PG, it is important to consider the trajectory of PG, including its course and persistence. The following section discusses various perspectives that researchers have held concerning the trajectory of PG; we also examine whether these perspectives are consistent with the empirical evidence.

Exposure and Adaptation

According to exposure theorists (e.g., Kindt 1994; Lorenz 1998; Volberg 2000), as gambling opportunities increase, there will be a corresponding increase in gambling problems among

individuals exposed to these new opportunities. Further, this exposure model proposes that upon the introduction of these new opportunities, there will be a steady progression of the prevalence of exposed individuals experiencing gambling problems and an increase in the severity of gambling-related problems. For example, Kindt hypothesized that "…current data shows that when gambling activities are legalized, economies will be plagued with 100% to 550% increases in the numbers of addicted gamblers (probably within one to five years, but almost certainly within fifteen years)" (1994, p. 59). In contrast, Shaffer and his colleagues proposed the adaptation model (LaPlante & Shaffer 2007; Shaffer et al 1997; 1999a; Shaffer et al 2004a; Shaffer & Zinberg 1985)to suggest that, following initial increases in the number and types of adverse reactions to new and novel social opportunities (e.g., substance use, gambling), people will adapt gradually and become more resistant to those events, eventually leading to stable or lower prevalence rates associated with the adverse circumstance (Shaffer et al 2004c; Zinberg 1981; 1984). Contrary to the presumptions of many exposure model pundits, the adaptation model argues that gamblers are dynamic and reactive rather than passive victims of gambling exposure. In the next section, we will discuss in more detail recent empirical research, completed almost fifteen years after Kindt's speculation, that supports the adaptation model; these studies indicate that individuals adapt relatively quickly after exposure to gambling opportunities and the prevalence of PG only increases in the short term after the introduction of new gambling opportunities (LaPlante & Shaffer 2007).

Consistent with the exposure model, observers often identify increases in the rate of gambling-related problems soon after new opportunities to gamble become available. However, consistent with the adaptation model, research also shows that the prevalence rate of gambling disorders only increases in the short-term; over time the rate stabilizes and then tends to decline (Bondolfi et al 2008; Jacques & Ladouceur 2006; Jacques et al 2000). The adaptation curve is similar to a curve reflecting the spread of infectious diseases (LaPlante & Shaffer 2007). For example, just as with infectious diseases (e.g., H1N1), the most vulnerable develop the disorder first, the population begins to understand risks and preventive measures, and the spread of the illness gradually diminishes. Consequently, claims about the rate of PG require careful consideration about the time frame within which these observations are made. As the next section will show, in addition to broad population trends, individuals also shift their relationship to gambling and gambling disorders.

Adaptation to Gambling Exposure

Contrary to the idea that PG is a relentlessly progressive disorder, prospective research reveals that individuals move in and out of PG (e.g., Jacques & Ladouceur 2006; LaBrie et al 2007b; Slutske et al 2003). For example, Slutske (2006) reported that, among individuals with a lifetime history of PG, 36%-39% did not experience any gambling-related problems during the past year. Furthermore, longitudinal studies focusing on disordered gambling behavior provide support for the view that (1) disordered gambling is not necessarily progressive, (2) individuals with more severe gambling problems can improve just as those with less severe gambling problems, and (3) individuals with some gambling problems decline at a rate similar to those with no gambling problems (LaPlante et al 2008a); also, individuals often adapt to the risks and hazards of exposure to new gambling opportunities (LaPlante & Shaffer 2007).

Nevertheless, LaPlante and Shaffer (2007) also found evidence to suggest that exposure is associated with increases levels of gambling-related problems, but this effect was not necessarily durable

Comment About Lifetime Prevalence Rates

There is a growing interest in avoiding the use lifetime prevalence estimates for psychiatric disorders. For example, many studies focusing on psychiatric epidemiology are cross-sectional and report lifetime estimates of disordered gambling prevalence; these results consistently show declining prevalence rates with advancing age (Streiner et al 2009). However, in a closed cohort with no mortality, the lifetime prevalence of a disorder must increase or remain constant. The observed paradoxical decline in lifetime prevalence is likely due to a variety of factors, such as study design and/or participant forgetting and reframing (Streiner et al 2009). Lifetime prevalence also is insensitive to changes in treatment effectiveness or demand for services (Streiner et al 2009). Finally, lifetime estimates fail to account for temporal symptom clustering. In other words, lifetime estimates do not consider the time period for which the individual met the individual diagnostic criterion (Shaffer et al 2004b). From a clinical perspective, symptoms must co-occur to be diagnostically valid. A lifetime prevalence estimate permits a case to be identified despite disjunctive symptoms; that is, symptoms can occur at different times during the life course but are counted as representing a disorder. With lifetime estimates, the extent of a gambling problem can be overestimated because time is not weighted (Shaffer et al 2004b). For these reasons, Streiner et al (2009) argue that scientists drop lifetime prevalence from the lexicon of psychiatric epidemiology and eliminate it from epidemiological research designs.

across settings and time points. Other research provides evidence for a novelty effect followed by adaptation. For instance, more recent Nevada residents reported more gambling-related problems than long-term residents (Volberg 2002) and newer casino employees claimed more gambling-related problems than employees who had been working longer in the industry (Shaffer et al 1999b). In addition, the past year prevalence rates of disordered gambling have remained stable in Switzerland despite widespread openings of casinos during the previous ten years (Bondolfi et al 2008).

As we mentioned previously, evidence supporting the adaptation model also comes from examining lifetime PG prevalence rates over time. During the middle 1970s, the lifetime rate of PG in the US was 0.7% (Commission on the Review of the National Policy Toward Gambling 1976; Kallick et al 1979). This first national estimate of PG prevalence is similar to recently reported lifetime estimates of prevalence among a national sample (0.4-0.6%; Kessler et al 2008; Petry et al 2005). The current available evidence suggests that the rate of PG has remained relatively stable during the past 35 years despite an unprecedented increase in opportunities and access to gambling (e.g., lotteries, casinos, internet gambling). Recently, researchers also have estimated the past year rate of PG in the US (e.g., 0.2-0.3%; Kessler et al 2008; Slutske 2006).

Internet Gambling

Another example of the adaptation model at work is the case of Internet gambling. The rates of Internet gambling and the extent of betting on Internet gambling are more moderate (e.g., LaBrie et al 2008; LaBrie et al 2007a; LaPlante et al 2008a; Wardle et al 2007) than some researchers have speculated (e.g., Griffiths 2003; Griffiths & Barnes 2008; Messerlian et al 2004; Wood et al 2007). For instance, the National Comorbidity Survey Replication found that 1% of the population used the Internet for gambling (Kessler et al 2008). Similarly, a study examining gambling behavior among a representative sample of adolescents and young adults observed that only 2-3% of participants had gambled via the Internet during the past year (Barnes et al 2010; Welte et al 2009). Further, a longitudinal study examining the gambling behavior of newly subscribed Internet bettors indicated that this population adapted to the Internet gambling service rapidly (i.e., quickly developing declines in participation, number of bets and size of stakes; LaPlante et al 2008b). As evidenced by these studies, the case of Internet gambling provides

little evidence that exposure is the primary driving force behind the prevalence and intensity of gambling.

Games People Play

The relationship between participation in a particular game and the emergence of disordered gambling is another important trajectory-related area of inquiry. The following section discusses how researchers have perceived the relationship between particular games and the prevalence of disordered gambling. We also examine whether the relationship between the extent of gambling "involvement" is a better predictor of disordered gambling than any particular game that people play.

Popular convention holds that exposure to electronic gambling machines is associated with higher rates of incidence and prevalence of gambling addiction compared to more traditional gambling activities (e.g., table games, sports betting, card games); yet, a comprehensive review (Dowling et al 2005) of existing empirical evidence concerning gambling behavior and electronic gambling machines casts doubt on the presumption of "addictive games." As we mentioned previously, some studies have found an association between specific gambling activities and disordered gambling (e.g., Breen & Zimmerman 2002), leading to the assumption that these games, for example slot machines, are especially "addictive" because of their high rate of gambling opportunities. Often, studies that claim such an association use samples of treatment seekers, who report disproportionate rates of play or more rapid rates of developing problems on specific games. However, treatment seekers are not representative of others with similar problems. Berkson's bias is the error that results from making generalizations from treatment seekers to the general population (Berkson 1946). Treatment seekers are different from non-treatment seekers in the general population with similar problems. Further, a high correlation between a specific type of gambling (e.g., Internet gambling) and problem gambling does not reflect a causal influence, but might mean that devotees of that type of gambling also engage in many forms of gambling.

Gambling Involvement

Gambling involvement (e.g., the number of games one plays) has emerged as a better predictor of PG than participation in a particular game; further, this finding has held true among both US

youths and UK adults (LaPlante et al 2009b; Welte et al 2009). An examination of gambling behavior among US youths illustrates the importance of controlling for other gambling involvement when examining relationships between specific gambling activities and gambling problems (Welte et al 2009). In this case, initial analyses between gambling types and gambling-related problems that did not control for participation in other gambling types indicated that Internet gambling is more strongly associated with gambling problems than other gambling activities; however, subsequent analyses controlling for participation in other forms of gambling found Internet gambling did not confer increased risk for gambling problems. Similarly, among a nationally representative sample of UK adults, LaPlante et al (2009b) identified an association between gambling involvement and gambling-related problems in a study that replicated the Welte et al (2009) methodology. It is important to note one limitation of these studies: the investigators used only one measure of gambling involvement. To date, researchers have defined involvement as the number of games played; there are other possible definitions for involvement. More research is necessary to examine other – and multiple – measures of involvement to refine more accurately the meaning of gambling involvement. Despite this limitation, these findings stress the importance of controlling for other gambling activities when examining associations between the games people play and gambling-related problems.

Treating Gambling Disorders: Clinical Considerations

The etiology and trajectory of gambling disorders provide twin pillars of information upon which clinicians can assess patients and develop treatment plans for those who are struggling with excessive gambling. The process of assessment and diagnosis marks the beginning of treatment. For this discussion, we separate diagnostic activities from other treatment for convenience only.

The assessment and treatment process rests upon a broad paradigmatic perspective (Shaffer & Freed 2005). How clinicians view gambling, in general, and excessive gambling in particular, will determine in large measure the nature and parameters of their assessment and treatment plan. For example, if a clinician views excessive gambling as a moral weakness, piety might become the object of treatment. Alternatively, if clinicians view the etiology of excessive gambling as the result of the confluence of genetic, psychological, and social forces, a much more sophisticated, and likely complicated, treatment plan will emerge. Historically, disordered gambling has been viewed from a number of perspectives; for example, PG has been referred to

as a moral (Quinn 1891), psychosocial (Orford 2001), impulse control (American Psychiatric Association 2000), and biologically-driven behavior (Comings 1998; Comings et al 1999). Consistently, and despite the many and varied perspectives toward intemperate gambling, the treatment options for gambling problems have included inpatient or outpatient therapy, and self-help groups. The evolution of perspectives toward excessive gambling continues. Notably, as we mentioned previously and will discuss in more detail below, the DSM-V now proposes that clinicians consider gambling as an addiction (American Psychiatric Association 2010).

Nosology and Pathological Gambling

As we discussed previously, PG is a relatively new addition to the DSM; along with recognition of this disorder has come diagnostic and social ambivalence (American Psychiatric Association 1980). The following section discusses criticisms of the current DSM-IV-TR criteria, proposed changes to these DSM criteria, and comments about the proposed criteria.

Criticisms of the Current Diagnostic Criteria

One criticism of the current DSM criteria is "...DSM's categories and their particulars—the 'same things' that scientists are studying—may not be 'things' at all" (Schmidt et al 2004; p. ix). Another concern is "...the categories and indicators are decided more by committee than by science...the basic methodology of the DSM for inclusion and delineation of disorders is based on committee consensus, the pitfalls and gross errors of which can be substantial" (Schmidt et al 2004; p. ix-x). Uncertainty about whether the DSM-created "things" that clinicians study and treat and whether these are distinct or dimensional "things" is essential to understanding gambling disorders (Waller & Meehl 1998; Widiger & Samuel 2005). The current diagnostic system and uncertainty about the DSM categories and particulars lead clinicians to suggest that diagnostic reliability is improved at the expense of validity (Barron 1998; Vaillant 1984).

Proposed Changes to the DSM-IV-TR Diagnostic Criteria

As we mentioned earlier, the APA is considering a number of changes to the PG criteria in DSM-V, which is currently being drafted. One significant change is moving PG from the Impulse Control Disorder category to the Substance-related Disorder category, which will be renamed Addiction and Related Disorders (American Psychiatric Association 2010). The APA justification is that PG has commonalities in clinical expression, etiology, comorbidity, physiology and treatment with substance use disorders (e.g., Shaffer et al 2004c).

The APA also is considering lowering the threshold for diagnosing PG. The current threshold is to satisfy any five of the criteria; statistical analyses concerning the appropriate threshold are in progress (American Psychiatric Association 2010). The justification for this change, according to the APA, is that several empirical studies have supported lowering the threshold for a diagnosis of PG (e.g., Jimenez-Murcia et al 2009; Stinchfield 2003; Stinchfield et al 2005). Finally, the APA is considering eliminating the DSM-IV-TR criterion concerning committing illegal acts (i.e., Have you committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling?). According to the APA, this change is justified because the PG illegal act criterion has such low prevalence that its elimination has little or no effect on the prevalence of PG and little effect on the information associated with the diagnosis in the aggregate (e.g., Strong & Kahler 2007). Next, we will consider the rationale and implications of the APA's proposed changes.

Considering the Proposed Criteria Revisions: Toward DSM-V

Although the DSM-IV criteria for diagnosing PG emerged from a limited empirical foundation (Lesieur & Rosenthal 1991), it is worth noting that the APA is now considering eliminating a criterion because it has been shown to have little or no effect on disorder prevalence and little effect on the information associated with the diagnosis in the aggregate. Interestingly, removing a criterion diminishes the potential heterogeneity among people who satisfy the diagnostic criteria for PG. A diagnosis using DSM-IV-TR, for example, allowed for 10 criteria to be taken 5 at a time; the new criteria allow for 9 criteria to be taken 5 at a time. Because not all symptoms are equally prevalent, the following oversimplification illustrates how eliminating a criterion can impact heterogeneity. The combinations of diagnostic possibility can be represented for DSM-IV-TR as 10P5=10!/10!-5!=10!/5! (i.e., 30,240). Consequently, DSM-IV-TR permits many types of PG among those diagnosed. If the proposed unspecified lower threshold goes into effect and one criterion is removed in DSM-V, there will be 50% fewer potential combinations of symptoms associated with the diagnostic threshold. For instance, if the threshold gets lowered to 4, these permutations of diagnostic possibility would be represented as 9P4=9!/9!-4!=9!/4! (i.e., 15,120).

With their proposal to eliminate the illegal act criterion for PG, the APA work group provides the following rationale: "The illegal act criterion of PG has been shown to have a low prevalence

with its elimination having little or no effect on prevalence and little effect on the information associated with the diagnosis in the aggregate" (American Psychiatric Association 2010). This rationale suggests there are other diagnostic criteria that the work group should consider for elimination. Jeopardizing relationships, work, or education appears to occur only at high severity levels of PG; in addition, this particular criterion does not have high sensitivity or positive predictive value (PPV; the proportion of individuals who endorse this criterion and who qualify to be diagnosed as pathological gamblers). Alternatively, both preoccupation and chasing have such high prevalence among both non-PGs and PGs that these criteria have little diagnostic value under the current system (e.g., Gebauer et al 2010; Nelson et al 2009; Strong & Kahler 2007; Toce-Gerstein et al 2003; Zimmerman et al 2006).

As we mentioned previously, the APA work group proposes to reclassify the diagnosis of PG from impulse-control disorders not elsewhere classified to substance-related disorders, which will be renamed addiction and related disorders. Although PG has clinical, epidemiological, etiological, physiological, and treatment commonalities with substance use disorders, it is worth noting that these similarities also exist among the substance use disorders and a variety of other behavioral expressions of addiction (e.g., excessive shopping). A relatively large literature evidences these commonalities. Consequently, the DSM-V work group should avoid creating a long list of Addiction and Related Disorders diagnoses organized by the objects of addiction. Instead, the syndrome model of addiction encourages an addiction diagnosis that is independent of the objects of addiction; diagnostic systems should identify the core features of addiction and then illustrate these with substance-related and behavioral expressions of this diagnostic class. Conceptualizing addiction this way avoids the incorrect view that the object causes the addiction and shifts the diagnostic focus toward patient needs (Shaffer et al 2004c). Further, this approach precludes reiterating a set of Aristotelian diagnoses for all possible objects of addiction, thereby avoiding Allen Frances' concern about the slippery slope of behavioral addictions (Wetzstein 2010).

Finally, the current and proposed diagnostic systems employ an equally weighted count system for diagnostic criteria; that is, in DSM-IV-TR, the diagnosis of PG requires endorsing five equally weighted criteria. Our previous suggestions only apply if we continue to employ an equally weighted count system. Alternatively, it is more desirable to advance a diagnostic system for PG (and most other psychiatric disorders) that would avoid simple counts and do more to

recognize the dynamic nature of the target disorders and the different pathways through which people arrive at these disordered states. This alternative system would maintain diagnostic criteria with extremely high sensitivity (e.g., preoccupation and chasing) and utilize those criteria as diagnostic gates. Once patients satisfy criteria to move through these gates, clinicians would then evaluate other criteria (e.g., the presence, severity and progression of gambling problems) that would advance diagnostic specificity.

In sum, the syndrome model of addiction encourages making the diagnosis of addiction independent of the object of addiction (though objects can be described for illustrative purposes); however, if an object driven diagnostic taxonomy remains, it should employ more, not fewer, criteria. These criteria would include the use of sensitive diagnostic gates and other diagnostic criteria that would advance specificity. Next, we will discuss whether there is evidence to support the view that PG is a unique disorder.

Distinguishing Characteristics between PGs and Recreational Gamblers

General validity concerns about the DSM diagnostic criteria, and more specific ambivalence about PG as a unique disorder, stimulate and sustain a long-standing debate in the psychopathology literature about whether we can describe mental disorders as extreme expressions of continuously distributed traits, or as qualitatively discrete patterns (e.g., Beauchaine 2007). For example, in the case of PG, a dimensional view of gambling would locate gambling disorders at the end of a continuum, and despite the quantitative distinction, this extreme behavior would be qualitatively similar to behaviors located at other points along the continuum. Conversely, a categorical view of gambling would locate disorders within a discrete extreme interval and the behaviors would be qualitatively different from behaviors outside the interval (i.e., a unique taxon). To date, most clinicians and researchers have defined and treated pathological gambling as a categorical illness, preferring nosological schemes that consider this excessive behavior pattern as a distinct disorder (e.g., Beauchaine 2007).

To explore whether PG is dimensional or categorical, researchers recently have engaged in taxometric research. This study provides the opportunity to determine whether it is possible to identify a distinct category or taxon of betting characteristics among heavily involved Internet sports gamblers (Braverman et al under review). Evidence of a taxon would imply that some heavy gamblers are qualitatively different from more involved recreational Internet gamblers.

After applying taxometric procedures (i.e., MAMBAC and MAXCOV) to three indicators of actual betting behavior (i.e., total amount of lost, total amount of bets and the total amount wagered), Braverman et al (under review) concluded that the results failed to provide support for the view that the most involved Internet sports gamblers include a distinct category of gamblers. Although much more research is necessary, in the absence of evidence for a distinct group of PGs, we can conclude that recreational gamblers and PGs have similar characteristics; the difference is that PGs just have more of these characteristics and/or more intense play patterns. In other words, this finding suggests that PG is not a unique disorder.

Taxometric findings have a number of public policy and clinical implications. Concerning public policy, Braverman et al (under review) recommend that responsible gaming programs: (1) encourage new technology that limits excessive patterns of play as opposed to trying to identify players with distinctive personal risk characteristics; and (2) consider emphasizing limits to gambling opportunities. These findings encourage clinicians to integrate the categorical and dimensional approaches. For example, Braverman et al (under review) suggest that a dimensional perspective can guide clinicians and researchers to track symptom intensity and severity during treatment, or when evaluating the efficacy of prevention efforts. Thus, preparing patients for long-term treatment outcomes. Conversely, a categorical approach is useful to solve pragmatic administrative needs (e.g., patient grouping, insurance billing). Next, we will discuss how PG-related epidemiological and trajectory evidence informs PG-related treatment considerations.

Treatment Considerations: Lessons from Epidemiological and Trajectory Evidence

People with gambling disorders are hesitant to enter treatment. Findings from the National Epidemiological Survey on Alcohol and Related Conditions (NESARC; Grant et al 2003a) and the Gambling Impact and Behavior Study (National Gambling Impact Study Commission 1999) indicated that among individuals who had experienced PG in their lifetime, 36%-39% had not experienced any gambling-related problems during the past year; however, only 5.5% of PGs received professional treatment for their gambling problems and only 7.3% had attended one or more gamblers anonymous meetings, a form of self-help (Slutske 2006). None of the participants in the National Co-morbidity Survey Replication (Kessler & Merikangas 2004) with a PG diagnosis ever received treatment for a gambling problem; however, 49% were treated for other

mental disorders (Kessler et al 2008).These studies indicate that about half of the PGs received treatment for other psychiatric disorders. However, neither they nor their treatment providers recognized that their disordered gambling required treatment. Consequently, it is imperative that clinicians learn about gambling-related disorders so that patients can integrate their care without having to enter specialized treatment, which attends only to gambling disorders.

There are many different situations that contribute to the hesitancy of people with gambling disorders to enter treatment. For example, excessive gamblers might be ashamed of their circumstance, unaware that help is available, uninsured or without financial resources to enter treatment, of the opinion that they can change on their own, or repelled by the treatments that are available (Pulford et al 2009; Suurvali et al 2009). The conventional response to these issues has been to encourage everyone with a gambling problem to enter treatment. This places an unnecessary burden on the health care system because the epidemiological evidence shows that excessive gamblers change more rapidly than observers expect (LaPlante et al 2008a) and that about half already are in treatment for other problems (Kessler et al 2008). As a result, it is imperative that we provide clinical tools (e.g., brief public health screens; guides for self-directed change; e-health resources) and public policies that allow and, under the proper circumstances, encourage individuals with gambling problems to recover on their own (i.e., self-help).

The epidemiology and trajectory of disordered gambling encourages clinicians to adjust their treatment strategies and practices. The following section discusses some of the clinical considerations and consequences of that emerge from the recent evidence focusing on gambling disorders. We suggest that clinicians recognize the probability and influence of co-morbid conditions, describe the chronicity of the disorder, discuss the merits of employing brief screening strategies and consider a treatment option unique to PG (i.e., gambling exclusion programs). Lastly, we encourage clinicians to consider the value of behavioral markers for gambling disorders. Because research concerning gambling-related treatment for gambling disorders is nascent, we encourage readers to consider our suggestions as fundamental guidelines rather than particular prescriptions about how to deal with specific clients seeking help for gambling-related problems.

Age of Onset, Multimorbidity, and Chronicity

The epidemiological and trajectory evidence reveals that the age of PG onset typically is consequent to the age of onset for other psychiatric disorders. More specifically, approximately 98% of individuals with PG have a co-occurring disorder; among these individuals, the co-occurring disorder preceded or emerged simultaneously with PG 76.5% of the time, while PG preceded the co-occurring disorder 23.5% of the time (Kessler et al 2008). This finding indicates that in most cases, psychiatric disorders provide a foundation upon which PG can emerge and evolve. Once developed, PG can stimulate new mental disorders that, in turn, can exacerbate the excessive gambling.

Epidemiological evidence also suggests that it is important for clinicians to evaluate and consider patterns of clinical and sub-clinical signs and symptoms when they develop treatment plans for clients who seek treatment for gambling-related problems. As mentioned previously, PGs are significantly more likely to have co-occurring mental disorders (e.g., mood, anxiety, and personality disorders) and/or substance use disorders (e.g., alcohol and other drug use disorder) compared to non-PGs (Kessler et al 2008; Petry et al 2005). In addition, individuals with a diagnosis of PG also experience distinctive sub-clinical symptom patterns that are associated with symptoms clusters often associated with other diagnostic categories (i.e., dysthymia, anxiety, specific phobias) than non-PGs (Boudreau et al 2009). By identifying and addressing these associated clinical and sub-clinical syndromes, clinicians might be better able to serve their clients and, in turn, observe improved clinical outcomes.

Further, as mentioned previously, epidemiological evidence reveals that PG is an unstable and complex disorder. Consequently, the gambling behavior of clients seeking treatment for gambling disorders typically varies over time – as does the intensity and influence of the co-occurring clinical disorders and shadow syndromes. As a result, it is important for clinicians to understand that, for some people struggling with a gambling disorder, the nexus of PG and its co-occurring symptoms will be chronic. Consequently, those clients who experience chronic PG might intermittently be back and forth into treatment; thus, clinicians need to be prepared to treat PG clients from time to time throughout their lifespan. Next, we will discuss the use of brief gambling screens.

Brief Screening

As we mentioned previously, PGs are likely to experience co-occurring clinical and sub-clinical psychiatric problems. However, few PGs seek treatment for their gambling problems; they do seek help for their co-occurring psychiatric disorders (Kessler et al 2008). There is evidence that simultaneous awareness and treatment for these co-existing disorders is the most efficient way to obtain optimal treatment outcomes (Petry et al 2005; Winters & Kushner 2003). Consequently, it is necessary for clinicians to assess PG and any co-occurring disorders via a rapid assessment. As a result, we encourage clinicians – from gambling specialists to non-specialists who simply need to screen their caseload comprehensively – to consider utilizing a battery of the most efficient screening devices to sustain a comprehensive and careful diagnostic posture. The Brief Bio-social Gambling Screen (BBGS; Gebauer et al 2010) and NODS-CLiP (Toce-Gerstein et al 2009) are two recently developed 3-item screens for identifying gambling disorders. Table 1 summarizes the items for each of these brief-screening instruments.

Table 1. BBGS (Gebauer et al 2010) and NODS-CLiP (Toce-Gerstein et al 2009) items

BBGS	**NODS-CLiP**
• Withdrawal: During the past 12 months, have you become restless, irritable or anxious when trying to stop/cut down on gambling? • Deception: During the past 12 months, have you tried to keep your family or friends from knowing how much you gambled? • Need Money: During the past 12 months, did you have such financial trouble that you had to get help with living expenses from family, friends, or welfare?	• Loss of Control: Have you ever tried to stop, cut down, or control your gambling? • Lying: Have you ever lied to family members, friends or others about how much you gamble or how much money you lost on gambling? • Preoccupation: Have there been periods lasting 2 weeks or longer when you spent a lot of time thinking about your gambling experiences, or planning out future gambling ventures or bets?

The authors of the BBGS sought to create a brief screen for the general population that would have strong psychometric properties, thereby encouraging broad application of the instrument. Using a national sample (i.e., NESARC; Grant et al 2003a), the BBGS allows for more accurate identification of past year PG with three questions than the 10 DSM-IV-TR criteria (American Psychiatric Association 2000) for diagnosing PG. Gebauer et al used the NESARC to develop this optimal brief screen; to date, the NESARC survey, which collected information about DSM-

IV-TR-based PG criteria from the general household population, has produced the largest sample of pathological gamblers drawn from the general household population.

Gebauer et al (2010) argued that the best candidate items for an optimal brief screen would identify correctly the largest proportion of PGs with the fewest false positives. Operationally, this means that the most effective screening items would be those with (1) high sensitivity and (2) high specificity. To develop a brief and efficient public health screen, Gebauer et al applied a four step analytic procedure that included stepwise discriminant function analyses. As Table 1 summarizes, the BBGS includes biologically, psychologically, and socially-oriented criteria: (1) withdrawal; (2) deception; and, (3) needing money (NODS; Gerstein et al 1999).

The NODS-CliP (Toce-Gerstein et al 2009) is another recently developed brief screen. Toce-Gerstein et al (2009) developed the NODS-CLiP using a lifetime time frame for DSM-IV PG adapted items from the NORC Diagnostic Screen for Gambling Disorders (NODS; Gerstein et al 1999). Researchers administered the NODS to participants (N=17,180) in eight different general adult population field studies and targeted participants who endorsed five or more DSM-IV symptoms or signs as the group of PGs to be distinguished from participants who failed to meet these criteria. The researchers developed the NODS-CliP by analyzing how well each possible subset of 2-4 NODS items identified PGs. As Table 1 summarizes, the NODS-CLIP items include: (1) loss of control; (2) lying; and, (3) preoccupation (Toce-Gerstein et al 2009).

As noted, an important difference between the two screens is that the NODS-CliP assesses the presence of lifetime PG, while the BBGS evaluates past-year PG. Using a lifetime context for symptom clustering is clinically and scientifically problematic because this strategy yields more endorsed problems than does a past-year time frame. More importantly, using a lifetime frame for screening is inconsistent with the process of making a clinical diagnosis. For example, research shows that gambling-related problems evidence considerable waxing and waning from year to year (in press-a). As a result, the NODS-CliP might not reflect accurately the current problems that often associate with treatment seeking. As Table 2 shows, the BBGS evidenced better specificity than the NODS-CLiP (0.99 for the BBGS versus 0.88 for the NODS-CLiP) and corresponding positive predictive value advantages (i.e., PPV; 0.37 for the BBGS versus 0.13 for the NODS-CLiP). These psychometric characteristics make the BBGS valuable to clinical

programs because fewer false diagnoses will, in turn, lead to fewer resources being utilized unnecessarily.

Table 2 Comparing the Psychometric Values of the NODS-CLiP (Toce-Gerstein et al 2009) and the BBGS (Gebauer et al 2010)

	NODS-CLiP	BBGS
Sensitivity (the proportion of PGs the screen accurately estimates as PGs)	0.99	0.96
Specificity (the proportion of non-PGs the screen accurately estimates as non-PGs)	0.88	0.99
Positive Predictive Value (PPV; the proportion of estimated lifetime (NODS-CLiP) or past-year (BBGS) PGs that were actually PGs)	0.13	0.37

In addition to screening for PG, we encourage clinicians to screen PG treatment-seekers for co-morbid psychiatric disorders, including substance abuse and mental health disorders (e.g., depression, anxiety). While a full discussion of brief screens for co-occurring disorders exceeds the scope of this review, a series of screens can be found in Shaffer et al (in press-a).

Once clinicians screen a client, most of the treatment options for PG are similar to those used to treat other addictive disorders. For instance, psychological treatments such as cognitive behavioral therapy (e.g., Ladouceur et al 2001; Ladouceur et al 1998; Petry et al 2006; Petry et al 2008; Petry et al 2009) and motivational enhancement (e.g., Petry et al 2008; Petry et al 2009; Wulfert et al 2003) have been shown to have a positive impact on gambling disorders. Further, pharmaceutical treatments such as opioid antagonists (i.e., naltrexone, nalmefene; Grant et al 2006; Kim & Grant 2001; Kim et al 2001) and selective serotonin reuptake inhibitors (i.e., paroxetine, fluvoxamine, citalopram, escitalopram; Black et al 2007; Grant et al 2003b; Grant & Potenza 2006; Hollander et al 2000; Kim et al 2002; Zimmerman et al 2002) have been effective for some PGs. Because these psychological and pharmaceutical treatments are not unique to PG, we are not going to discuss them in further detail. However, in the next section we will discuss a treatment option that is unique to PG – exclusion programs.

Exclusion Programs

When considering a treatment plan for clients struggling with a gambling disorder, it also is important to consider the potential impacts of involuntary and voluntary exclusion programs on individuals' gambling behavior. Casinos and online gambling sites reserve the right to exclude individuals from their premises for any reason (e.g., card counting, suspected money laundering, disorderly conduct, cheating, etc.). Similarly, in some instances, a state gaming board can exclude an individual from gambling at any state-sanctioned gambling venue (e.g., Gaming

Control Board February 1, 2010). These types of exclusion are best described as an involuntary exclusion programs. Conversely, self-exclusion programs are a player-initiated, voluntary form of exclusion that provides gamblers with the opportunity to ban themselves from gambling venues (LaBrie et al 2007b; Ladouceur et al 2000; Nelson et al 2010). Most land-based exclusion programs include risk of criminal prosecution (e.g., trespassing) if excluders violate the agreement. Whether these programs are self-exclusion or involuntary exclusion programs, the gambling provider (e.g., casino) typically carries the burden to identify and keep out those who have been excluded. As a result, there is conceptual confusion with self-exclusion programs: individuals initiate enrollment in these programs but gambling providers or jurisdictions manage the program. Research indicates that self-exclusion programs result in a percentage (22-55%; Ladouceur et al 2000; Ladouceur et al 2007; Nelson et al 2010) of self-excluders entering casinos undetected, thereby breaching their contracts. Clinicians need to be aware of the limitations of self-exclusion programs and treat these agreements as they do no-suicide contracts. Like no-suicide agreements, self-exclusion agreements are pseudo-legal documents that offer patients or patrons some assistance with gaining self-control (e.g., Goin 2003).

Despite these limitations, self-exclusion programs have shown some efficacy. There are two longitudinal studies that provide evidence about such efficacy. One longitudinal study of a self-exclusion program reported that self-excluders' urge to gamble, number of gambling problem symptoms, and intensity of negative consequences of gambling significantly decreased as their sense of control significantly increased between the intake interview and six month follow-up (Ladouceur et al 2007). Another longitudinal study revealed that most gamblers who signed up for a lifetime exclusion ban reduced their gambling and had significantly fewer gambling-related problems at follow-up; the rate of PG among self-exclusion program participants declined from 79% at enrollment to 15% at follow-up (Nelson et al 2010). It is important to note that self-exclusion might not be the cause of these promising findings; instead, it is possible that the act of self-excluding can reflect a motivation to stop gambling. In other words, those motivated to change their gambling behavior are more inclined to sign up for self-exclusion. Next, we will conclude this paper with a consideration for future gambling research – the identification of PG-related behavior markers.

Toward the Future: Behavioral Markers for Gambling Disorders

Now that researchers have established a better understanding of the diagnosis, etiology and trajectory of PG, research needs to begin focusing on the development of early prevention and effective treatment programs for gambling disorders. Identifying behavioral markers that are associated with PG will provide the foundation for this course of action. Identifying behavioral markers is a process similar to the sequence of research that leads to the identification of biomarkers. Biomarkers are measurable biological characteristics that can serve as indicators of normal or pathogenic processes in the body or as tools to track pharmacological responses to therapeutic drugs (Vanderstichele et al 2008). The objective of biomarker research is to identify specific markers for use in improving early diagnosis, surveying therapeutic outcomes, and facilitating the development of novel drug or treatment candidates (De Bock et al 2010).

Researchers have used marker development techniques with other disorders. While a full discussion of these advances exceeds the scope of this review, it is worth noting that marker identification work is proceeding apace for cancer and Alzheimer's disease. Efforts to combat cancer by identifying biomarkers have evidenced striking advances. Completion of the human genome sequence in 2001 sparked a dramatic increase in knowledge about the genetic causes of cancer, and there have been parallel advances in technologies that allow for the rapid and accurate measurement of cancer-related genes and their respective proteins (e.g., mass spectrometry; Hudson 2009). Behavioral scientists can use similar marker development strategies to identify behavioral markers for gambling and other mental disorders.

Although the identification of behavioral markers for gambling disorders is in its infancy, studies focusing on the actual betting behavior of online gamblers have been beneficial to begin identifying potential behavioral markers. These studies have identified a number of potential PG-related behavioral markers, including closing accounts (Braverman & Shaffer 2010; Xuan & Shaffer 2009), self-limiting gambling expenditures (Nelson et al 2008), exceeding company-imposed deposit limits (Broda et al 2008), exhibiting intense betting soon after enrollment (LaBrie & Shaffer in press) and extreme betting patterns (e.g., making more and larger bets, betting more frequently; LaBrie et al 2008; LaBrie et al 2007a; LaBrie & Shaffer in press; LaPlante et al 2009a; LaPlante et al 2008b). Taken together, these studies of actual Internet gambling behavior provide some potential behavioral markers for gambling disorders. Overall, these studies illustrate that a high level of involvement (e.g., reacting to wins and losses; time

and money spent, etc.) is more indicative of disordered gambling than participation in any particular game.

The next step toward the development of sensitive, specific and reliable markers that can predict the emergence of gambling disorders is to utilize probable markers as building blocks to create an algorithm to predict, within the context of longitudinal studies, the development of PG. Researchers should use these potential markers to prospectively identify gamblers who are at higher risk for the development of PG. One strategy would be to use a large sample of Internet gambling subscribers who have not yet reached a discernable gambling-related outcome (e.g., closing their account, setting limits on gambling behavior) and a combination of markers to predict which ones will reach that outcome. Creating an algorithm to predict PG among Internet gamblers would allow Internet gambling sites to provide evidence-based harm reduction services to their customers who evidence markers for PG. For instance, Internet gambling sites could potentially issue warnings to customers, provide PG-related intervention and/or exclude customers.

Conclusions

The empirical research focusing on PG has increased dramatically during the past 10-20 years. As a result, interested stakeholders have an evolving and more advanced understanding of the epidemiology, trajectory, diagnosis, and treatment of gambling-related disorders. We encourage clinicians to utilize evidence-based diagnostic and treatment approaches to improve treatments for clients struggling with gambling-related disorders. Most notably, we encourage clinicians to be cognizant of the likelihood of co-occurring clinical and sub-clinical mental disorders with PG and to use a treatment "cocktail" approach (i.e., multifaceted and layered) for treatment seeking clients struggling with gambling addiction.

Despite the advances in PG-related research and treatment, there is still much to be learned about gambling disorders. For example, one promising next step for gambling-related research is to identify and test behavioral markers that can reliably predict the development of gambling disorders. We encourage scientists to continue advancing the gambling-related research field and for clinicians serving PG clients to utilize the findings of emerging research to best serve their clients. "Ultimately, improving treatment outcomes for addiction might be achieved primarily by revising and reordering existing clinical activities and developing new, unobtrusive diagnostic

tools. This observation leads to the interesting and promising conclusion that the necessary tools for improving addiction treatment might be already available. All that is required to enhance the use of these devices is a rethinking of addiction" (Shaffer et al 2004c, p. 372).

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