

A pathways model of problem and pathological gambling

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ABSTRACT

At the moment, there is no single conceptual theoretical model of gambling that adequately accounts for the multiple biological, psychological and ecological variables contributing to the development of pathological gambling. Advances in this area are hampered by imprecise definitions of pathological gambling, failure to distinguish between gambling problems and problem gamblers and a tendency to assume that pathological gamblers form one, homogeneous population with similar psychological principles applying equally to all members of the class. The purpose of this paper is to advance a pathways model that integrates the complex array of biological, personality, developmental, cognitive, learning theory and ecological determinants of problem and pathological gambling. It is proposed that three distinct subgroups of gamblers manifesting impaired control over their behaviour can be identified. These groups include (a) behaviourally conditioned problem gamblers, (b) emotionally vulnerable problem gamblers and (c) antisocial, impulsivist problem gamblers. The implications for clinical management are discussed.

KEYWORDS Addiction, biology of gambling, impulsivity, pathological gambling, pathway model.

INTRODUCTION

Pathological gambling is defined as 'persistent and recurrent maladaptive gambling behaviour' characterized by an inability to control gambling, leading to significant deleterious psychosocial consequences: personal, familial, financial, professional and legal (APA 1994). The literature contains numerous studies describing prevalence estimates, demographic and clinical profiles, personality traits and neurobiological substrates presumed to play an integral role in the development of impaired control in pathological gambling. However, to date there are no papers describing an empirically validated theoretical model of pathological gambling that effectively integrates into a coherent conceptual framework, the complex array of biological, psychological and ecological factors to explain the aetiology of the disorder (Brown 1988; Shaffer & Gambino 1989; Ferris, Wynne & Single 1998; Blaszczynski 1999). As Shaffer & Gambino (1989) suggest, further advances in the understanding and treatment of pathological gambling are dependent upon

the development of a comprehensive explanatory model of gambling and pathological gambling behaviour, which integrates knowledge sourced from research, theory and practice. The purpose of this paper is to present a conceptual model, delineating a series of three discrete pathways leading to the development of distinct subgroups of pathological gambling.

The pathways model is predicated on the argument that the quest to impose one theoretical model to apply equally and validly to all pathological gamblers is a misguided venture. An alternative and more productive approach is to acknowledge the existence of specific subtypes of gamblers, each influenced by different factors yet displaying similar phenomenological features. Clinical wisdom has long recognized that, although symptoms of depression, substance use, impulsivity and antisocial type behaviours are observed typically in pathological gamblers, the role and implication of these variables in the aetiology and management of the disorder varies widely for each case. For example, three-quarters of problem gamblers manifest symptoms of depression

(Blaszczyński & McConaghy 1988; Linden, Pope & Jonas 1986). For some, gambling is used as a means to induce dissociation to reduce or escape states of chronic depression (Jacobs 1986; Blaszczyński & McConaghy 1989) while, for others, depression appears to represent the emotional reaction to financial crises and other problems created by excessive gambling behaviours. Each has its own significant implication in determining appropriate interventions for clinical management.

PROBLEM GAMBLING VERSUS GAMBLING PROBLEMS

Historically, the terms 'compulsive gambler' and 'pathological gambler' have been used interchangeably to denote individuals who report uncontrollable urges to gamble. Moran (1970) argued for the exclusive use of the term 'pathological', a term subsequently adopted as the official psychiatric classification on the basis that a 'compulsion' denotes an ego dystonic behaviour. In contrast, most gamblers with problems view the behaviour as ego syntonic, with little desire to cease despite the adverse consequences.

More recently, alternative terms have been employed: 'problem', 'at-risk', 'in-transition', 'disordered', 'excessive' and 'Level 2' gamblers. Each utilizes different criteria and classification schemes. For example, Abbott, Palmisano & Dickerson (1995) classify gamblers as either 'excessive' or 'normal', based on amount of time, expenditure and number of trips to gambling venues. In contrast, Winters, Stinchfield & Fulkerson (1993) employed a complicated classification scheme based on symptom count and frequency of gambling. Others use symptom count alone and differing categories (e.g. Fisher 1993 (social gambler/pathological gambler); Gupta & Derevensky 1998b (social/problem/pathological); Shaffer *et al.* 1994 (non-pathological/in-transition/pathological); Vitaro, Arseneault & Tremblay, 1997 (recreational/low problem/high problem).

The Victorian Casino & Gaming Authority (VCGA) (1997) argued that the presence of harm rather than symptom count should be used to define problem gambling. This position is exemplified by the VCGA's consensus definition: "Problem gambling" refers to the situation when a gambling activity gives rise to harm to the individual player, and/or to his or her family, and may extend into the community' (VCGA 1997; p. 106), and by the definition advanced by Ferris *et al.* (1998): 'Problem gambling is excessive gambling behaviour that creates negative consequences for the gambler, others in his/her social network, and for the community' (p. 58). Under both definitions, the presence of harm dictates diagnosis, an approach that Walker (1998) severely

criticizes for defining 'harm' based on subjective value judgements. In illustration, Walker (1998) states that, according to the VCGA's definition, a person would be classified a problem gambler if his/her spouse had strict religious objections to gambling and was distressed by the mere purchase of \$2.00 weekly lottery tickets. Under these circumstances, gambling may cause marital discord (harm) but it remains questionable as to whether the gambler should be considered a pathological gambler according to DSM-IV criteria.

Defining groups based on subjective criteria results in expanding the population pool of potential clients by including gamblers with problems in the same category as pathological gamblers, resulting in increased Type I errors. Gamblers experiencing gambling-related problems are thus misclassified as those who are unable to control and regulate impulses to gamble.

From early intervention and public health perspectives, this approach may be associated with certain advantages in encouraging gamblers to enter counselling at an earlier stage of progress. A negative aspect of this trend, however, is that it confuses concepts of gambling problems and pathological gambling, ultimately leading to the position where problem and non-problem gamblers are merged into one heterogeneous grouping. As a consequence of this heterogeneity, contradictory and confusing results have been reported in the research literature, and this confusion is further reflected in varied approaches to treatment and the absence of accepted 'best practice' guidelines.

Gambling problems may be defined as a friction or difficulty in any area of functioning that results from some element of gambling behaviour. Typically, gambling problems may arise as a result of differences of opinion regarding amounts potentially risked or time spent away from home/family in the absence of any excessive financial losses relative to disposable income, preoccupation with gambling absent impaired control or other adverse consequences. This situation, no doubt, is similar to the complaints often heard by the spouse of golfers or other ardent hobbyist.

In contrast, the defining feature of a problem gambler is not only the emergence of negative consequences but also the presence of a subjective sense of impaired control, construed as a disordered or diseased state that deviates from normal, healthy behaviour. Impaired behavioural control, defined by repeated, unsuccessful attempts to resist the urge in the context of a genuine desire to cease, is the central, diagnostic and foundational feature of pathological gambling.

While several researchers have attempted to identify typologies of gamblers (Moran 1970; Kusyszyn 1972), most studies tend to neglect the difficult task of clustering subjects into homogeneous samples based on actiology,

personality, gender or form of gambling. Consequently, as Ferris *et al.* (1998) observe in their review, current theoretical models with their own strengths and weaknesses have contributed to our understanding of the aetiology of problem gambling, but none are sufficiently comprehensive in scope to explain all aspects of gambling. There is little agreement on typologies beyond the view expressed by Jacobs (1986) and Blaszczynski, Winter & McConaghy (1986) that there are at least two subgroups of gamblers: one chronically understimulated and the other, overstimulated.

CURRENT MODELS OF GAMBLING

Popular models of pathological gambling include the following: addictions (Jacobs 1986; Blume 1987), psychodynamic (Bergler 1958; Rosenthal 1992; Wildman 1997), psychobiological (Blaszczynski *et al.* 1986; Carlton & Goldstein 1987; Lesieur & Rosenthal 1991; Rugle 1993; Comings *et al.* 1996), behavioural (Anderson & Brown 1984; McConaghy *et al.* 1983), cognitive (Sharpe & Tarrrier 1993; Ladouceur & Walker 1996) and sociological (Rosecrance 1985; Ocean & Smith 1993) approaches.

These models are not mutually exclusive but, rather, share many common elements. For example, principles of reinforcement derived from learning theory are incorporated as core elements in addictions, behaviour therapy and biological models in explaining persistence in gambling. Essentially, each of the above models acknowledges the interaction of key biopsychosocial variables in the aetiological process but emphasizes a different set of operations to account for the progression from initial participation to impaired control and persistence.

The pervasive but faulty assumption embedded within each model is that pathological gamblers form a homogeneous population, and that theoretically derived treatments can be applied effectively to all pathological gamblers irrespective of gambling form, gender, developmental history or neurobiology. Learning theories (Dickerson 1979) invoke the operation of fixed and variable schedules of reinforcement but fail to explain why only a small proportion of the total population of gamblers lose control. Similarly, cognitive theories (Sharpe & Tarrrier 1993; Ladouceur & Walker 1996) emphasize the role of distorted and irrational cognitive schemas but lack empirical evidence establishing that these are of causal significance and not secondary cognitive dissonance effects. Psychodynamic approaches (Lesieur & Rosenthal 1991) focus on intrapsychic processes associated with attempts to deal with unresolved conflicts but see it variably as a compulsive neurosis or impulse disorder along the lines of addictions and perversions.

Conceptually, pathological gambling is perceived as either a categorical disorder or as an end-point on a continuum of gambling involvement. Both the psychodynamic and the disease model of addiction with its biological derivatives argue that pathological gamblers are categorically distinct from their non-pathological counterparts. This has led to the search for qualitative similarities and differences between social and pathological gamblers and other substance use disorders. These include aspects of personality traits (Blaszczynski, Buhrich & McConaghy 1985; McCormick *et al.* 1987; Castellani & Rugle 1995), co-morbidity (Hall *et al.* 2000; Slutske *et al.* 2000; Langenbucher *et al.* 2001) and biological correlates (Comings *et al.* 1996; Rugle & Melamed 1993).

Those adhering to a dimensional view suggest that pathological gamblers do not manifest qualitatively different and defining features except amount and time spent gambling (Walker 1992). Pathological gamblers are classified according to an arbitrary cut-off point set along the dimensional continuum. The concept of subgroups is discounted or neglected.

However, as described below, converging lines of research are pointing to differences between populations supporting the existence of distinct subgroups of pathological gamblers (Rugle & Melamed 1993; Steel & Blaszczynski 1996; Gonzalez-Ibanez, Jimenez & Aymami 1999).

GAMBLERS WITH MOOD DISORDERS

Blaszczynski *et al.* (1986) and Blaszczynski (1988) have argued that there exist at least two subsets of gamblers who differentially seek to reduce or augment arousal states. Reducers suffer anxiety and select low skill activities to narrow their focus of attention and produce states of dissociation, while augmenters may choose high skill games to overcome states of dysphoria, a view consistent with Jacobs' general theory of addictions model (Jacobs 1986). Studies have reported a high prevalence of mood disorders, particularly anxiety and/or depression, among problem and pathological gamblers (Black & Moyer 1998; Beaudoin & Cox 1999; Vitaro, Arseneault & Tremblay 1999). In a sample of African American elderly people, Bazargan, Barzargan & Akanda (2001) found a statistically significant positive relationship between gambling behaviours and levels of anxiety.

Affective states may differ by gender. Marks & Lesieur (1992) reviewed the literature and concluded that female gamblers differed systematically from male gamblers in relation to manifest psychological distress. In a study of Gamblers Anonymous (GA) members, Getty, Watson & Frisch (2000) found that GA members manifested significantly higher levels of depression than controls, and

female GA members reported more depression than males. Similarly, in a study of 817 high school students, Gupta & Derevensky (1998a) found that adolescent problem or pathological gamblers exhibited evidence of hyper- or hypo-arousal, greater emotional distress, higher levels of dissociation and higher rates of co-morbidity than non-problem gamblers. However, anxiety (hyperarousal) and dissociation emerged as the highest predictors for males and depressed mood, dissociation and use of stimulants were significantly predictive of female problem and pathological gamblers.

Depression is a common co-morbid condition found among pathological gamblers but, within this cohort, a number of important subtypes have been reported. Graham & Lowenfeld (1986) identified a depressive reaction personality subtype using the MMPI, while both McCormick (1994) and Castellani & Rugle (1995) found a chronic dysthymic subgroup with a depressogenic cognitive style, which is prognostic for predicting relapse. Pathological gamblers within the depressive category, particularly females, were reportedly more likely to choose modes of gambling that were socially isolating, repetitive, or monotonous to modulate this mood state (Rosenthal & Lesieur 1992; McCormick 1994).

Boredom is related to aspects of depression, and it has been demonstrated that pathological gamblers have poor tolerance for boredom (Blaszczynski, McConaghy & Frankova 1990). McCormick (1994) described a hyperactive subtype, characterized as chronically understimulated and constantly searching for relief from boredom. Lesieur and colleagues (Lesieur & Blume 1991; Rosenthal & Lesieur 1992) referred to these gamblers as 'action seekers'. Not only were these individuals chronically bored, but even the action provided by gambling became boring unless it was novel, varied and capable of producing increasing levels of arousal. These action-seekers sought big payoffs, played competitive, skill-oriented forms of gambling and possessed a need to impress.

Action-seeking gamblers have also been characterized by high energy levels, a need for stimulating situations, hyperactive, impulsive, unable to endure emotional tensions, unable to relax and hypomanic (Custer 1984; Peck 1986; McCormick & Taber 1987). Those falling within this profile tend toward activities considered highly stimulating such as horse racing (Blaszczynski *et al.* 1986) and stand in contrast to the depressed profile gamblers who typically prefer slot machines (Blaszczynski *et al.* 1986).

IMPULSIVE GAMBLERS

There is a growing body of evidence to suggest that pathological gambling may be associated in some individuals

with high levels of trait impulsivity, and that impulsivity has a direct relationship to the severity of problems in both gambling and non-gambling domains and responses to treatment (Moran 1970; McCormick *et al.* 1987; Rugle & Melamed 1993; Castellani & Rugle 1995; Steel & Blaszczynski 1996; Gonzalez-Ibanez *et al.* 1999).

Studies have identified an 'antisocial impulsivist' subtype of gamblers who demonstrate elevated levels of impulsivity that is highly correlated with measures of psychopathology and clinical criteria for antisocial personality disorder (Blaszczynski, Steel & McConaghy 1997; Steel & Blaszczynski 1996). These gamblers exhibit a family history of problem gambling, early onset, more severe levels of gambling, a history of suicidal ideation and/or attempts, co-morbid substance dependency, anti-social and narcissistic traits, affective instability, widespread dysfunction in non-gambling related areas and unresponsiveness to treatment (Blaszczynski, Steel & McConaghy 1997; Steel & Blaszczynski 1996).

BIOLOGICAL CORRELATES OF GAMBLING

Strengthening the concept of the existence of defined subgroups of gamblers is the fascinating work within the field of biochemistry (Carrasco *et al.* 1994; Moreno, Saiz-Ruiz & Ipoez-Ibor 1991) and genetics (Comings *et al.* 1996), tentatively linking receptor genes and neurotransmitter dysregulation to reward deficiency, arousal, impulsivity and pathological gambling. Preliminary evidence supports the hypothesis that serotonin (mood regulation), norepinephrine (mediating arousal) and dopamine (reward regulation) may all play a role in impulsivity, mood disorders and impaired control (Lopez-Ibor 1988; Roy, De Jong & Linnoila 1989; Moreno *et al.* 1991; DeCaria *et al.* 1996; Bergh *et al.* 1997).

Genetic studies have also reported that, similar to substance users, pathological gamblers are significantly more likely than controls to possess the dopamine D2A1 allele receptor gene (Comings *et al.* 1996), which prove a significant risk factor in pathological gambling. This genetic variant has also been found more often in individuals with impulse control disorders and has been associated with reduced D2 receptor density and deficits in dopaminergic reward pathways. Of note, 76.2% of pathological gamblers who were co-morbid alcohol abusers carried the gene compared to 49.1% of males without co-morbid alcohol abuse or dependency. It is hypothesized that a lack of D2 receptors cause individuals to seek pleasure-generating activities, placing them at high risk for multiple addictive, impulsive and compulsive behaviours, including substance abuse, binge eating, sex addiction and pathological gambling (Blum *et al.* 2000).

Thus, the genetic research suggests that the drive toward intense and, sometimes, detrimental pleasure-seeking is biologically prescribed, though the choice of behaviour differs by individual.

The discovery of a link between the D2A1 allele and impulsive-addictive-compulsive behaviours such as pathological gamblers may also have implications for pharmacological treatment. Blum *et al.* (1996) speculate that pharmacological sensitivity to dopaminergic agonists may be determined in part by DRD2 genotypes and that carriers of the A1 gene would be more responsive to D2 antagonists. Thus, it is possible that pathological gamblers carrying the D2A1 allele would respond favourably to D2 agonists such as bromocryptine, bupropion and n-propyl-nor-apomorphine.

It is possible that biologically based traits of impulsivity may create a subset of gamblers who manifest differential responses to reward and punishment, characterized by a marked propensity to seek out rewarding activities, an inability to delay gratification, a dampened response to punishment and failure to modify behaviour because of adverse consequences.

There is consistent evidence emerging to support the argument that subgroups of problem and pathological gamblers with distinct clinical features and aetiological processes. The first group lacks psychiatric pathology but falls prey to a highly addictive schedule of behavioural reinforcement. The second group is biologically and emotionally vulnerable, characterized by high levels of depression and/or anxiety, while the third group, also possessing these vulnerabilities, is decidedly impulsive, antisocial and typically dually addicted.

A PATHWAYS MODEL OF GAMBLING

This paper postulates a preliminary model that attempts to integrate biological, personality, developmental, cognitive, learning theory and environmental factors described in the literature into a theoretical framework. The model postulates three major pathways culminating in pathological gambling; each pathway is associated with specific vulnerability factors, demographic features and aetiological processes. All pathways contain certain processes and symptomatic features in common but are distinguishable by empirically testable factors.

The starting block common to the three pathways must be availability and access to gambling. Ecological determinants are those that relate to public policy and regulatory legislation that create and foster an environment in which gambling is socially accepted, encouraged and promoted. Epidemiological surveys indicate that availability and access to gambling facilities is associated with a higher incidence of pathological gambling

(Abbott & Volberg 1996; Volberg 1996; Grun & McKeigue 2000).

The next process commonly applicable to all gamblers in the pathway is the influence of classical and operant conditioning leading to increasing participation and the development of habitual patterns of gambling, and cognitive process resulting in faulty beliefs related to personal skill and probability of winning.

Studies have demonstrated an association between subjective excitement (Dickerson, Hinchey & Fabre 1987), dissociation (Jacobs 1986), increased heart rate (Anderson & Brown 1984; Leary & Dickerson 1985; Brown 1988; Griffiths 1995) and gambling. Operant conditioning occurs when intermittent wins delivered on a variable ratio produce states of arousal often described as equivalent to a 'drug-induced high', while with repeated pairings, this arousal is also classically conditioned to stimuli associated with the gambling environment (Dickerson 1979; Sharpe & Tarrrier 1993). In addition, negative reinforcement is produced when aversive anxiety states and depression are reduced by the excitement of gambling, further increasing the probability of continued gambling. Eventually, a habitual pattern of gambling emerges.

From a neo-Pavlovian perspective, a 'neuronal model' of the habitual behaviour is built through a process of cortical excitation (McConaghy 1980). Once triggered by gambling-related cues, the behaviour completion mechanism underlying this neuronal model is stimulated to produce a drive to carry out the habitual behaviour to completion (McConaghy *et al.* 1983). Attempts to resist completing the habit provoke a state of aversive arousal experienced as a drive, compulsion or urge to carry out the behaviour. This state persists as a compulsive drive to carry out the habitual pattern of behaviour through to its completion.

As the frequency of gambling progresses, strong biased and distorted cognitive schemas appear. These schemas shape beliefs surrounding attribution, personal skill and control over outcome, biased evaluations, erroneous perceptions, superstitious thinking and probability theory (see Griffiths 1995; Ladouceur & Walker 1996 for a comprehensive review of these processes). The potency and pervasiveness of distorted and irrational cognitive belief structures strengthen with increasing levels of involvement in gambling (Griffiths 1990, 1995).

Invariably, due to the nature of gambling odds, losing streaks occur and losses begin to accumulate. Pressure mounts to chase losses through further gambling as debts rapidly escalate (Lesieur 1984), and the gambler desperately tries to extricate him/herself from a deteriorating financial predicament. By this stage, diagnostic indicators for pathological gambling become readily identifiable.

PATHWAY 1: BEHAVIOURALLY CONDITIONED PROBLEM GAMBLERS

Principles of learning theory and cognitive processes are instrumental in fostering a loss of control for all pathological gamblers. However, it is argued that there is subset of 'behaviourally conditioned gamblers' who at times may meet formal criteria for pathological gambling but who are characterized by an absence of any specific premorbid feature of psychopathology. Essentially, these gamblers fluctuate between the realms of regular/heavy and excessive gambling because of the effects of conditioning, distorted cognitions surrounding probability of winning, and/or a series of bad judgements or poor decision-making rather than because of impaired control. As demonstrated in Fig. 1, members of this subgroup may be preoccupied with gambling, engage in chasing, abuse alcohol and exhibit high levels of depression and anxiety in response to the financial burden imposed by their behaviour. Most importantly, these symptoms are the consequence not the cause of patterns of repeated excessive gambling behaviour.

Entry into this subgroup may occur at any age and may be precipitated by exposure to gambling through chance, family members or peer groups. This subgroup reports the least severe gambling and gambling-induced difficulties of any pathological gamblers, and they do not manifest gross signs of major premorbid psychopathology, substance abuse, impulsivity, erratic or disorganized behaviours.

The profile of this subgroup is characteristic of the 'cluster one' sample identified in Gonzalez-Ibanez *et al.*'s (1999) cluster analytical study of 60 male fruit machine gamblers, and of the controlled gamblers in Blaszczynski's (1988) treatment outcome study. Subjects in both studies were found to display minimal levels of psychopathology or levels that fell to within normal limits following treatment.

Placed at the low end of the pathological dimension, they fluctuate between heavy and problem gambling, demonstrate motivation to enter treatment, comply with instructions and may successfully re-establish controlled levels of gambling post-treatment. It is proposed that counselling and minimal intervention programmes benefit this subgroup.

PATHWAY 2: EMOTIONALLY VULNERABLE PROBLEM GAMBLERS

For this subgroup, the identical ecological determinants, conditioning processes and cognitive schemas are present. However, in addition, these gamblers present with premorbid anxiety and/or depression, a history of poor coping and problem-solving skills, and negative

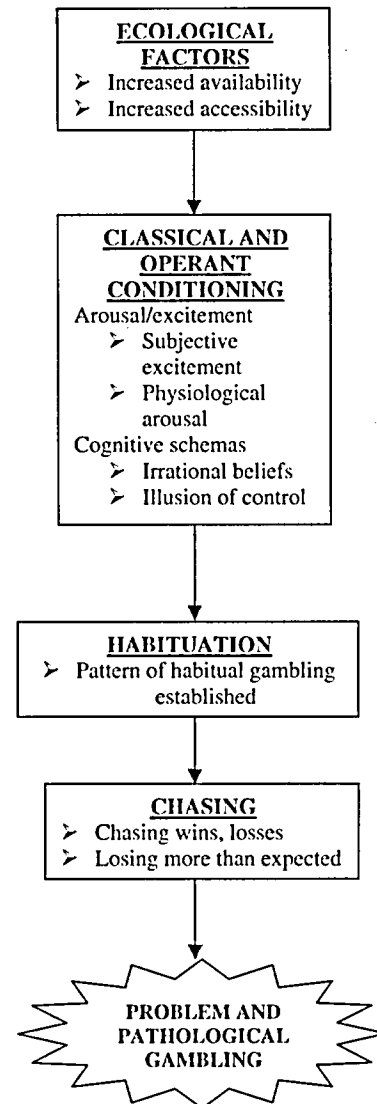


Figure 1 Integrated model of problem gambling, Pathway 1

family background experiences, developmental variables and life events. As Fig. 2 shows, these factors each contribute in a cumulative fashion to produce an 'emotionally vulnerable gambler', whose participation in gambling is motivated by a desire to modulate affective states and/or meet specific psychological needs.

Jacobs (1988), Lesieur & Rothschild (1989) and Gambino *et al.* (1993) each reported strong evidence that a family history of pathological gambling is an important predisposing risk factor for children. In Gambino *et al.*'s (1993) study, subjects with parents identified as problem gamblers were three times more likely to be problem gamblers; that figure increased to 12 times the risk when both parents and grandparents were problem gamblers. A family history of problem gambling may be one risk factor; however, it cannot be construed as a sufficient cause alone.

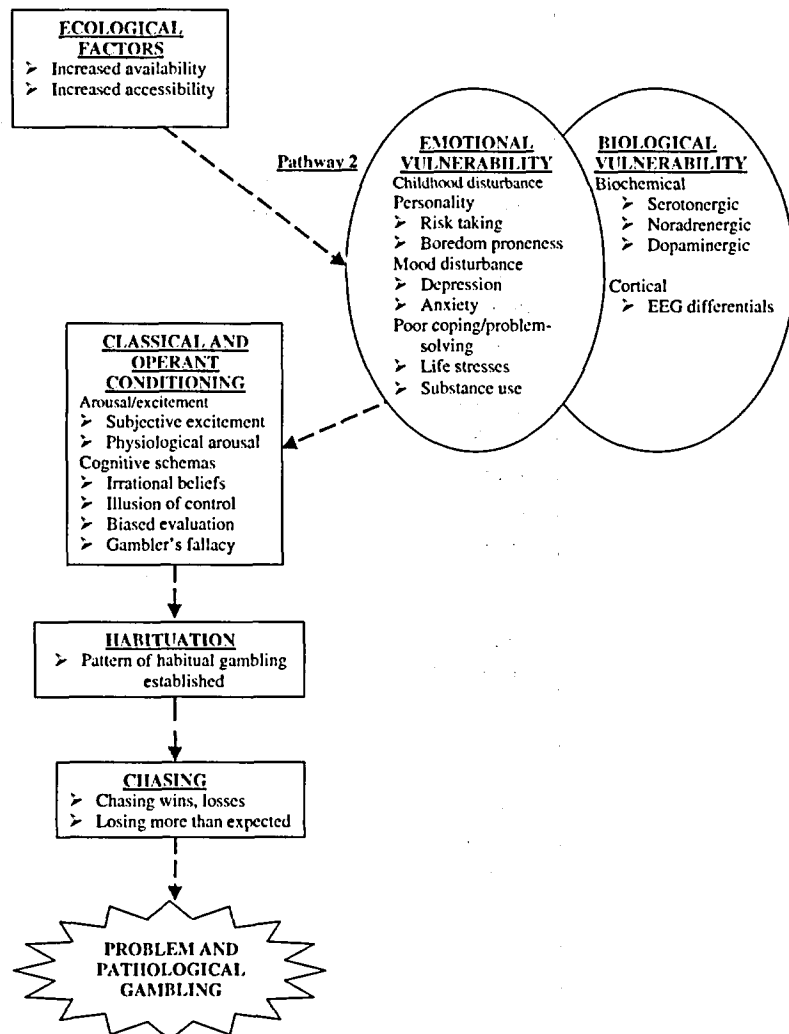


Figure 2 Integrated model of problem gambling, Pathway 2

Jacobs (1986), in his general theory of addiction, postulated that certain personality characteristics and life events, interacting with physiological states of arousal, are instrumental in influencing the development of gambling problems. He states that excessive gambling is produced by the interaction of two sets of predisposing factors; abnormal physiological resting states of hyper- or hypo-arousal states, and a history of negative childhood experiences. Personal vulnerability is linked to childhood experiences of inadequacy, inferiority, low self-esteem and rejection (McCormick *et al.* 1987; McCormick, Taber & Kruedelbach 1989). In this context, gambling is viewed as a means of producing emotional escape through the effect of dissociation on mood alteration and narrowed attention (Anderson & Brown 1984; Jacobs 1986). This subgroup of gamblers displays higher levels of psychopathology, in particular depression, anxiety and alcohol dependence. Females show a preference over males for low-skill gaming devices such as slot machines, video-draw poker and fruit machines, whereas males are

more likely to engage in table games and sports betting, which generate higher levels of arousal.

Gonzalez-Ibanez *et al.*'s (1999) 'cluster two' sample provides evidence in support of a subgroup of emotionally vulnerable gamblers, a group occupying an intermediary position between the less severe cluster one and the more dysfunctional cluster three sample. Similarly, the factorial structure reported by Steel & Blaszczynski (1996) identified one group, comprised primarily of females, that loaded highly on a psychological distress factor and was characterised by higher scores on psychological distress indices, history of depression, suicidal attempts and family psychiatric history. Compared to males, these female gamblers were older, obtained significantly higher Beck depression and anxiety inventory scores, and indicated a stronger preference for slot machines even though their South Oaks gambling screen scores were identical. They also reported high levels of impulsivity that equalled those for males but lower levels of financial debt.

The psychological profile is also exemplified by the abstinent gamblers in Blaszczynski's (1988) and Blaszczynski, McConaghy & Frankova's (1991) 2–5-year treatment outcome study, involving a sample of 63 gamblers. On psychological measures, abstinent subjects showed an intermediate position between the more adjusted controlled and severely disturbed uncontrolled gamblers in terms of psychopathology. Because of their negative developmental history and poor coping skills, these subjects were considered too fragile to maintain sufficient control over behaviour to permit controlled gambling.

Figure 2 illustrates the essential differences between the first two pathways. Pathway 1 gamblers gamble initially for entertainment or socialization, facilitated by access and availability. In contrast, Pathway 2 gamblers are emotionally vulnerable as a result of psychosocial and biological factors, utilizing gambling primarily to relieve aversive affective states by providing escape or arousal. Once initiated, a habitual pattern of gambling fosters behavioural conditioning and dependence in both pathways. However, psychological dysfunction in Pathway 2 gamblers makes this group more resistant to change and necessitates treatment that addresses the underlying vulnerabilities as well as the gambling behaviour.

PATHWAY 3: 'ANTISOCIAL IMPULSIVIST' PROBLEM GAMBLERS

The third subgroup of pathological gamblers describes highly disturbed individuals with substantial psychosocial interference from gambling and characterized by signs suggestive of neurological or neurochemical dysfunction. Similar to Pathway 2 gamblers, this subgroup possesses both psychosocial and biologically based vulnerabilities. However, this group is distinguished by features of impulsivity and antisocial personality disorder (Steel & Blaszczynski 1996; Blaszczynski *et al.* 1997) and attention deficit (Rugle & Melamed 1993), manifesting in severe multiple maladaptive behaviours and impulsivity affecting many aspects of the gambler's general level of psychosocial functioning (Fig. 3).

Clinically, gamblers with a background history of impulsivity engage in a wider array of behavioural problems independent of their gambling, including substance abuse, suicidality, irritability, low tolerance for boredom and criminal behaviours. In an interactive process, the effect of impulsivity is aggravated under pressure and in the presence of negative emotions. Poor interpersonal relationships, excessive alcohol and poly drug experimentation, non-gambling-related criminality and

a family history of antisocial and alcohol problems are characteristic of this group. Gambling commences at an early age, rapidly escalates in intensity and severity, may occur in binge episodes and is associated with early entry into gambling-related criminal behaviours. These gamblers are less motivated to seek treatment in the first instance, have poor compliance rates and respond poorly to any form of intervention. Blaszczynski *et al.* (1997) have labelled these gamblers the 'antisocial impulsivist' subtype.

In support of this clinical description, Steel & Blaszczynski (1996) investigated the relationship between impulsivity, antisocial features, and gambling in a cohort of 115 gamblers. This study replicated earlier findings, showing levels of psychological distress to be significantly correlated with impulsivity and antisocial personality characteristics, a finding consistent with McCormick's (1994) observation that pathological gamblers with concurrent substance abuse problems were more impulsive and manifest higher levels of affective disturbance than substance abusers. Gamblers in Gonzalez-Ibanez *et al.*'s (1999) 'cluster three' group exhibited similar features: higher levels of gambling problems, impulsivity, thrill and adventure seeking, disinhibition and susceptibility to boredom than other gamblers.

The hyperactive subtype of attention deficit hyperactivity disorder is a developmental disorder characterised by impulsivity that commences in childhood and is often found in conduct disorder and antisocial personality behaviours. Goldstein and colleagues (Goldstein *et al.* 1985; Carlton *et al.* 1987) reported differential patterns of EEG activity and self-reported symptoms that paralleled those found in childhood attention deficit disorder in a series of small samples of recovered gamblers.

Carlton & Manowitz (1994), in an extension of their work, found high levels of impulsivity in 12 members of Gamblers Anonymous but impulsivity scores of these subjects were not related to personal or social disruption due to gambling. However, as the authors acknowledge, the lack of correlation may be a Type II error given the low power associated with the small sample size. Similarly, in a study of substance abusers, pathological gamblers and controls, Petry (2001) found a significant association between impulsivity, substance abuse and pathological gambling.

Rugle & Melamed (1993) administered several neuropsychological measures of attention deficits to 33 male pathological gamblers and a similar number of normal controls. Significant differences between the samples on measures of executive functions led these authors to conclude that childhood differences in behaviours related to overactivity, destructibility and difficulty inhibiting conflicting behaviours were of primary importance in differ-

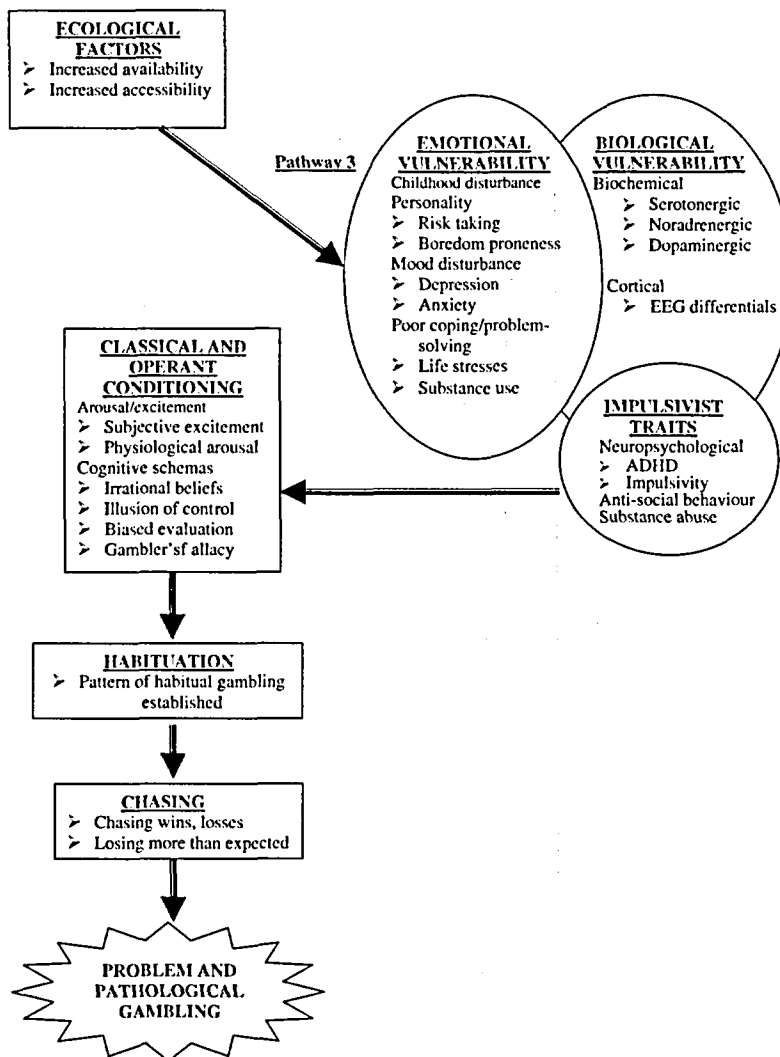


Figure 3 Integrated model of problem gambling, Pathway 3

entiating gamblers from controls. Ruge & Melamed (1993) concluded that there is some support for the notion that at least attention deficit-related symptoms reflecting traits of impulsivity are present at childhood and predate the onset of pathological gambling behaviour. This biological vulnerability weakens behavioural control not only in the domain of gambling but also in other areas of life. This gives rise to the hypothesis that impulsivity proceeds and is independent of gambling, and functions as a good predictive factor for severity of involvement in at least a subgroup of gamblers.

In summary, Fig. 4 illustrates the integrated pathways model, in which problem gambling is initiated due to ecological factors, proceeds through one of three distinct pathways, and ultimately converges at the level of classical and operant conditioning that fosters habituation, chasing, and problem and pathological gambling behaviour.

DISCUSSION

The majority of studies report findings that are based on samples of gamblers compared to control groups. Until recently, little consideration appears to have been directed beyond gender and age toward determining whether or not intragroup differences exist among pathological gamblers. In most cases samples are regarded as homogeneous in type.

Single domain models that assume pathological gamblers form a homogeneous population may no longer be adequate in the face of data that putatively demonstrates gambling to be a heterogeneous and multidimensional disorder, the end result of a complex interaction of genetic, biological, psychological and environmental factors. Simple consideration of gambling as an addiction or as a compulsive or impulse control disorder is too limiting in scope. There is a need to identify clinically distinct

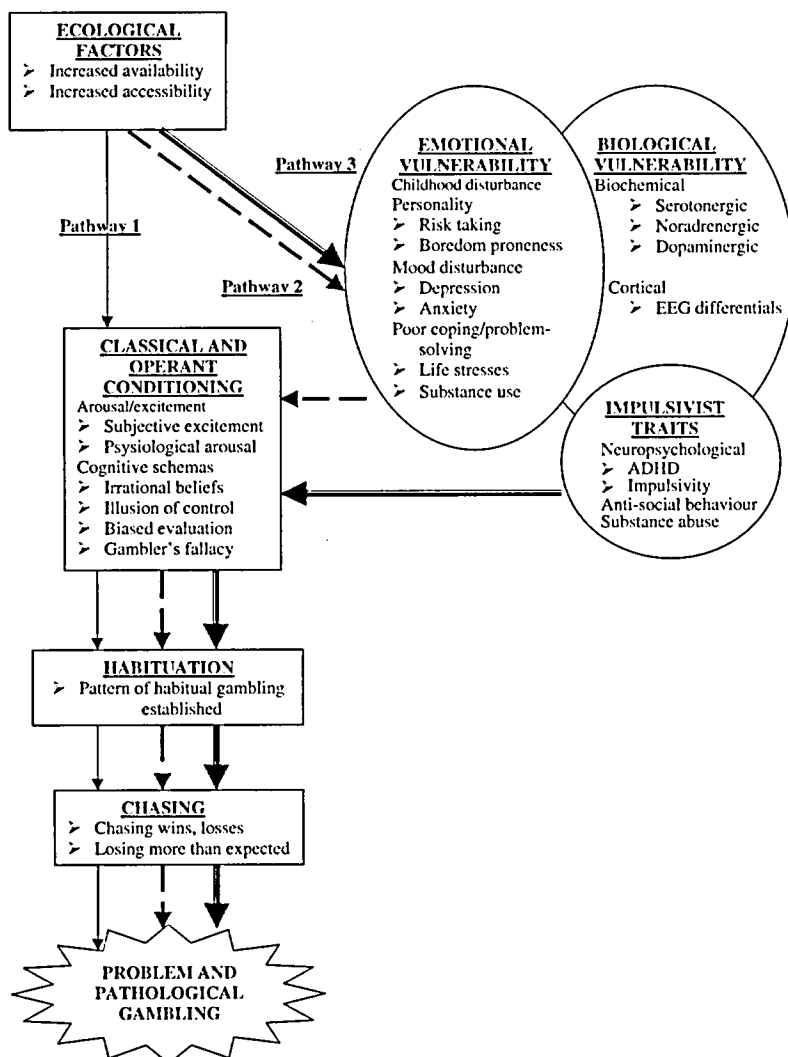


Figure 4 Integrated model of problem gambling

subgroups of gamblers who exhibit common, overt cardinal symptoms, but, at the same time, differ significantly with respect to key variables that are of aetiological relevance and determine approaches to management and prognosis: premorbid psychopathology, childhood history and neurobiological maturity.

The pathways model is a preliminary, empirically testable schema that hypothesizes the existence of three subgroups of pathological gamblers. All three are subject to ecological variables, operant and classical conditioning, and cognitive processes. The strength of this model is its recognition that a proportion of gamblers are essentially 'normal' in character; that is, they do not show signs of premorbid psychological disturbance but simply lose control over gambling in response to the effects of conditioning and distorted cognitions surrounding probability of winning. Their 'pathological gambling' is a transient state where fluctuations between heavy and excessive gambling are observed, a condition which also may remit spontaneously or with minimal interventions.

Pathway 1 gamblers may achieve sustained controlled gambling post-intervention.

The model also acknowledges a second subgroup characterized by disturbed family and personal histories, poor coping and problem-solving skills, affective instability due to both biological and psychosocial deficits and later onset of gambling. Gambling is pursued as a means of emotional escape through dissociation or a medium aimed at regulating negative mood states or physiological states of hyper- or hypo-arousal.

The third group in this schema is characterized by a biological vulnerability toward impulsivity, early onset, attentional deficits, antisocial traits and poor response to treatment. Dysfunctional neurological structures and functions and dysregulation of neurotransmitter systems underpin this vulnerability.

From a clinical perspective, each pathway contains different implications for choice of management strategies and treatment interventions. Clinical observations supported by empirical data suggest that Pathway 3 gam-

blers are typified by an antisocial impulsivist personality dimension manifesting a wide range of multiple dysfunctional behaviour including substance abuse, criminal offences and social instability (Steel & Blaszczynski 1996). These clinical features correlate with early onset gambling, more severe gambling related problems, general psychopathology, and salient features of attention deficit hyperactivity disorder.

If biological correlates contribute to the aetiology of the disorder in cases of such impulsive gamblers, clinicians must be cognisant of the need to attend to problems related to attention and organizational deficits, emotional lability, stress intolerance, poor-problem solving and coping skills. Issues of compliance and attrition from treatment also need to be highlighted given the tendency for impulsive gamblers to be inconsistent, unreliable and intolerant of boredom. These gamblers may require intensive cognitive-behavioural interventions aimed at impulse control administered over longer terms.

In contrast, the treatment needs of this group differ significantly from depressed or anxious gambler who seek emotional solace through the dissociation associated by repetitive electronic gaming machine play (Anderson & Brown 1984). Depression or anxiety may result from neurotransmitter or genetic deficits (Comings *et al.* 1996), result from experienced trauma or loss (Taber, McCormick & Ramirez 1987), or be reactive to a current stressor. Psychotherapeutic strategies designed to enhance coping skills, deal with stress-related issues, and the provision of non-judgemental support are relevant to these cases. Both Pathways 2 and 3 gamblers may require medication to balance their neurochemistry; however, the onset of the disorder, and its severity, course and prognosis of the emotionally vulnerable gamblers differ from that of the impulsive gambler. An understanding of the essential differences defining subgroups of gamblers will, therefore be important in dictating the necessary and appropriate form of intervention required.

The pathways model provides a conceptual framework that integrates research data and clinical observation to provide a structure that assists clinicians in identifying and separating distinct subgroups of gamblers that require differing management strategies. Optimally, the model should provide a practical and useful clinical guide that will ultimately improve the effectiveness of treatment interventions by refining diagnostic processes. The model is open to empirical testing.

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