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Pathways to Pathological Gambling: Identifying Typologies

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Abstract

The majority of explanatory models of pathological gambling fail to differentiate specific typologies of gamblers despite recognition of the multi-factorial causal pathways to its development. All models inherently assume that gamblers are a homogenous population; therefore theoretically derived treatments can be effectively applied to all pathological gamblers. This article describes a comprehensive and alternative conceptual-pathway model that identifies three main subgroups: "normal," emotionally vulnerable and biologically based impulsive pathological gamblers. All three groups are exposed to common influences related to ecological factors, cognitive processes and contingencies of reinforcement. However, predisposing emotional stresses and affective disturbances for one group, and biological impulsivity for another, are additional risk factors of aetiological significance in identifying separate subtypes. The implications for treatment are discussed with particular reference to the need to match client subtype with specific treatment interventions.

Introduction

Historically, societal attitudes toward gambling were influenced by the effects of gambling on public order, the erosion of prevailing moral values and social mores, and the cheating and exploitation of the masses (Peterson, 1950; Ploscowe, 1950; Blakely, 1977). The move to medicalize pathological gambling originated from the case studies of early psychoanalytic writers (Von Hattinger, 1914; Bergler, 1957), and by the inclusion of pathological gambling in DSM-III (American Psychiatric Association, 1980), as a psychiatric disorder of impulse control. The formalization of pathological gambling as a psychiatric disorder led to recent attempts to develop theoretical models, which explain the aetiology of problem gambling (Ferris, Wynne & Single, 1998).

Contemporary psychological models include gambling as:

- an addictive disorder (Jacobs, 1986; Blume, 1987)
- an unresolved intrapsychic conflict (Bergler, 1957; Rosenthal, 1992; Wildman, 1997)
- having its causation through a biological/psychophysiological dysregulation (Blaszczynski, Winter & McConaghy, 1986; Carlton & Goldstein, 1987; Lesieur & Rosenthal, 1991; Rugle, 1993; Comings, Rosenthal, Lesieur & Rugle, 1996)
- a learned behaviour (McConaghy, Armstrong, Blaszczynski & Allcock, 1983; Anderson & Brown, 1984)
- a result of distorted/irrational cognitions (Sharpe & Tarrier, 1993; Ladouceur & Walker, 1996).

This diversity of models has led to the search for qualitative similarities and differences between social and pathological gamblers in personality traits (Blaszczynski, Buhrich & McConaghy, 1985; McCormick, Taber, Kruedelbach & Russo, 1987; Castellani & Rugle, 1995), co-morbidity (Kruedelbach & Rugle, 1994) and biological correlates (Rugle, Semple, Goyer & Castellani, 1995; Comings et al., 1996).

The fundamental assumption contained within each model is that pathological gamblers constitute a homogenous population, and that theoretically derived treatments can be effectively applied to all pathological gamblers. There is minimal evidence to support this implicit assumption. On closer inspection, learning theories (Dickerson, 1979) refer to fixed and variable schedules of reinforcement. But these learning theories fail to explain why not all gamblers suffer impaired control. Cognitive theories (Sharpe & Tarrier, 1993; Ladouceur & Walker, 1996) emphasize irrational cognitive schemas but have not demonstrated that these are of

causal significance. Heated debate continues on the validity of the addiction model of gambling, particularly by those adhering to the socio-cognitive approach.

Divergent frameworks, however, can be reconciled if gamblers are accepted as a heterogeneous group (Blaszczynski, 1996) with multi-factorial causes. It cannot be denied that the majority of gamblers seek monetary gain. But some continue to participate and persist because they are inexorably motivated to find relief from boredom, to dissociate and to escape from negative life circumstances, or to modulate negative mood states. The task confronting clinicians is to refine the categorization of problem gamblers into increasingly homogenous subgroups or typologies of gamblers.

In a series of long-term controlled outcome studies (Blaszczynski, 1988; McConaghy, Blaszczynski & Frankova, 1991), three types of responses to treatment were observed: controlled gambling, abstinence and uncontrolled gambling. Controlled gamblers were characterized by an absence of psychopathology, abstinent gamblers continued to exhibit moderate levels of affective disturbances and elevated neuroticism; while uncontrolled gamblers persisted in showing high levels of psychopathology across a number of domains. These findings matched my clinical experience. I found that some gamblers displayed integrated personalities; others showed evidence of depressive affect and situational stresses which precipitated increased gambling. Others manifested traits of impulsivity and severe disruptive behaviours in gambling and in other parts of their lives.

These findings made me question if the response to treatment was predicated on personality or demographic differences, which were present between groups prior to treatment. However, no such differences emerged when statistical comparisons were applied to group variables. An alternative possibility was therefore considered: that is, that the end results of gambling had affected their psychological profile so that it masked group differences. I argued that with gambling the common manifestation of affective disturbances (anxiety, substance use and criminality) were a complex mixture and/or interaction of both primary and secondary processes involved in gambling. In some cases, depression was instrumental in causing impaired control over gambling; while in others, gambling produced depression resulting from financial and marital difficulties. During a psychometric assessment, both groups obtained similar scores on depression. But this depression had significantly different implications in respect to etiological significance and relevance to treatment strategies. This led to the postulate that specific subgroups of gamblers existed and shared features in common, yet differed significantly in many respects.

I have proposed a prototypical model that attempts to integrate biological,

personality, developmental, cognitive, learning theory and environmental factors into one model. This model is based on clinical experience and attempts to integrate relevant research findings. It suggests the existence of three major types of gamblers: the gambler who is not pathologically disturbed, the gambler who is emotionally vulnerable, and the gambler whose impulsivity is biologically based.

There are three elements relevant to all gamblers irrespective of subgroup membership. The first relates to ecological determinants. These determinants revolve around public policy issues that promote availability and access to gambling facilities. Substantive data clearly demonstrates that the incidence of pathological gambling is inextricably tied to the number of available gambling outlets (Abbott & Volberg, 1996; Volberg, 1996; Productivity Commission, 1999).

The second element resides in the role of classical and operant conditioning. Studies have demonstrated that gambling produces a state of subjective excitement (Dickerson, Hinchy & Fabre, 1987), dissociation (Jacobs, 1986) and increased heart rate (Anderson & Brown, 1984; Leary & Dickerson, 1985; Brown, 1988; Griffiths, 1995). Wins, delivered at variable ratios that are resistant to the effects of extinctions, produce states of excitement described as equivalent to a "drug-induced high." Repeated pairings classically condition this arousal to stimuli associated with the gambling environment (Dickerson, 1979; Sharpe & Tarrier, 1993). Through second order conditioning, gambling cues elicit an urge to gamble, which results in a habitual pattern of gambling. As Rosenthal and Lesieur (1992) observe, excitement can be experienced in anticipation, during, or in response to exposure to gambling situations or cues. This process of conditioning can be used to explain gambling as an addiction produced by the effects of positive and negative conditioning, tolerance and withdrawal.

An alternative non-addiction explanation has also been offered, and is based on a neo-Pavlovian "neuronal model" of habitual behaviour, which relies on the concept of cortical excitation (McConaghy, 1980).

Superimposed on the conditioning framework and irrespective of whether or not an addiction type model is adopted, is the development of cognitive schemas. Early and repeated wins result in irrational belief structures that promote gambling as an effective source of income. These schemas shape illusions of control, biased evaluations, erroneous perceptions, superstitious thinking and faulty understandings of probability (Langer, 1975; Gilovich, 1983; Ladouceur & Walker, 1996; Walker, 1992; Griffiths, 1995).

The reinforcing properties of gambling and the irrational cognitive schemas combine to consolidate and strengthen habitual gambling practices. At this point, the downward spiral of gambling, perceptively described by Lesieur (1984), takes

its toll. When gamblers lose they attempt to recoup losses through further chasing, which results in accumulating financial debts. Despite acknowledging the reality that gambling led them into financial problems, they irrationally believe that gambling will solve their problems.

It is emphasized that the above processes are applicable to all gamblers. At this point additional factors can be invoked to differentiate between three broad subgroups of gamblers.

Subgroup one: "Normal" problem gamblers

The first subgroup can be labelled, perhaps somewhat oxymoronically, as the "normal" pathological gambling subgroup. Members of this subgroup may meet formal criteria for pathological gambling at the height of their gambling disorder. What distinguishes this subgroup is the absence of any specific premorbid psychopathology. Conceptually, these gamblers can be seen as occupying the diffuse domain between regular-heavy and excessive gambling. Excessive gambling behaviour occurs as a result of bad judgments or poor decision-making strategies, which are independent of any intrapsychic disturbance. Features of a preoccupation with gambling, chasing losses, substance dependence and depression and anxiety are all seen as the end response to the presence of financial pressures caused by continual losses. These symptoms are the consequence not the cause of excessive gambling.

Clinically, the severity of difficulties in the "normal" gambling subgroup is the lowest of all pathological gamblers. They do not manifest gross signs of major premorbid psychopathology, substance abuse or impulsivity behaviours. Placed at the low end of the problem-gambling scale, these gamblers move between heavy and problem gambling. They are more motivated to seek treatment, to comply with instructions and post treatment are able to achieve controlled levels of gambling. Counselling and minimal intervention programs are of benefit.

Subgroup two: Emotionally disturbed gamblers

The next subgroup is characterized by the presence of predisposing psychological vulnerability factors where participation in gambling is motivated by a desire to modulate affective states and/or meet specific psychological needs. This subgroup manifests a history of problem gambling in the family, negative developmental

experiences, neurotic personality traits and adverse life events. These problems may contribute in a cumulative fashion to produce an emotionally vulnerable gambler.'

Evidence in support of this contention comes from a number of sources. Jacobs (1988), Lesieur and Rothschild (1989), Gambino, Fitzgerald, Shaffer, Renner, and Courtage (1993) observed that a family history of pathological gambling was an important predisposing risk factor for children. Jacobs (1986), in his General Theory of Addiction, postulated that certain personality characteristics and life events, which interacted with physiological states of arousal, influenced the development of gambling problems. He stated that excessive gambling was produced by the interaction between abnormal physiological resting states of hyper or hypo-arousal, and a history of negative childhood experiences. Personal vulnerability was linked to negative childhood experiences of inadequacy, inferiority, low self-esteem and rejection (McCormick, et al., 1987; McCormick, Taber & Kruedelbach, 1989).

This subgroup of gamblers displays higher levels of premorbid psychopathology. In particular, they display depression, anxiety, substance dependence, and deficits in their ability to cope with and manage external stress. Gamblers within this subgroup cannot express their emotions directly and effectively, and they show a tendency to engage in avoidance or passive aggressive behaviours. Emotionally vulnerable gamblers see gambling as a means of achieving a state of emotional escape through the effect of dissociation on mood alteration and narrowed attention (Anderson & Brown, 1984; Jacobs, 1986).

The abstinent gamblers in Blaszczynski's (1988) and Blaszczynski, McConaghy and Frankova's, (1991) two-to-five year treatment outcome study appear to fall within this subgroup. In respect to psychopathology, the abstinent gamblers were placed on an intermediate position between the more adjusted controlled and severely disturbed uncontrolled gamblers. Because of their negative developmental history and poor coping skills, these gamblers were regarded as too fragile to maintain sufficient control over behaviour to permit controlled gambling.

Subgroup three: Biological correlates of gambling

The third subgroup of pathological gamblers is defined by the presence of neurological or neurochemical dysfunction reflecting impulsivity (Steel & Blaszczynski, 1996) and attention-deficit features (Rugle & Melamed, 1993). Briefly, evidence supporting neurological deficits in gamblers is found in

electrophysiological, neuropsychological and biochemical studies.

Goldstein and his colleagues (Goldstein, Manowitz, Nora, Swartzburg & Carlton, 1985; Carlton, Manowitz, McBride, Nora, Swartzburg & Goldstein, 1987) reported differential patterns of EEG activity and self-reported symptoms among gamblers found in childhood attention deficit disorder. Supporting this finding, Rugle and Melamed (1993) on the basis of neuropsychological measures of executive functions concluded that childhood differences in behaviours related to overactivity, destructibility and difficulty inhibiting conflicting behaviours were of primary importance in differentiating gamblers from controls. These authors noted that attention- deficit related symptoms reflecting traits of impulsivity were present in childhood. These traits predated the onset of pathological gambling behaviour and gave rise to the hypothesis that impulsivity precedes gambling; and that impulsivity is independent of it and is a good predictor factor for severity of involvement in at least a subgroup of gamblers.

From preliminary evidence in the field of genetics and from neurotransmitter activity comes the tentative hypothesis which links receptor genes and neurotransmitter dysregulation in reward deficiency, arousal, impulsivity and pathological gambling (Roy, De Jong & Linnoila, 1989; Lopez-Ibor, 1988; Moreno, Saiz-Ruiz & Lopez-Ibor, 1991; Carrasco, Saiz-Ruiz, Hollander, Cesar & Lopez-Ibor, 1994; Comings et al, 1996; Bergh, Eklund, Sodersten & Nordin, 1997; DeCaria, Hollander, Grossman, Wong, Mosovich & Cherkasky, 1996).

Genetic studies have recently reported that pathological gamblers, similar to substance abusers, are much more likely to have the D2A1 allele for the dopamine D2 receptor gene than controls leading Comings et al., (1996) to suggest that the D2A1 allele may be a major risk factor in pathological gambling. When gamblers were evaluated on severity, 63.8 per cent of them in the upper range carried the D2A1 allele compared to 40.9 per cent in the lower range. Of note: 76.2 per cent of pathological gamblers who were co-morbid alcohol abusers carried the gene compared to 49.1 per cent of males without co-morbid alcohol abuse or dependency.

It is argued that gamblers manifest differential responses to reward and punishment because of their biologically based impulsivity. These gamblers manifest a marked propensity for seeking out rewarding activities. They are unable to delay gratification, and have a diminished response to punishment. When the consequences of their actions are painful, they fail to modify their behaviour.

Clinically, impulsive gamblers display a broad spectrum of behavioural problems which are independent of gambling. These problems include substance abuse, suicidality, irritability, low tolerance for boredom, sensation seeking and criminal

behaviours. Poor interpersonal relationships, excessive alcohol and poly-drug experimentation, non-gambling related criminality, and a family history of antisocial behaviour and alcoholism are characteristic of this group. Gambling commences at an early age, rapidly escalates in intensity and severity, occurs in binge episodes and is associated with early gambling-related criminality. These gamblers are less motivated to seek treatment in the first instance, have poor compliance rates, and respond poorly to any form of intervention.

Discussion

The starting premise of the proposed pathway typology model is that problem gamblers form a heterogeneous population; the end result of a complex interaction of genetic, biological, psychological and environmental factors. From this population, subgroups of gamblers sharing commonalties can be extracted. The strength of this approach is that it integrates disparate findings reported in the literature. It takes into account the notion that there are groups of non-disturbed gamblers. These gamblers lose transient control over their behaviour because of irrational cognitions, which lead to a series of poor judgments and they become temporarily over-involved in gambling. Fluctuations between heavy and excessive gambling are observed; their disordered gambling may remit spontaneously or with minimal interventions. At the same time, the pathway typology recognizes subgroups of gamblers who participate for emotional reasons: to dissociate as a means of escaping painful life stresses, to reduce boredom, or to deal with unresolved intrapsychic conflicts or childhood traumas. The model also acknowledges that there are some gamblers who exhibit biological correlates of disturbed behaviours. These traits qualify them as sufferers of a medical and/or psychiatric condition characterized by impulsivity and features of attention deficit disorder.

All three subgroups are affected by environmental variables, conditioning and cognitive processes. From a clinical perspective, each pathway contains different implications for managing ement strategies and treatment interventions. "Normal" pathological gamblers require minimal interventions, counselling and support strategies and may resume controlled gambling post intervention. Self-help groups such as Gamblers Anonymous are effective, as are self-control self-help educational materials.

The needs of emotionally vulnerable gamblers who seek solace through dissociation produced by gambling (Anderson & Brown, 1984) to deal with emotional distress, life circumstances or trauma and loss (Taber, McCormick &

Ramirez, 1987) require more extensive psychotherapeutic interventions. Relevant here are stress management and problem-solving skills, as are therapeutic endeavours directed toward resolving intrapsychic conflicts and procedures designed to enhance self-esteem and self-image.

For those gamblers with biological correlates, clinicians must attend to problems related to attention and organizational deficits, emotional liability, stress intolerance, and poor problem solving and coping skills. These gamblers may require intensive cognitive behavioural interventions aimed at impulse control, which is administered over longer terms. Medication aimed at reducing impulsivity through its calming effects may be considered (for example, Prozac); although more random-controlled outcome trials are needed before the benefits of the medication can be established with confidence.

The proposed pathway model is a conceptual framework that attempts to integrate research data and clinical observation to assist clinicians in the identification of distinct subgroups of gamblers requiring different treatment strategies. It is hoped that the model will provide a practical clinical guide that will improve the effectiveness of treatment by refining diagnostic processes and matching gamblers to intervention techniques. The model is open to empirical testing.

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