The Diagnostic Classification of Pathological Gambling

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Abstract

Further study is required on the etiology of pathological gambling. This paper applies Pennington’s four levels of diagnostic taxonomy of psychopathology to pathological gambling. Pathological gambling is examined from a bidirectional perspective on the levels of epidemiology, behavioral and molecular genetics, neurobiology, and neuropsychology.

Keywords: gambling, disorder, impulse, addiction, pathology, psychology, neuropsychology, neurobiology, epidemiology, etiology, genetics, DSM-IV-TR.

Nosology and Terminology

Pathological Gambling is classified by the DSM-IV-TR as a disorder of impulse control. This class of disorders, which includes kleptomania, pyromania, and the explosive disorders, is characterized by an individual’s marked inability to resist harmful impulses. Some experts consider pathological gambling an “addiction,” but the DSM-IV-TR does not include this word; nor does it place the disorder in the same category as that of substance abuse and dependence (APA 2000). The pathological gambler does not require external chemical inducement to achieve his “high;” rather, it is the experience of gambling itself that creates a subjective and internal experience that stimulates the individual to engage in impulsive behaviors.

Some researchers have speculated that pathological gambling is best categorized as an obsessive-compulsive spectrum disorder (Blaszczynski 1999). As mentioned below, there is some genetic basis for this. Other experts classify pathological gambling and substance dependence together due to similarities other than exogenous chemical inducement, for example, “cravings” and “triggers” for gambling behavior as well as antisocial behavior, such as lying about gambling or stealing to support the habit. As with the substance dependent individual, the pathological gambler may experience denial and remorse. There is much debate over whether gambling should be classified as an impulse control or obsessive-compulsive disorder. There is evidence to support the idea that pathological gambling shares characteristics of both diagnostic categories (Blanco, et al. 2001). Gambling behaviors are mildly obsessive in the sense that they can preoccupy a person’s thoughts, but these thoughts are not perceived as controlling or beyond one’s ability to resist. Some repetitive gambling behaviors bear a resemblance to compulsive behaviors in that they at times relieve anxiety, but unlike pathological compulsions they are not usually rigidly ritualistic or stereotypical or perceived as going against one’s wishes. Unlike the pathological gambler, the obsessive-compulsive person derives no pleasure from his or her compulsive behavior (other than the release of tension).

A non-diagnostic theory asserts that human beings developed a proclivity to gambling in psychoevolutionary development (Petry 2005). In a state of nature predators need to make quick decisions about whether to go for small game, which are easier to catch but provide fewer calories, or “gamble” for larger game, which provide more calories, but are more difficult to hunt. Human history may have involved numerous risky dilemmas, such as whether to move a great distances to better hunting grounds. If so, it is reasonable to
speculate that human beings began to think in terms of risk/loss, and perhaps some of our ancestors developed a greater proclivity for risk taking ventures than others.

Of Pennington’s classifications, pathological gambling can only loosely fit into the category of “disorders of action regulation,” which includes conduct disorder, schizophrenia, attention-deficit hyperactivity-disorder, and obsessive-compulsive disorder (Pennington 2002). What it shares in common with these disorders includes problems with behavior regulation, thought selection, and self-defeating behavior. However, Pennington’s taxonomy seems awkward and too general for pathological gambling: perceptual and developmental disorders are etiologically and cognitively very different from pathological gambling. Moreover, the differences between the above disorders and pathological gambling are greater than their similarities.

Unlike the chemical addiction disorders, which are measured by degree of severity and are differentiated between abuse and dependence, the DSM-IV-TR criteria do not distinguish between levels of gambling severity. However, expert consensus is that gambling problems exists on a continuum, with “problem gambling” being less severe than pathological gambling, but still requiring clinical attention. There is no official definition of “problem gambling,” but the term is widely used and included in several assessments.

Harvard researchers Shaffer and Hall (1996) developed a now widely used model for classifying gambling patterns across four levels as follows. Level 0 gambling includes those who have never wagered money in gambling. Level 1 includes leisure or social gamblers who have never had serious gambling-related problems. Level 2 gamblers are problem gamblers who have had some serious problems, but do not meet full criteria for pathological gambling. In most cases, an individual who has ever met even one of the criteria for pathological gambling is considered a Level 2 gambler. Level 3 gamblers are pathological and meet DSM-IV-TR criteria for the disorder. The above classifications are helpful in describing what level of gambling problems exists among different demographics. The bullets below list the DSM-IV and DSM-IV-TR criteria for pathological gambling.

Pathological gambling involves five or more of the following:

- Preoccupation with past, present, and future gambling experiences and with ways to obtain money for gambling.
- Need to increase the amount of wagers.
- Repeated unsuccessful efforts to cut back or stop.
- Becoming restless or irritable when trying to cut back or stop.
- Gambling to escape from everyday problems or to relieve feelings of helplessness, anxiety, or depression.
- Trying to recoup immediately after losing money (chasing losses).
- Lying about gambling.
- Committing illegal acts to finance gambling.
- Losing or jeopardizing a personal relationship, job, or career opportunity because of gambling.
- Requesting gifts or loans to pay gambling debts. (American Psychiatric Association, 2000).

The term “problem gambler” or “Level 2 gambler” refers to someone who meets at least one but less than five of the above criteria.

At least 25 instruments have been developed that screen for symptoms and surface level properties of problem and pathological gambling (Shaffer, et al. 1999). The most widely used instruments are the Southern Oaks Gambling Screen (SOGS) and the National Opinion Diagnostic Screen (NODS). 48 states have made use of at least one of the above screening tools for official data collection (Shaffer, et al. 1999). The SOGS assesses for gambling frequency; lying about gambling; self-perception of a problem; gambling more than intended; concerned others; guilt; inability to stop; hiding evidence; relationship problems related to gambling; borrowing; interference with work or school. Based on assessed score, individuals are classified as 0, 1, 2, or 3 Level gamblers. It was developed by Henry Lesieur and Shelia Blume.
at South Oaks Psychiatric Hospital, California. It is sometimes used in combination with the NODS, a seventeen question survey based on DSM-IV criteria. Rather than describe the coding and instrument interpretation of the above assessments, this paper will focus on the symptom levels addressed.

**Epidemiology**

Petry (2005) cites several international studies on gambling disorders which collectively suggest at worldwide lifetime pathological gambling prevalence of 1 to 2% (Shaffer, *et al.* 1999). Meta-analyses of North American gambling indicates a lifetime prevalence of Level 3 gambling of 1.92%, and 1.46% for the last year. It is worth noting that these general population prevalence rates are similar to other disorders, such as schizophrenia and obsessive-compulsive disorder.

Volberg and Moore (1999) completed an epidemiological study on problem gambling in the State of Washington from the years 1992 to 1998. In the 1998 survey, the sample size was 1,501 adults and a survey questionnaire was used. The authors concluded that the population of the State of Washington has a lifetime 5.0% rate of combined problem and pathological gambling with 2.5% prevalence rate for the last year. This compares with a national average of 2.7% (lifetime rate) and 1.3% (within the last year). (However, gross receipts for gambling establishments have decreased every year since 2001 in the State of Washington.) Incidences of gambling disorders tend to increase proportionately to gambling availability.

Comorbidities for pathological gambling include psychopathy, narcissistic personality disorder, alcohol dependence, cocaine dependence, attention-deficit hyperactivity disorder, depression and bipolar disorder. Level 3 gamblers are more likely to suffer from alexithymia, an unclassified disorder characterized by low emotional affect, difficulty recognizing one’s own emotions and those of others, and mundane thinking (Lumley and Roby 1995). It is possible that for some, gambling is a surrogate form of relatedness.

Anxiety disorders and obsessive-compulsive disorder have not yet been found to be typically co-occurring with pathological gambling (Petry 2005).

Those with substance use disorders have higher rates of problem and pathological gambling. Shaffer, *et al.*’s (1999) meta-analysis of 18 studies indicated a lifetime Level 2 rates of 15.01% and Level 3 rates of 14.23% in adults in treatment for substance use disorders. Other research indicates that disordered gambling among substance users exists at a higher rate than that of the general population regardless of substances used, including cannabis (Toneatto and Brennan 2002).

Level 3 gamblers tend to be Caucasian, married and employed younger males of lower socioeconomic status with at least a high school degree (Petry 2005; Volberg 1994). (It is possible, however, that married gamblers are more likely to seek treatment from family pressure). Pathological gamblers are mostly males at a 2:1 ratio to females (APA 2000). Senior citizens might gamble at higher rates, but are less likely to seek treatment and represent a minority of Level 3 gamblers.

**Behavioral and Molecular Genetics**

Genes responsible for dopamine, norepinephrine, and serotonin development are associated with pathological gambling, specifically Monamine Oxidase A/B (MAOA A, MAOA B) and Dopamine Receptors D2 (DRD2) and DRD4. Those diagnosed with pathological gambling are more likely to have shortened allele markers on MAOA and DRD2 genes (Ibanez, *et al.* 2000). It is significant that these genes are associated with other disorders. MAOA alleles are also associated with substance dependence disorders. DRD1, DRD2 and DRD4 genetic variants are associated with obesity, obsessive-compulsive disorder, Tourette syndrome, and substance dependence, suggesting a genetic locus for compulsive, addictive, and impulsive behaviors (Blum, *et al.* 1995). However, no one specific gene or allele marker is independently correlated with pathological gambling. With over 30 genes
A family study on pathological gambling exist, but it may be impossible to distinguish between environmental and hereditary factors because of childhood exposure to gambling behavior. In one study, 86% of children who gambled reported gambling with their parents (Gupta and Derevensky 1997). As of the present date, no studies have been done on endophenotypic traits of pathological gambling. Furthermore, there is no instrument to detect prodromal signs of pathological gambling.

Adoption and twin studies are much more likely to reveal an authentic correlation between the pathological gambling and heritability. Children who are adopted and do not maintain contact with their biological parents sometimes experience the same disorders as their biological parents due to genetic inheritance. Unfortunately, this writer could not find any adoption studies on pathological gambling. Recently Petry (2005) also confirmed that there are no adoption studies on this subject.

Twin studies can indicate a genetic etiology when identical (monozygotic) twins, who share all their genes, experience a disorder at different rates than that of fraternal (dizygotic) twins, who share approximately half their genes. A disorder with a predominately environmental origin would not show substantial differences in the manifestation of the disorder in either kind of twin set. A genetic etiology, however, would result in different rates for monozygotic and dizygotic twins.

In 1991 Eisen surveyed 6,718 male (3359 twin set) members of the Vietnam-era Twin Registry as the basis for his study (Eisen, et al. 1998). These individuals were contacted by telephone and surveyed using the Diagnostic Interview Schedule Gambling Assessment model (DIGS), which is a 20 question item questionnaire that assesses for gambling problems, psychiatric symptoms, environmental influences, and legal problems. The DIGS has well-established psychometric properties and includes DSM-III-R (identical to DSM-IV-TR criteria). Monozygotic twins had higher rates of lifetime pathological gambling at 22.6% compared to 9.8% for dizygotic twins. Subclinical pathological gambling, or meeting less than 5 of the DSM-III-R criteria, occurred at rates of 6.1% for monozygotic twins and 3.1% for dizygotic twins. This study indicates a greater than chance genetic vulnerability to pathological gambling. Eisen’s research concluded that inheritable factors explain 62% of the variance in pathological gambling.

**Neurobiology**

It has been theorized that pathological gambling behavior is reciprocally influenced by multifarious neurobiological factors. Pathological gamblers have been studied on several neurobiological levels including the neurochemical, hormonal, and noradrenergic. Various mediating neurochemicals are produced by and interact with various regions of the brain. Neurochemical levels can change in response to external stimuli, such as the experience of gambling, without any ingestion of an external chemical agent. Human perception and experience create intrapsychic processes that produce neurochemical changes, most notably in catecholamines (norepinephrine, dopamine), indoleamine (serotonin), and opioid alkaloids.

Norepinephrine is a stress hormone and neurotransmitter released from the adrenal glands into the central nervous system and blood. As part of the noradrenergic system, norepinephrine levels change based on arousal, for example, physiological reactivity in the flight-or-flight experience which produces increased heart rate and muscle tenseness. Mood is also influenced by norepinephrine. Since it is theorized that the gambling
experience involves “fight” (betting) and “flight” (loss of the wager), it is not surprising that researchers have found increased norepinephrine levels in gamblers in urine and cerebrospinal fluid (Roy, et al. 1988). It should be noted however, that not all gambling experiences are equal; some subgroups of gamblers, such as horse race gamblers, do not appear to have elevated norepinephrine levels during the gambling experience as do other types of gamblers (Blaszczynski, et al. 1986). The central nervous system locus of norepinephrine is the ceruleus area of the brain, extending to the cortex, basal ganglia, and limbic system.

Pleasure pathways in the brain are also associated with the gambling experience. Several studies have shown or indicated that dopamine function plays a role in pathological gambling. Subjects have been recorded as having elevated levels of dopamine in cerebrospinal fluid, urine, and plasma (Meyer, et al. 2004). Although samples have been small, dopamine agonist medications administered to Parkinson’s patients have been known to iatrogenically induce pathological gambling behavior. In one study, 7 of 11 Parkinson’s patients developed pathological gambling while taking a dopamine agonist (Dodd, et al. 2005). When the medication was discontinued, the gambling behavior ceased. The fact that agonists that mimic naturally occurring dopaminergic function by occupying cell receptors cause pathological gambling in some subjects implies that non-medicated pathological gamblers may experience abnormally elevated dopaminergic activity. Because the experience of gambling initially involves pleasure, it is not surprising to find activity in the dopaminergic reward pathway system in the brain of the pathological gambler.

Alkaloid opioid levels can increase during peak gambling experiences; however, some subgroups of gamblers do not appear to have this experience. Under usual experiences, endorphins are produced by the pituitary and interact with dopamine to reduce pain. However, they are also activated by physiological arousal, a well-documented phenomenon in pathological gambling (Meyer, et al. 2004) A few studies have been done on opioid levels among pathological gamblers, but those that exist reveal increased opioid activity during peak gambling experiences. Recently Nalmefene, an opioid antagonist, has been shown to be a promising treatment for pathological gambling (Grant, et al. 2006).

Other possible relationships between neurochemistry and pathological gambling have not been studied sufficiently to draw conclusions about their relevance to the disorder. It has been speculated that because men tend to suffer from pathological gambling at higher rates than women testosterone might be involved in the development of the disorder, but there is no evidence that testosterone plays a significant role in the development of the disorder (Blanco, et al. 1998). Neuroendocrine chemicals, such as cortisol, have been demonstrated to be elevated in disordered gamblers, but his may be a secondary response to physiological arousal (Meyer, et al. 2004).

Specific research has not been carried out on the role histamine might have in the neurophysiology of pathological gambling. Low levels of histamine have been correlated with depression and schizophrenia, and pathological gamblers have higher levels of these disorders than the general population. However, it is important not to infer too much from this speculative potential correlation. Mice with low levels of histamine show decreased levels of activity and arousal (Yanai, et al. 1998) whereas pathological gamblers experience higher rates of arousal and activity.

**Neuropsychology**

According to Pennington (2002), the neuropsychological level of analysis includes cognition, memory, perception, attention, and the executive functions (organizing, sequencing, and abstracting.) For purposes of this paper, the focus will be on the cognitive appraisals that pathological gamblers exhibit that influence their behavior.

The basis for many of the pathological gambler’s cognitive distortions lies in an illusion of control. Quite simply, this is the idea
that one has more control over events than one actually does. In gambling cognition, this takes the form of belief that one can predict outcomes at a rate higher than chance. In one study, participants placed more value on the lottery tickets they purchased themselves than on those randomly given to them. In another study, subjects in a casino were found to throw dice harder for high numbers, and softer for lower numbers (Griffiths 1990). Probabilities of winning are not in fact increased by superstitious behavior.

A similar distortion is an attributional bias. Positive outcomes are perceived as caused or influenced by the gambler whereas negative outcomes are considered the result of chance or bad luck. Gambling “skill” and “strategy” are viewed as causal factors in outcomes. (It is interesting to note that ideas of reference and magical thinking are also characterized by false perceptions of cause-effect relationships). Moreover, illusion of control and attributional bias are common to gamblers who do not meet criteria for pathological gambling. Attribution biases when a gambler does happen to win can create overconfidence. Some studies have shown that narcissistic cognitions, such as unrealistic overconfidence and self-appraisal, exist among problem and pathological gamblers (Goodie 2005).

Another logical error has been called the representativeness heuristic. This error occurs when the gambler mistakenly believes that dissimilar numbers or sequences are more likely to occur than similar sequences. In a lottery drawing with six numbers, a gambler will tend to believe that the sequence 1-9-15-36-45-50 is more likely to be the winning number than the sequence 1-2-3-4-5-6. In terms of probability, the chance of either sequence occurring is equal. As with the distortions mentioned above, the representativeness heuristic is common among gamblers in general, not only Level 2 and Level 3 gamblers.

Sometimes gamblers become emotionally invested in gambling with a specific machine or using specific number sequences. Entrapment occurs when a commitment to a gambling behavior persists in the hope it will “pay off.” If a man loses $500 in a slot machine, he might think that it is “ready to pay off” and will continue putting money into that specific machine. Interestingly, stock market investors have been known to exhibit this behavior. Another term for this phenomenon is chasing losses.

Conclusion

Pathological gambling is adequately comprehended along the levels discussed by Pennington. As with many psychiatric disorders, there is no unidirectional causation for pathological gambling. Behavior, neuropsychology, brain function and etiology interact in the development and surface level manifestations of the disorder. Based on current data, it is probable that some individuals are predisposed to pathological gambling, and their experience of gambling activates arousal and pleasure systems in the brain that reinforce gambling behavior.

Some questions remain. A predominantly hereditary developmental picture of pathological gambling is difficult to determine because of the environmental influences of gambling parents or caregivers. Multiple interacting genes responsible for neurochemical development may produce a predisposition for pathological gambling in some individuals; however, these same interacting genes are believed to influence other psychiatric disorders as well.

Another difficulty is classification of the disorder. Pathological gambling is not satisfactorily categorized within Pennington’s diagnostic categories. The criteria for “disorders of action regulation” is too inclusive and broad in its scope for pathological gambling and the other several disorders Pennington includes that are substantially diagnostically different from pathological gambling. Some experts have difficulty classifying pathological gambling as an impulse control disorder because it is genetically similar to obsessive-compulsive disorder and can be characterized in a similar way as “repetitive” and “compulsive.” Others view pathological gambling as being most similar to substance dependence.

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References


