Pathological Gambling: obsessive-compulsive disorder or behavioral addiction?*

Analucía Alegría¹
Silvia Bernardi²
Carlos Blanco³

Abstract

Introduction: There has considerable debate about the appropriate conceptualization of pathological gambling (PG) and its place in psychiatric nosology. PG has been hypothesized to represent both an Obsessive-Compulsive spectrum disorder (OCD) and a behavioral addiction, that is an addiction without a drug. Conceptualization of pathological gambling is vital to guide research strategies and the development and testing of effective treatments. We will review the existing research supporting the non-pharmacologic addiction model and that supporting the obsessive-compulsive spectrum conceptualization. Objetive: To review the conceptualization of PG and the aspects associated with the OCD or a behavioral addiction. Results and Conclusions: Although PG resembles OCD in some domains, the majority of the existing data suggests substantial differences between them. Findings from phenomenology, epidemiology, treatment response, and imaging study appear to support that PG resembles more closely an addiction. Nevertheless, despite the progress over the last decade in understanding addictions, PG, and OCD, existing data are often limited and include methodological concerns that complicate interpretation and comparisons across subject groups.

Key words: Pathological gambling, treatment, obsessive-compulsive disorder.

Titulo: Juego patológico: ¿trastorno obsesivo-compulsivo o conducta adictiva?

Resumen

Introducción: Existe un debate considerable acerca de la conceptualización de la ludopatía o juego patológico (JP) y su lugar en la nosología psiquiátrica. Se ha formulado la hipótesis de representarlo tanto como un trastorno del espectro obsesivo-compulsivo como una conducta adictiva, que es una adicción sin drogas. La conceptualización del juego patológico

* Funding/Support: This study is supported by NIH grants DA019606, DA020783, DA023200, DA023973 and MH082773, a grant from the American Foundation for Suicide Prevention and the New York State Psychiatric Institute (Dr. Blanco).

¹ B.S. From the Department of Psychiatry of the New York State Psychiatric Institute/ Columbia University.
² M.D. From the Department of Psychiatry of the New York State Psychiatric Institute/ Columbia University.
³ M.D., Ph.D. From the Department of Psychiatry of the New York State Psychiatric Institute/ Columbia University.
es fundamental para guiar las estrategias de investigación y desarrollo de tratamientos eficaces. En este artículo se revisará la investigación ya existente que apoya el modelo de adicción no farmacológica y la que apoya la conceptualización del espectro del trastorno obsesivo-compulsivo (TOC).

**Objetivo:** Revisar la conceptualización del JP y los aspectos asociados con el TOC o una adicción de comportamiento. **Resultados y conclusiones:** Aunque al parecer el JP tiene rasgos del TOC en algunos dominios, la mayoría de los datos existentes sugieren diferencias sustanciales entre ellos. Los hallazgos de la fenomenología, la epidemiología, la respuesta al tratamiento y estudio por imágenes parecen apoyar que el JP se parece más a una adicción. Sin embargo, a pesar de los progresos en la última década en la comprensión de las adicciones, el JP y el TOC, los datos existentes son a menudo limitados e incluyen deficiencias metodológicas que complican la interpretación y las comparaciones entre grupos de sujetos.

**Palabras clave:** juego patológico, tratamiento, trastorno obsesivo-compulsivo.

**Introduction**

Gambling is a common activity in almost all societies around the world. It is defined as risking something of value on the outcome of an event when the probability of winning or losing is determined by chance (1). Although the vast majority of individuals who gamble never experience any adverse consequences from the behavior, it is estimated that 1% of the population meets criteria for pathological gambling (PG). An additional 5% have serious problems related to gambling with certain groups, such as young adults or individuals with psychiatric comorbidities considered at increased risk.

In 1980, the American Psychiatric Association officially included pathological gambling (PG) as an impulse control disorders in the *Diagnostic and Statistical Manual of Mental Disorders*, third edition (*DSM-III*), and where it remained in the fourth edition (*DSM-IV*) (2). Similarly, in the International Classification of Disorders, PG is classified along with pyromania, kleptomania and trichotillomania, under the category of “habit and impulse disorders”. PG is characterized by persistent and recurrent maladaptive gambling behavior resulting in damage to vocational, employment, family and social interests. It is also associated with financial losses and legal problems, along with medical and psychiatric comorbidity.

There has considerable debate about the appropriate conceptualization of pathological gambling and its place in psychiatric nosology. PG has been hypothesized to represent both an OC-spectrum disorder and an addiction without a drug (3), and data exist to support each categorization (4,5). Conceptualization of pathological gambling is vital to guide research strategies and the development and testing of effective treatments we will review the existing research supporting the non-pharmacologic addiction model.
and that supporting the obsessive-compulsive spectrum conceptualization (6).

**Phenomenology**

Repetitive, intrusive thoughts about gambling in PG share features with obsessions in OCD. Like OCD, PG is characterized by repetitive behaviors. In PG, gambling and gambling-related behaviors (e.g., handicapping, getting money to gamble) are performed repeatedly (7). The obsessive-compulsive spectrum disorders model of PG is based on the observation that PG frequently report having repetitive gambling-related thoughts leading them to gamble against their better judgment. This experience is consistent with characteristics found in other obsessive-compulsive spectrum disorders. Patients with obsessive-compulsive spectrum disorders report unpleasant feelings and a physiological activation that results in an intense desire to perform a specific behavior to relieve the unpleasant feelings (8).

Data suggest that a diminished ability to resist gambling thoughts leads to excessive gambling, especially in advanced phases of pathological gambling (9). However, important differences between PG and OCD exist. Patients with OCD frequently experience excessive doubt, a feature that is not characteristic of pathological gamblers (2,8). The compulsions of OCD are characterized by an increased sense of harm avoidance, risk aversion, and anticipatory anxiety (10). Pathological gamblers typically do not display these characteristics (11). In contrast to the egodystonic behaviors related to OCD, gambling in PG is often initially egosyntonic or hedonic in nature, although over time the pleasure derived from gambling may diminish. In this respect, the gambling in PG may be similar to drug use in drug dependence, and this and other phenomenological similarities have suggested that PG may represent a “behavioral addiction” (4,5,12,13). Several DSM-IV-TR criteria for PG resemble those of substance dependence: (a) presence of an intense desire to satisfy the need or craving to gamble or bet similar to that experienced by substance abusers; (b) loss of control over the substance use or behavior despite negative consequences, (c) It has been reported that about one third of PGs experience irritability, psychomotor agitation and difficulties concentrating following periods of gambling similar to withdraw symptoms. (d) PGs often increase the frequency of their gambling activities or the amount of money in order to achieve same levels of excitement sharing similarities with drug tolerance.

**Epidemiology**

The existing data on the clinical courses of PG and substance
abuse disorders (SUD) also suggest similarities. The ratio of men: women with PG (about 2:1) resembles that in SUD, more than the ratio seen in OCD, which is about 1:1 (14). Furthermore, a “telescoping” phenomenon has been reported for PG similar to the one reported in drug and alcohol dependence disorders in which female on average initially engage in disorder-related behavior at a later age but progress more quickly (“telescope”) than do men (15-17).

**Personality traits**

Another rationale for considering the addiction model for PG are personality traits common to both patients with SUD and PG. Personality measures suggest that individuals with PG, like those with substance dependence, are impulsive and sensation-seeking (18,19). whereas those with OCD are more harm-avoidant (20). When examining personality traits of novelty seeking, reward dependence and impulsivity among patients with PG and OCD, Kim and Grant (11) found that those with PG showed significantly greater scores of the previously mentioned traits whereas lower scores of anticipatory worry, fear of uncertainty, and harm avoidance than patients with OCD. Thus, although there are phenomenological and personality similarities between PG and OCD, those between PG and substance dependence appear more robust.

**Comorbidity**

Further support for the categorization of PG as a behavioral addiction is the high comorbidity with SUD. Results from general population surveys report high rates of substance use disorders among PG. Almost three quarters (73.2%) of pathological gamblers had an alcohol use disorder, 38.1% had a drug use disorder, 60.4% had nicotine dependence (12). Together, the high comorbidity rates between these disorders suggest common underlying etiological mechanisms.

Epidemiological studies examining the comorbidity of OCD and PG have had mixed results, with some (21) but not all studies observing an association between them (22). Linden et al. (23) found that 5 of 25 PG attending Gamblers Anonymous also met criteria for OCD. However, more than half of those met additional criteria for alcohol abuse or bipolar disorder, complicating the interpretation of the results. Similarly, a study of more than 700 individuals with OCD did not find elevated rates of pathological gambling (24). In neither the National Epidemiologic Survey on Alcoholism and Related Conditions, nor the Vietnam Era Twin samples were diagnostic assessments of OCD obtained. Thus, existing community-based data suggest a stronger connection between PG and a broad range of other psychiatric disorders than is found between PG and OCD.
**Familial Studies**

Familial comorbidities studies held mixed results. Black et al (25) conducted a familial association study of patients with OCD and found that family members of those with OCD were not more likely to report gambling problems than family members of healthy controls. In a larger study involving 343 family members of OCD patients, Bienvenu and colleagues (26) reported no increased rates of PG. Familial studies also highlighted comorbidities with SUD. Ramírez et al. (27) reported that 50% of PG had a parent who abused alcohol, while Roy et al (28) reported that of first-degree relatives of gamblers 33% had mood disorders and 24% abused alcohol.

**Neuropsychology**

Studies of performance on neurocognitive tasks targeting these processes have revealed differences between PG and healthy comparison subjects (29,30). Differences between PG and control subjects in decision-making task performance have been found (30), and these differences are similar to those between OCD and control subjects (31) and those between drug dependent and control subjects (32). In a recent study, Goudriaan and colleagues (33) compared 46 PGs with abstinent-alcohol dependent patients in a number of neurocognitive tasks and found that both the PG and the alcohol dependent groups were characterized by diminished performance on inhibition, time estimation, cognitive flexibility and planning tasks in similar magnitudes. The resemblance in the performance of both groups suggested common neurocognitive etiology for these disorders.

**Genetics**

Molecular genetic findings supported a substance use model for PG. Specifically, associations between polymorphisms of the dopamine D2 receptor gene (DRD2), the monoamine oxidase A gene (MAO-A), and the serotonin transporter gene (SLC6A4) have been reported in both pathological gamblers and drug abusers (34,35). The Taq-A1 polymorphism of the gene encoding the D2 dopamine receptor has been associated with PG, attention deficit hyperactivity disorder, Tourette’s syndrome, alcohol and drug abuse/dependence, antisocial behaviors, and poor inhibitory control (36,37). Although some of the same allelic variants (e.g., variants of the 5HT transporter gene) have been implicated in OCD and PG, the nature of the association has differed, with the long allele found in association with OCD and the short allele found in association with PG (38,39).

**Brain-imaging studies**

Frontostriatal circuitry has been implicated across species in tasks
involving impulsive and risk-taking choices (40) and also in human studies of PG (41). Brain-imaging highlighted a diminished activation of the ventromedial prefrontal cortex during gambling urges (7), cognitive control (42), and simulated gambling (43). On the contrary, increased activation of frontostriatal circuitry has been repeatedly observed in OCD (44), concurrent imaging investigation of PG, OCD, substance dependent and control is needed.

Treatment

Over the past decade our knowledge about effective treatments for PG has advanced considerably, however, no medication received FDA approval in gamblers yet. Serotonin reuptake inhibitors (SRIs) are the most frequently studied: clomipramine, fluvoxamine (45), paroxetine (46), citalopram (47) and recently escitalopram (48). The efficacy of serotonergic agents, as well as the result of clomipramine and meta-chlorophenylpiperazine m-CPP challenge studies that pointed out a dysfunction of the serotonergic system in PG (49), were long considered the base of the hypothesis of PG as part of the OCD spectrum.

However, although serotonergic agents have been demonstrated to be efficacy in at least one subgroup of patients, they have also been shown to exacerbate symptoms in at least another subgroup of gamblers (50). Furthermore, other studies suggested similarities between PG and SUD: naltrexone, a treatment effective for alcohol and opiate dependence, has shown efficacy in the treatment for PG (51). In contrast, the opioid antagonist naloxone has been associated with symptom exacerbation with OCD (52). Furthermore, the opioid antagonist nalmefene was shown to have efficacy in PG treatment (53), and similarly, bupropion (54). In addition, whereas mood stabilizers like lithium may be helpful in groups of subjects with PG (55), their efficacy in OCD seems questionable (56).

Cognitive-behavioral interventions, specifically those relying on abstinence reinforcement and relapse-prevention strategies have been shown effective in the treatment of PG (57). Cognitive-behavioral therapy is also an established treatment for OCD but modeled on exposure and prevention of response, in which repeated and prolonged exposure is believed to provide information that disconfirms mistaken associations and evaluations held by the patient and promotes habituation to previously fearful thoughts and situations (58). Cognitive-behavioral approaches in PG tend to be modeled after the ones with demonstrated efficacy in the treatment of drug addiction, rather than the exposure/response prevention strategies that are
effective for treating OCD. Moreover, existing data suggest that PG may respond similarly to 12-step programs (e.g. Gamblers Anonymous) as individuals with SUD in self-help groups (e.g. Alcoholics Anonymous, Narcotics Anonymous).

**Conclusion**

Although PG resembles OCD in some domains, the majority of the existing data suggests substantial differences between them. Findings from phenomenology, epidemiology, treatment response and imaging study appear to support that PG resembles more closely an addiction. Nevertheless, despite our progress over the last decade in understanding PG, SUD and OCD, existing data are often limited and include methodological concerns that complicate interpretation and comparisons across subject groups. Future studies, particularly those in the areas of genetics, neuropsychology, and imaging, are likely to lead to an improved understanding of the mechanisms linking PG, OCD and SUD.

**References**


49. Pallanti S, Bernardi S, Quercioli L, DeCaria C, Hollander E. Serotonin dysfunction in pathological gamblers: increased prolactin response to oral m-CPP versus placebo. CNS Spectr. 2006;11(12):956-64.


Psychopharmacol. 1991;11(3):175-84.


Conflicto de interés: los autores manifiestan que no tienen ningún conflicto de interés en este artículo.

Recibido para evaluación: 15 de abril del 2010
Acceptado para publicación: 24 de julio del 2010

Correspondencia
Carlos Blanco
Department of Psychiatry
of the New York State Psychiatric Institute
Columbia University
cb255@columbia.edu