Pathological gambling and compulsive buying: do they fall within an obsessive-compulsive spectrum?

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Both compulsive buying (CB) and pathological gambling (PG) have been proposed as members of a spectrum of disorders related to obsessive-compulsive disorder (OCD). The spectrum hypothesis originated in the early 1990s and has gained considerable support, despite the lack of empirical evidence. Interest in this hypothesis has become critical because some investigators have recommended the creation of a new category that includes these disorders in DSM-5*, now under development. In this article, the authors describe the origin of the obsessive-compulsive (OC) spectrum and its theoretical underpinnings, review both CB and PG, and discuss the data both in support of and against an OC spectrum. Both disorders are described in terms of their history, definition, classification, phenomenology, family history, pathophysiology, and clinical management. The authors conclude that: (i) CB and PG are probably not related to OCD, and there is insufficient evidence to place them within an OC spectrum in* DSM-V*; (ii) PG should stay with the impulse-control disorders (ICDs); and (iii) a new diagnosis of CB should be created and be classified as an ICD.*

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In the early 1990s, interest began to grow around the concept of an obsessive-compulsive (OC) spectrum. Hollander and others[1-3] wrote of a spectrum of disorders related to obsessive-compulsive disorder (OCD). Based on his experience as an OCD researcher, Hollander considered OCD to be at the center of the spectrum, and described its breadth and overlap with many other psychiatric disorders. These disorders were considered to lie along orthogonal axes of impulsivity vs compulsiveness, uncertainty vs certainty, and cognitive vs motoric (features). The OC spectrum concept was quickly embraced by other investigators because it offered a new way to think about the relationship among many neglected disorders, and it potentially offered new treatment options.[4,5] Not all investigators have agreed, and several critical reviews have appeared.[6-9]

Despite the criticism, the concept of a group of disorders being related to OCD remains of great theoretical interest. The idea that disorders are related is crucial to classification schemes, and why should a group of disorders *not* be related to OCD? This question is now of singular interest because those responsible for developing the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)* must decide whether to create a separate category for OCD and potentially related disorders, or to keep OCD with the anxiety disorders. If

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Selected abbreviations and acronyms

CB	*compulsive buying*
ICD	*impulse-control disorder*
OC	*obsessive-compulsive*
OCD	*obsessive-compulsive disorder*
PG	*pathological gambling*
SSRI	*selective serotonin reuptake inhibitor*

they create a new category for the OC spectrum they will need to determine its breadth.

The OC spectrum's boundaries have expanded or contracted according to the views of the investigator concerned. It has been described as including disorders of impulse control such as pathological gambling (PG), trichotillomania, and kleptomania; Tourette's and other tic disorders; impulsive personality disorders (eg, borderline personality disorder); hypochondriasis and body dysmorphic disorder; eating disorders; and several disorders not currently recognized in *DSM-IV-TR*[10] such as compulsive buying (CB) and sexual addiction.[1-4] Few investigators have offered evidence to validate a relationship among the disorders. Typically, such evidence might include comparisons of phenomenology, natural history, family history, biological markers, and treatment response.[11]

OCD holds an important place at the center of the spectrum. Currently classified in *DSM-IV-TR*[10] as an anxiety disorder, OCD is independent of other anxiety disorders in the International Classification of Diseases (ICD) system,[12] and a strong rationale has been presented by Zohar et al[13] for its separation from these disorders. First, OCD often begins in childhood, whereas other anxiety disorders typically have a later age of onset. OCD has a nearly equal gender distribution, unlike the other anxiety disorders, which are more common in women. Studies of psychiatric comorbidity show that, unlike the other anxiety disorders, persons with OCD generally tend not to have elevated rates of substance misuse. Family studies have not shown a clear association between OCD and the other anxiety disorders. Brain circuitry that mediates OCD appears to be different from that involved in other anxiety disorders. Lastly, OCD is unique with regard to its response to the serotonin reuptake inhibitors (SSRIs), while noradrenergic medications, effective in mood disorders, and somewhat effective in anxiety disorders, are largely ineffective in OCD. On the other hand, the benzodiazepines, which have little effect on OCD, are often effective for the other anxiety disorders. Further, Zohar et al[13] have argued that recognizing the spectrum would contribute to improved classification, thus enabling a more precise description of endophenotype and biological markers that characterize these conditions, and that better classification could lead to more specific treatments.

Apart from the possibility of an OC spectrum, there has been no consistent approach to categorizing impulsive and compulsive disorders. While some have decried the "medicalization" of problematic behaviors such as CB,[14] discussion has mainly focused on how these disorders should be classified, their relationship to other putative OC spectrum disorders, and whether some of them stand alone as independent disorders (eg, CB, compulsive sexual behavior).

Alternative classification schemes have emphasized the relationship of a putative OC spectrum disorder to depression or other mood disorders, to the impulse-control disorders (ICDs), or to the addictive disorders. Recently, it has been suggested that at least some of the disorders included in the OC spectrum be placed within a new diagnostic category that combines behavioral and substance addictions.[15] "Behavioral addictions" include disorders that the National Institute on Drug Abuse (NIDA) considers to be relatively pure models of addiction because they are not contaminated by the presence of an exogenous substance.

With this background in mind, this article will focus on the status of PG and CB. Are these disorders part of an OC spectrum as defined by Hollander and coworkers? Are they more appropriately considered impulse control disorders (ICDs) or addictions? Are they related to one another? These and other questions will be considered as we explore CB, PG, and the OC spectrum.

Compulsive buying

CB has been described in the psychiatric nomenclature for nearly 100 years. German psychiatrist Emil Kraepelin[16] wrote about the uncontrolled shopping and spending behavior called *oniomania* ("buying mania"). He was later quoted by Swiss psychiatrist Eugen Bleuler[17] in his *Lehrbuch der Psychiatrie*:

> As a last category, Kraepelin mentions the buying maniacs (oniomaniacs) in whom even buying is compulsive and leads to senseless contraction of debts with continuous delay of payment until a catastrophe clears the situation a little – a little bit never altogether because they never admit all their debts. …. The particular element is impul-

siveness; they cannot help it, which sometimes even expresses itself in the fact that not withstanding a good school intelligence, the patients are absolutely incapable of thinking differently and conceiving the senseless consequences of their act, and the possibilities of not doing it." (p 540).

Kraepelin and Bleuler each considered "buying mania" an example of a *reactive impulse* or *impulsive insanity*, and placed it alongside kleptomania and pyromania. They may have been influenced by French psychiatrist Jean Esquirol's[18] earlier concept of *monomania*, a term he used to describe otherwise normal persons who had some form of pathological preoccupation.

CB attracted little attention until the late 1980s and early 1990s when consumer behavior researchers showed the disorder to be widespread[19-21] and descriptive studies appeared in the psychiatric literature.[22-25] McElroy et al[22] developed an operational definition that encompasses the cognitive and behavioral aspects of CB. Their definition requires evidence of impairment from marked subjective distress, interference in social or occupational functioning, or financial/legal problems. Further, the syndrome could not be attributed to mania or hypomania.

Other definitions have come from consumer behavior researchers or social psychologists. Faber and O'Guinn[26] defined the disorder as "chronic buying episodes of a somewhat stereotyped fashion in which the consumer feels unable to stop or significantly moderate his behavior" (p 738). Edwards,[27] another consumer behaviorist, suggests that compulsive buying is an "abnormal form of shopping and spending in which the afflicted consumer has an overpowering uncontrollable, chronic and repetitive urge to shop and spend (that functions) … as a means of alleviating negative feelings of stress and anxiety." (p 67). Dittmar[28] describes three cardinal features: irresistible impulse, loss of control, and carrying on despite adverse consequences. Some consumer behavior researchers consider CB part of a spectrum of aberrant consumer behavior, which includes pathological gambling, shoplifting, and credit abuse).[29]

CB is not included in either the *DSM-IV-TR*[10] or the World Health Organization *International Classification of Diseases, Tenth Edition*.[12] Whether to include CB in *DSM-5* is being debated.[30] McElroy et al[23] suggest that compulsive shopping behavior might be related to "mood, obsessive-compulsive or impulse control disorders." Lejoyeux et al[31] have linked it to the mood disorders. Some consider CB to be related to the substance use disorders.[32,33] Others suggest classifying CB as a disorder of impulse control[34] or a mood disorder.[35]

Faber and O'Guinn[26] estimated the prevalence of CB at between 1.8% and 8.1% of the general population, based on results from a mail survey in which the Compulsive Buying Scale (CBS) was administered to 292 individuals selected to approximate the demographic makeup of the general population of Illinois. (The high and low prevalence estimates reflect different score thresholds set for CB.) More recently, Koran et al[36] used the CBS to identify compulsive buyers in a random telephone survey of 2513 US adults, and estimated the point prevalence at 5.8% of respondents. Grant et al[37] utilized the MIDI to assess CBD and reported a lifetime prevalence of 9.3% among 204 consecutively admitted psychiatric inpatients.

CB has an onset in the late teens/early 20s, which may correlate with emancipation from the nuclear family, as well as with the age at which people can first establish credit.[34] Research suggests that 80% to 94% of persons with CBD are women.[38] In contrast, Koran et al[36] reported that the prevalence of CBD in their random telephone survey was nearly equal for men and women (5.5% and 6.0%, respectively). Their finding suggests that the reported gender difference may be artifactual, in that women more readily acknowledging abnormal shopping behavior than men. Men are more likely to describe their compulsive buying as "collecting."

Data from clinical studies confirm high rates of psychiatric comorbidity, particularly for the mood (21% to 100%), anxiety (41% to 80%), substance use (21% to 46%), and eating disorders (8% to 35%).[38] Disorders of impulse control are also relatively common (21% to 40%). The frequency of Axis II disorders in individuals with CB was assessed by Schlosser et al[25] using a self-report instrument and a structured interview. Nearly 60% of 46 subjects met criteria for at least one personality disorder through a consensus of both instruments. The most commonly identified personality disorders were the obsessive-compulsive (22%), avoidant (15%), and borderline (15%) types.

A distinctive and stereotyped clinical picture of the compulsive shopper has emerged. Black[39] has described four phases including: (i) anticipation; (ii) preparation; (iii) shopping; and (iv) spending. In the first phase, the person with CB becomes preoccupied either with having a specific item, or with the act of shopping. This is followed by a preparation phase in which plans are made. This

phase is followed by the actual shopping experience, which many individuals with CB describe as intensely exciting.[25] The act is completed with the purchase, often followed by a sense of let-down or disappointment.[36]

Perhaps the hallmark of CB is preoccupation with shopping and spending. This typically leads the individual to spend many hours each week engaged in these behaviors.[24,25] Persons with CB often describe increasing tension or anxiety that is relieved when a purchase is made. CB behaviors occur all year, but can be more problematic during the Christmas season and other holidays, as well as around the birthdays of family members and friends. Compulsive buyers are mainly interested in consumer goods such as clothing, shoes, crafts, jewelry, gifts, makeup, and compact discs (or DVDs)[24,25] CB has little to do with intellect or educational level, and has been documented in mentally retarded persons.[40] Similarly, income has relatively little to do with CB, because persons with a low income can be as preoccupied with shopping and spending as wealthier individuals.[38,40]

Nataraajan and Goff[42] have identified two independent factors in CB: (i) buying urge or desire, and (ii) degree of control over buying. In their model, compulsive shoppers combine high urge with low control. This view is consistent with clinical reports that compulsive buyers are preoccupied with shopping and spending and will try to resist their urges, often with little success.[24,38]

Cross-sectional studies suggest the disorder is chronic, though fluctuating in severity and intensity.[22,25] Aboujaoude et al[43] reported that persons who responded to treatment with citalopram were likely to remain in remission during a 1-year follow-up, suggesting that treatment can alter the natural history of the disorder. Lejoyeux et al[44] report that CB is associated with suicide attempts, although there are no reports of the disorder leading to completed suicide.

There is some evidence that CB runs in families and that within these families mood, anxiety, and substance-use disorders exceed population rates. Black et al[45] used the family history method to assess 137 first-degree relatives of 31 persons with CB. Relatives were significantly more likely than those in a comparison group to have depression, alcoholism, a drug use disorder, "any psychiatric disorder" and "more than one psychiatric disorder." CB was identified in nearly 10% of the first-degree relatives, but was not assessed in the comparison group.

Neurobiologic theories have centered on disturbed neurotransmission, particularly involving the serotonergic, dopaminergic, or opioid systems. Selective serotonin reuptake inhibitors (SSRIs) have been used to treat CB,[46-50] in part because of hypothetical similarities between CB and OCD, a disorder known to respond to SSRIs. Dopamine has been theorized to play a role in "reward dependence," which has been claimed to foster behavioral addictions, such as CB and PG.[15] Case reports suggesting benefit from the opioid antagonist naltrexone have led to speculation about the role of opioid receptors[51] There is no direct evidence, however, to support the role of these neurotransmitter systems in the etiology of CB.

Because CB occurs mainly in developed countries, cultural and social factors have been proposed as either causing or promoting the disorder.[39] Interestingly, Neuner et al[52] reported that the frequency of CB in Germany increased following reunification, suggesting that societal factors can contribute to the development of CB. These may include the presence of a market-based economy, the availability of goods, easily obtained credit, and disposable income.[14]

There are no standard treatments, and both psychotherapy and medication have been recommended. Several case studies report the psychoanalytic treatment of CB.[53-55] More recently, cognitive-behavioral treatment (CBT) models have been developed for CB, many of them employing group therapy.[56,57] Mitchell et al[57] found that group CBT produced significant improvement compared with a wait-list in a 12-week pilot study. Improvement attributed to CBT was maintained during a 6-month follow-up. Benson[58] has developed a comprehensive self-help program that can be used by both individuals and groups.

Treatment studies employing psychotropic medications have produced mixed results. Early reports suggested the benefit of antidepressants in treating CB[22,23] Black et al[46] reported the results of an open-label trial in which subjects given fluvoxamine showed benefit. Two subsequent randomized controlled trials (RCTs) found fluvoxamine treatment to be no better than placebo.[47,48] Koran et al[51] later reported that subjects with CB improved with open-label citalopram. In a subsequent study, subjects received open-label citalopram; those who were considered responders were randomized to citalopram or placebo. Compulsive shopping symptoms returned in 5/8 subjects (62.5%) assigned to placebo compared with 0/7 who continued taking citalopram. In an identically designed discontinuation trial, escitalopram did not separate from placebo.[52] Because the medication study findings are mixed, no empirically well-sup-

ported treatment recommendations can be made. Open-label trials have generally produced positive results, but RCTs have not. Interpretation of these study results is complicated by placebo response rates as high as 64%.[47]

Pathological gambling

PG is increasingly being recognized as a major public health problem.[59] PG is estimated to cost society approximately $5 billion per year and an additional $40 billion in lifetime costs for reduced productivity, social services, and creditor losses.The disorder substantially impairs quality of life in addition to its association with comorbid psychiatric disorders, psychosocial impairment, and suicide.[59-61] Family-related problems include financial distress, child and spousal abuse, and divorce and separation.[61]

While problematic gambling behavior has been recognized for centuries, it was often ignored by the psychiatric community. Bleuler,[17] citing Kraepelin,[16] considered PG, or "gambling mania," a *special impulse* disorder. Criteria for PG were first enumerated in 1980 in *DSM-III*.[62] The criteria were subsequently modified, and in *DSM-IV-TR*,[10] are patterned after those used for substance dependencies and emphasize the features of tolerance and withdrawal. PG is defined as "persistent and recurrent maladaptive gambling behavior (criterion A) that disrupts personal, family, or vocational pursuits…" Ten specific maladaptive behaviors are listed, and ≥5 are required for the diagnosis. The criteria focus on loss of control of gambling behavior; progressive deterioration of the disorder; and continuation despite negative consequences. The diagnosis can only be made when mania is ruled out (Criterion B). In an attempt to reconcile nomenclature and measurement methods, Shaffer and Hall[63] developed a generic multilevel classification scheme that is now widely accepted by gambling researchers.

PG is presently classified as a disorder of impulse control in *DSM-IV-TR*.[10] On the one hand, some investigators have suggested that PG is related to OCD,[1,64] yet others argue against such a relationship.[65] On the other hand, PG is widely considered an addictive disorder.[66,67] It has recently been proposed as a candidate for inclusion in a new category for "behavioral addictions." [15]

Recent estimates of lifetime prevalence for PG range from 1.2% to 3.4% in the general population.[68,69] Prevalence rates have risen in areas where gambling availability has increased.[70,71] A national survey showed that the availability of a casino within 50 miles is associated with a nearly twofold increase in PG prevalence.[59] Gambling behavior typically begins in adolescence, with PG developing by the late 20s or early 30s,[72] though it can begin at any age through senescence. Rates of PG are higher in men, but the gender gap may be narrowing.PG has a later onset in women yet progresses more rapidly ("telescoping") than in men,[73] at a rate similar to that observed in alcohol disorders. Populations at risk include adults with mental health or substance-use disorders, persons who have been incarcerated, African-Americans, and persons with low socioeconomic status.[74,75]

Research has not validated PG subtypes, but perhaps the most widely discussed distinction is between "escape-seekers" and "sensation-seekers." [76] Escape-seekers are often older persons who gamble out of boredom, from depression, or to fill time, and choose passive forms of gambling such as slot machines. Sensation-seekers tend to be younger, and prefer the excitement of card games or table games that involve active input.[76] Blaszczynski and Nower[77] have proposed a "pathways" model that integrates biological, developmental, cognitive, and other determinants of disordered gambling. They have identified three subgroups: a) behaviorally-conditioned gamblers; b) emotionally vulnerable gamblers; and c) antisocial, impulsive gamblers. Behaviorally conditioned gamblers have no specific predisposing psychopathology, but make bad judgments regarding gambling. Emotionally vulnerable gamblers suffer premorbid depression or anxiety, and have a history of poor coping. Finally, antisocial, impulsive gamblers are highly disturbed and have features of antisocial personality disorder and impulsivity that suggest neurobiological dysfunction.

Psychiatric comorbidity is the rule, not the exception, in persons with PG. Both community and clinic-based studies suggest that substance use disorders, mood disorders, and personality disorders are highly prevalent in persons with PG.[78] In clinical samples, from 25% to 63% of pathological gamblers meet lifetime criteria for a substance use disorder.[79] Correspondingly, from 9% to 16% of substance abusers are probable pathological gamblers.[79] PG is also associated with increased prevalence of mood disorders, and overall 13% to 78% of persons with pathological gambling are estimated to experience a mood disorder.[79] On the other hand, patients with mood disorders have not been found to have elevated rates of PG.

Rates of other impulse-control disorders (ICDs) appear higher in persons with pathological gambling than in the

general population. Investigators have reported rates ranging from 18% to 43% for one or more ICD.[79] CB appears to be the most frequent comorbid ICD in persons with PG, perhaps because both disorders share characteristics of focused attention, monetary gratification, and monetary exchange. Subjects with one ICD appear more likely to have another, suggesting considerable overlap among them.

Personality disorders are relatively common among individuals with PG, particularly those in "cluster B." Antisocial personality disorder has been singled out as having a close relationship with PG, perhaps because crime and gambling frequently co-occur, with rates ranging from 15% to 40%.[79,80] At least one study of persons with antisocial personality disorder showed high rates of PG.[81]

PG is widely thought to be chronic and progressive.[82,83] This view is embedded in *DSM-IV-TR*[10] which holds that the essential feature of PG is "persistent and recurrent maladaptive gambling behavior …that disrupts personal, family, or vocational pursuits" (p 671). These views were influenced by the pioneering observations of Custer[84] who described PG as a progressive, multistage illness that begins with a *winning phase*, followed in turn by a *losing phase*, and a *desperation phase*. The final phase, *giving up*, represented feelings of hopelessness.[85] Some contend that many pathological gamblers experience a "big win" early in their gambling careers that leads directly to their becoming addicted. Custer's four phases of PG have gained wide acceptance despite the absence of empirical data.

Recent work is leading to a reconsideration of these views. LaPlante et al[86] reviewed five studies[87-91] that met their criteria of reporting longitudinal data pertaining to gambling that did not involve a treatment sample. LaPlante et al report that, from the four studies that included level 3 gamblers (ie, persons with PG), most gamblers improved, and moved to a lower level, and that rates of classification improvement were "at least significantly greater than 29%." Results were similar for level 2 (ie, "at-risk") gamblers. Those who were level 0 to 1 gamblers at baseline were unlikely to progress to a higher (ie, more severe) level of gambling behavior, and with one exception,[91] the studies suggested that few level 2 gamblers improved by moving to level 1. La Plante et al[86] conclude that these studies challenge the notion that PG is intractable, and suggest that many gamblers spontaneously improve, as do many substance addicted persons. The findings suggest that those who do not gamble or gamble without problems tend to remain problem-free; those with disordered gambling move from one level to another, though the general direction is toward improved classification.

Family history data suggests that PG, mood disorders, and substance-use disorders are more prevalent among the relatives of persons with PG than in the general population.[92,93] Twin studies also suggest that gambling has a heritable component.[94] Functional neuroimaging studies suggest that among persons with PG, gambling cues elicit gambling urges and a temporally dynamic pattern of brain activity changes in frontal, paralimbic, and limbic brain structures, suggesting to some extent that gambling may represent dysfunctional frontolimbic activity.[95]

There is little consensus about the appropriate treatment of PG. Few persons with PG seek treatment,[96] and until recently the treatment mainstay appeared to be participation in Gamblers Anonymous (GA), a 12-step program patterned after Alcoholics Anonymous. Attendance at GA is free and chapters are available throughout the US, but follow-through is poor and success rates disappointing.[97] Inpatient treatment and rehabilitation programs similar to those for substance-use disorders have been developed, and are helpful to some[98,99] Still, these programs are unavailable to most persons with PG because of geography or lack of access (ie, insurance/financial resources). More recently, CBT and motivational interviewing have been become established treatment methods.[100] Self-exclusion programs have also gained acceptance and appear to benefit selected patients.[101] While rules vary, they generally involve voluntary self-exclusion from casinos for a period of time at the risk of being arrested for trespassing. Medication treatment studies have gained momentum, but their results are inconsistent. Briefly, the opioid antagonists naltrexone and nalmefene were superior to placebo in randomized controlled trials (RCTs)[102,103] but controlled trials of paroxetine and bupropion were negative.[104,105] Open-label studies of nefazodone, citalopram, carbamazepine, and escitalopram have been encouraging, but need to be followed up with adequately powered and controlled studies.[106-109]

Putative relationship between CB/PG and OCD

The relationship between CB/PG and OCD remains uncertain. The inclusion of CB and PG within an OC spectrum, while intriguing, rests on hypothesis and not

empirical data. How these disorders should be classified has been debated for nearly 100 years. Opinion has mainly favored their inclusion among disorders of impulse control. For historical reasons, and because of the lack of empirical data, we believe that the two disorders should remain with the ICDs until convincing evidence is presented to favor their inclusion either with the addictive disorders or an OC spectrum.

The most obvious connection between CB and PG and OCD is phenomenologic. Each disorder involves repetitive behavior that generally occurs in response to overwhelming thoughts and urges; engaging in the behavior—at least temporarily—will satisfy the urge, and/or reduce tension and anxiety that preceded the behavior. Nonetheless, a fundamental distinction between CB/PG and OCD is that the behaviors (shopping, gambling) are considered *ego-syntonic*; that is, they are viewed as pleasurable and desirable, while behaviors associated with OCD never are, and nearly all patients want to be rid of them. Not so with shopping and gambling: the person with CB or PG finds the behaviors highly pleasurable, and only wants to stop the behaviors when their deleterious secondary consequences become overwhelming.

Proponents of the OC spectrum point to the overlap between these disorders and OCD. Comorbidity studies have found that in clinical samples from 3% to 35% of individuals with CB have comorbid OCD.[22,46] In fact, the presence of CB may characterize a specific subset of OCD patients,[110,111] particularly those who hoard. Hoarding is a special symptom that involves the acquisition of and failure to discard, possessions that are of limited use or value.[112] Yet, unlike the items retained by the typical hoarder, the items purchased by the person with CB are not inherently valueless or useless.

CB frequently appears to be comorbid with the ICDs. Black and Moyer[80] and Grant and Kim[72] each reported elevated rates of CB among samples of pathological gamblers (23% and 8%, respectively). Likewise, other impulse control disorders are common among compulsive shoppers.[39] Comorbidity studies of PG are more mixed, although they generally report higher rates of OCD than in the general population. The reverse does not seem to be true. Axis II comparisons show that the predominant disorders associated with OCD are the "cluster C" disorders. While there are no axis II disorders specifically associated with PG or CB, "cluster B" disorders appear overrepresented, particularly antisocial personality disorder.

Direct investigations into OC characteristics of persons with PG found that those with PG scored higher than those without on scales measuring OC traits.[64] CB and PG also share high trait impulsivity.[19,113]

Other evidence could come from family studies of CB, PG, or OCD. There are few family studies regarding these disorders, and none have supported a familial relationship among these disorders. In the only controlled family history study of CB, Black et al[45] did not find a relationship with OCD. In two family studies, one using the family history method, the other using the family interview method, the investigators were unable to establish a connection between PG and OCD.[114,115] Looking at this connection through OCD family studies has also failed to find a connection. Neither Black et al[114] nor Bienvenu et al[115] were able to establish a familial relationship between OCD and PG.

Demographic similarities are often used to suggest that disorders might be linked, for example the fact that both alcohol disorders and antisocial personality disorder are predominantly found in men. Yet, there is no similarity in gender distribution among these disorders. With PG there is a clear male preponderance; with CB a female preponderance; with OCD, the gender distribution is evenly split.

If these disorders were related, their natural history and course might be similar as well. CB and OCD appear to have an onset in the late teens or early 20s. PG appears to have a slightly later onset, with women developing the disorder much later than men, but having a briefer course from onset of gambling to development of a disorder. This is what is seen with alcohol disorders, but not OCD. With CB, PG, and OCD are all considered mostly chronic, but the similarity stops there. For CB and PG, while there are no careful, longitudinal studies, the data suggest that the disorders may be episodic, that is, may remit for varying lengths of time depending on a host of external factors such as fear of consequences, eg, bankruptcy or divorce, or lack of income; OCD rarely remits. In terms of suicide risk, PG has been reported to carry a risk for suicide attempts and completed suicides; with CB, there are anecdotal reports of suicide attempts, but not completed suicides; with OCD, the data is somewhat mixed, but overall, the risk of completed suicide is considered low.

Here, too, when one considers treatment response, OCD is well known to respond well to serotonin reuptake inhibitor antidepressants, and to cognitive behavioral therapy. CB and PG have no clear response to medica-

tion, and the most robust treatment data suggests that PG may respond to opioid antagonists. Both CB and PG are reported to respond to CBT, but the completeness and quality of the response is unlike that seen with OCD.

The presence of similar biological markers is another way to assess the connection between these disorders. This task is hampered by the fact that none of these disorders has reliable markers. Nonetheless, a functional magnetic resonance imaging (fMRI) study of PG suggests that the disorder shows an abnormal pattern of activation in specific subcortical-frontal regions following cue exposure. Potenza et al[86] interpret these findings as evidence for the similarity of brain pathways in PG and drug addiction, while the opposite direction of higher brain activation is found in OCD. Similarly, Goodriaan et al[116] review the research on neurochemical and molecular genetic data involving PG. They conclude that there is evidence of disturbed neurotransmission involving dopamine (DA), serotonin, and norepinephrine; and "… are in accordance with the findings of abnormal brain activation in reward pathways, where DA is an important transmitter" (p 134). Dopamine is noted to play an important role in craving and withdrawal in the substance use disorders. While the neurotransmission involved in OCD has not been fully elucidated, the central serotonin system has been the most actively studied. This is perhaps due to the robust effect of SSRIs in the treatment of OCD.

On the whole, neuropsychological studies of PG indicate that pathological gamblers have impaired performance in several aspects of executive function including attention, delay discounting, and decision-making.[115-117] With OCD, neuropsychological research is less consistent; there is evidence of impaired response-inhibition and in attentional set-shifting, but little evidence of impaired reversal learning and decision-making.[118] To our knowledge, there are no neuropsychological studies of persons with CB.

Alternate classification schemes

If CB and PG are not part of an OC spectrum, where should they be classified? Because there is almost no evidence suggesting a relationship with the mood disorders, that possibility can probably be eliminated outright. Of the remaining schemes, the most likely candidates are to include PG and CB with the ICDs, or to move them to a category involving the substance-use disorders.

Keeping PG and CB with the ICDs is the easiest option: PG is already classified as an ICD, and while CB is not currently included in *DSM-IV-TR*, it has historically been considered an impulsive disorder. Both PG and CB share similar clinical features involving the presence of irresistible, ego-syntonic urges that prompt a behavioral response. The response (ie, gambling, shopping) satisfies the urge and/or temporarily reduces tension or anxiety, but is often followed by a sense of guilt or shame, and ultimately leads to adverse, secondary consequences. The behaviors are chronic or intermittent, and may spontaneously remit, sometimes in response to external circumstances. Age of onset and gender distribution differ, as discussed earlier. Possibly, CB may be considered the female equivalent of PG, because they tend to have a reverse gender distribution: men predominate among those with PG; women predominate among those with CB. Both appear to respond to CBT, yet neither has a clear response to medication; SSRIs do not produce consistent improvement. Comorbidity studies show overlap among the disorders, as a disproportionate number of pathological gamblers have CB and vice versa.

On the other hand, data suggest many commonalities with the substance use disorders. PG and CB are both associated with cravings that are not unlike those reported by substance abusers; PG is noted to produce "withdrawal" symptoms when the gambler is abstinent,[119] though this has not been studied in CB. Research shows that persons with PG or CB often have comorbid substance use disorders. Conversely, substance abusers have high rates of PG; there are no comparable data for CB. Family studies show that relatives of probands with PG or CB have high rates of psychiatric illness, particularly alcohol and drug use disorders. Further, Slutske et al[94] have reported that, based on twin data, PG appears to be related to the substance-use disorders and antisocial personality disorder. Finally, as noted earlier, the neuroimaging studies, and both neurotransmitters and molecular genetic research on PG suggest a relationship with the substance-use disorders.[116] These data support the inclusion of PG and perhaps CB in a category for "behavioral addictions," possibly comprising a subset of the substance-use disorders, but they do not support a relationship with OCD.

Conclusions

The review suggests that CB and PG are probably not candidates for inclusion in an OC spectrum. The review was not meant to judge the merit of the OC spectrum concept.

In fact, we have suggested that there appears to be sufficient evidence to support the existence of a limited OC spectrum that might include body dysmorphic disorder, Tourette's disorder, trichotillomania, subclinical OCD, and perhaps the grooming disorders.[8,120] While there are superficial phenomenologic similarities between CB/PG and OCD, other evidence suggests they are not associated: gender distribution, age at onset, and course; comorbidity studies; neuroimaging, neurotransmitter, and neuropsychological studies; and treatment response. We believe that PG and CB are likely related, despite their much different gender distribution. Further, we believe that in the absence of new and convincing evidence, PG ought to remain within the ICD category. Lastly, we believe that CB is an identifiable and distinct disorder that ought to be included in *DSM-5*, and should be included with the ICDs. ❑

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El juego patológico y el comprar compulsivo: ¿corresponde incluirlos dentro del espectro obsesivo-compulsivo?

Se ha propuesto que el comprar compulsivo (CC) y el juego patológico (JP) se integren en el espectro de los trastornos relacionados con el trastorno obsesivo compulsivo (TOC). La hipótesis del espectro se originó a comienzos de la década de 1990 y ha conseguido bastante apoyo, a pesar de la falta de evidencias empíricas. El interés en esta hipótesis ha llegado a un punto crítico ya que algunos investigadores han recomendado la creación de una nueva categoría que incluya estos trastornos en el DSM-V, que está actualmente en desarrollo. En este artículo los autores describen el origen del espectro obsesivo-compulsivo (OC) y sus fundamentos teóricos, revisan el CC y el JP, y discuten los datos a favor y en contra de un espectro OC. Ambos trastornos son descritos en términos de su historia, definición, clasificación, fenomenología, historia familiar, fisiopatología y manejo clínico. Los autores concluyen que: 1) el CC y el JP probablemente no se relacionan con el TOC y no es suficiente la evidencia para incluirlos en el espectro OC dentro del DSM-V, 2) el JP debiera incluirse dentro de los trastornos del control impulsivo (TCI) y 3) se debe crear un nuevo diagnóstico del CC y clasificarlo como un TCI.

Jeu pathologique et achat compulsif : font-ils partie du spectre des troubles obsessionnels-compulsifs ?

Certains auteurs ont proposé d'intégrer l'achat compulsif (AC) et le jeu pathologique (JP) dans le spectre des troubles obsessionnels-compulsifs (TOC), concept émergeant au début des années 90, et qui a reçu un soutien important en dépit d'un manque de preuves empiriques. L'intérêt pour cette hypothèse est devenu très important en raison de la recommandation de certains experts de créer une nouvelle catégorie incluant ces troubles dans le DSM-5 actuellement en rédaction. Dans cet article, les auteurs décrivent l'origine des troubles obsessionnels-compulsifs (TOC) et de leurs bases théoriques, analysent le JP et l'AC et examinent les arguments pour et contre leur appartenance au spectre des TOC. Les deux pathologies sont décrites en termes d'historique, de définition, de classification, de phénoménologie, d'antécédents familiaux, de physiopathologie et de prise en charge clinique. Les auteurs concluent que : (i) le JP et l'AC ne sont probablement pas liés aux TOC et que les preuves sont insuffisantes pour les placer dans le cadre OC du DSM-V ; (ii) le JP devrait rester au sein des troubles du contrôle de l'impulsion (TCI) ; et (iii) une nouvelle définition de l'AC devrait être créée pour le classer également dans les TCI.

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