

Impulse Control Disorders: Clinical Characteristics and Pharmacological Management

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This article reviews the current knowledge of the clinical characteristics and pharmacological management of pathological gambling, kleptomania, and compulsive buying. Specifically, the article summarizes the phenomenology and associated psychopathology of these disorders and presents study results of the various pharmacological agents used to treat these disorders—serotonin reuptake inhibitors, opioid antagonists, mood stabilizers, and atypical antipsychotics.

Keywords Impulse control disorders; Pathological gambling; Kleptomania; Compulsive buying; Pharmacology; Phenomenology.

INTRODUCTION

The impulse control disorders (ICDs) not elsewhere classified include pathological gambling, kleptomania, intermittent explosive disorder, trichotillomania, pyromania, and impulse control disorders not otherwise specified, which may include compulsive Internet use, compulsive sexual behavior, and compulsive buying (1). The basic characteristics of impulse control disorders are: 1) repetitive or compulsive engagement in a behavior despite adverse consequences; 2) diminished control over the problematic behavior; 3) an appetitive urge or craving state prior to engagement in the problematic behavior; and 4) a hedonic quality during the performance of the problematic behavior.

This article reviews the clinical characteristics, associated psychopathology, and pharmacological management of three of these understudied disorders: pathological gambling, kleptomania, and compulsive buying. Although perhaps relatively common and significantly disabling, these disorders often go undiagnosed and untreated.

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EPIDEMIOLOGY

The precise prevalences of ICDs are currently incompletely known. Some evidence suggests that the ICDs may in fact be relatively common. Based upon several national studies of pathological gambling, lifetime rates have been estimated as ranging from 0.8% to 1.5%, with past-year rates ranging from 0.1% to 0.9% (2). Slightly higher but comparable estimates (lifetime rate of 1.6%, past-year rate of 1.1%) were derived from a meta-analysis of prevalence estimate surveys completed in North America over the past 30 years (3).

Other ICDs, such as kleptomania and compulsive buying, have not generated detailed epidemiological studies. Preliminary evidence, however, suggests that the lifetime prevalence of kleptomania may be 0.6%, and 1.1% to 5.9% for compulsive buying (4,5).

PATHOLOGICAL GAMBLING

Clinical Characteristics

Gambling usually begins in early adulthood, with males tending to start at an earlier age (6,7). As in substance use disorders, females appear to develop pathological

gambling in a shorter time period after beginning to gamble (7–9), a phenomenon originally observed in alcohol dependence and termed “telescoping.” In epidemiological studies, women represent approximately 32% of the pathological gamblers in the United States (10). Pathological gambling, if left untreated, appears to be a chronic, recurring condition, although there exists a distinct need for longitudinal studies to investigate the natural history of the disorder.

Pathological gamblers tend to be fairly specific about their choice of gambling activities. Female pathological gamblers tend to play and have problems with non-strategic games such as slot machines and bingo, whereas males choose sporting events, blackjack, and cards (8). Both female and male gamblers report that advertisements are a common trigger of their urges to gamble, although females are more likely to report that feeling bored or lonely may also trigger their urges to gamble (6,8). Financial and marital problems are common (6). Financial concerns may become so distressing that many pathological gamblers engage in illegal behavior, such as stealing, embezzlement, and writing bad checks (6,9).

Comorbidity and Family History

Several studies have consistently reported that patients with pathological gambling suffer from high rates of lifetime mood disorders (60%–76%), anxiety disorders (16%–40%), and substance use disorders (33%–63%) (11–14). Elevated rates of compulsive buying, compulsive sexual behavior, and intermittent explosive disorder have also been found (12,14,16).

Family studies have consistently demonstrated that first-degree relatives of pathological gamblers suffer from elevated rates of substance use disorders, mood disorders, antisocial personality disorder, pathological gambling, and generalized anxiety disorder (17–20). These studies suggest that there exist shared genetic contributions to pathological gambling and other psychiatric disorders, and this has been confirmed by the results of twin studies suggesting in men, shared genetic contributions to pathological gambling, alcohol dependence, and anti-social behaviors (21,22).

Pharmacological Treatment

Antidepressants

Antidepressants have been used with varying degrees of success in treating pathological gambling, and results from double-blind, placebo-controlled trials are summarized in Table 1. Clomipramine was the first antidepressant to

demonstrate efficacy in reducing excessive gambling. In a double-blind study with one subject, 125 mg/day resulted in significant improvement. The patient sustained improvement for 28 weeks on a dose of 175 mg/day (23).

Citalopram has also shown some benefit as a possible treatment option for pathological gambling in a single open-label study of eight patients followed for three months. Seven of the eight patients were treatment responders as measured by the Clinical Global Improvement scale (24).

Fluoxetine (20 mg/day) plus monthly supportive psychotherapy was compared against supportive psychotherapy alone in a 6-month study. Therapy sessions occurred monthly. Those assigned to combined treatment demonstrated greater improvement in gambling symptoms and greater adherence to treatment than those undergoing supportive therapy alone (25).

Nefazodone, a 5-HT₁/5-HT₂ receptor antagonist, was evaluated in 14 patients in an 8-week open-label trial. Twelve patients were responders as defined by both the Clinical Global Impression scale and the Yale-Brown Obsessive Compulsive Scale Modified for Pathological Gambling (PG-YBOCS) (26).

In an open label study of fluvoxamine, 16 subjects were first enrolled in an 8-week placebo treatment phase and then treated with fluvoxamine for another eight weeks (27). Using an average fluvoxamine dose of 207 mg/day, seven of the 10 patients who completed the study showed much or very much improved ratings on the Clinical Global Improvement scale and had greater than 25% reduction of their gambling behavior scores on the PG-YBOCS.

In a double-blind study, 15 pathological gamblers were treated with fluvoxamine (28). All patients entered a 7-day single-blind placebo phase, followed by a double-blind crossover study design. Of the 10 subjects who completed the study, 6 patients received placebo for the first 8 weeks and then fluvoxamine for the following 8 weeks. Four patients received the treatments in the reverse order. The percent improvement was significantly greater for fluvoxamine (40.6%) than for placebo (16.6%). The average dose of fluvoxamine for responders was 207 mg/day.

Another double-blind study of fluvoxamine with 34 subjects, however, had less impressive results (29). Treating the subjects with 200 mg/day of fluvoxamine for 6 months, and using reduction in money and time spent gambling per week as the outcome measures, the study found that, except in the cases of male or young pathological gamblers, fluvoxamine did not result in statistically significant improvement compared to placebo. This study, however, reported high rates of discontinuation due to subjects being lost-to-follow-up and the high drop-out rate complicates interpretation of the results.

Table I Double-Blind, Placebo-Controlled Pharmacotherapy Trials

Disorder	Treatment	Sample size	Mean daily dose (\pm SD)	Outcome
Pathological Gambling (28)	Fluvoxamine (Luvox)	15 enrolled, 10 completers	195 mg (\pm 50)	Fluvoxamine superior to placebo on CGI and PG-YBOCS
Pathological Gambling (29)	Fluvoxamine (Luvox)	32 enrolled, 13 completers	200 mg	Fluvoxamine not statistically significant from placebo except in young males
Pathological Gambling (30)	Paroxetine (Paxil)	53 enrolled, 41 completers	51.7 mg (\pm 13.)	Paroxetine group significantly improved compared to placebo on CGI
Pathological Gambling (31)	Paroxetine (Paxil)	76 enrolled, 45 completers	50 mg (\pm 8.3)	Paroxetine and placebo groups with comparable improvement on all measures
Pathological Gambling (35)	Naltrexone (ReVia)	89 enrolled, 45 completers	188 mg (\pm 96)	Naltrexone group significantly improved compared to placebo on CGI and G-SAS
Pathological Gambling (40)	Lithium carbonate SR (Lithobid SR)	40 Bipolar-spectrum patients enrolled, 29 completers	1,170 mg (\pm 221)	Lithium group significantly improved compared to placebo on CGI, PG-YBOCS, and CARS-M
Pathological Gambling (42)	Olanzapine (Zyprexa)	23 Video Poker gamblers enrolled, 21 completers	10 mg (\pm 0)	No significant difference found between olanzapine and placebo-treated groups
Compulsive Buying (66)	Fluvoxamine (Luvox)	37 enrolled, 23 completers	215 mg (\pm 76.5)	Fluvoxamine and placebo groups with comparable improvement on YBOCS-CB, CGI, and GAF
Compulsive Buying (67)	Fluvoxamine (Luvox)	23 enrolled, 18 completers	220 mg	Fluvoxamine and placebo groups with comparable improvement on YBOCS-SV and the CGI
Compulsive Buying (68)	Citalopram (Celexa)	24 enrolled, 15 randomized	42.1 mg (15.3)	Citalopram group significantly improved compared to placebo on YBOCS-SV and CGI

PG-YBOCS = Yale-Brown Obsessive Compulsive Scale Modified for Pathological Gambling.

YBOCS-SV = Yale-Brown Obsessive Compulsive Scale—Shopping Version.

YBOCS-CB = Yale-Brown Obsessive Compulsive Scale Modified for Compulsive Buying.

CGI = Clinical Global Impression Scale G-SAS = Gambling Symptom Assessment Scale.

GAF = Global Assessment of Functioning CARS-M = Clinician-Administered Rating Scale for Mania.

Using a mean end-of-study dose of 52 mg/day in a placebo-controlled, double-blind, flexible-dosing, randomized clinical trial, paroxetine was found to decrease thoughts of gambling and gambling behavior after approximately 6 to 8 weeks of treatment (30). A multi-center, randomized, placebo-controlled, flexible-dosing, double-blind study using paroxetine also demonstrated improvement for those taking medication, but the improvement was not statistically more significant than for those assigned to placebo, perhaps in part due to the high placebo response rate (48% to placebo, 59% to active drug) (31).

Several important findings emerge from these antidepressant studies. First, antidepressants, particularly those that influence serotonergic systems like the serotonergic reuptake inhibitors and possibly 5-HT₁/5-HT₂ receptor antagonists, may be effective in reducing the symptoms of pathological gambling. Second, as in the treatment of obsessive-compulsive disorder, doses of antidepressants required to treat pathological gambling symptoms appear to be higher than the average doses generally required to treat depressive disorders. Third, antidepressant treatment effects appear to be independent of underlying depressive symptoms. In studies in which study participants had no or minimal depressive (or anxious)

symptoms (28,30,31), antidepressants were still effective in reducing gambling symptoms. The findings suggest that these drugs may operate by targeting serotonergic systems implicated in impaired impulse regulation (32). Response to antidepressants usually means decreased thoughts about gambling, decreased gambling behavior, and improvement in social and occupational functioning. Patients may initially report feeling less preoccupied with gambling and feeling less anxious about having thoughts of gambling.

Opioid Antagonists

Many pathological gamblers describe urges or cravings to gamble that preoccupy them and interfere with daily functioning. There is a small body of literature suggesting that naltrexone may be effective in reducing urges associated with pathological gambling.

Crockford and el-Guebaly reported a patient suffering from both pathological gambling and alcohol dependence who responded to naltrexone (33). The patient continued to gamble following treatment with fluoxetine at 20 mg/day and was then treated with naltrexone at 50 mg/day. Naltrexone addition resulted in significant reductions in

cravings for both alcohol and gambling within 48 hours of starting naltrexone and the patient continued to report improvement 4 weeks after initial treatment.

In an open label study, 17 subjects were treated for 6 weeks with 50 mg/day to 250 mg/day of naltrexone (34). Naltrexone resulted in a significant decline in the average intensity of urges to gamble, gambling thoughts and gambling behavior during the 6 weeks. The average effective dose of naltrexone was 157 mg/day.

In a double-blind, placebo-controlled, randomized, flexible-dosing, clinical trial of naltrexone, 75% of subjects taking active medication were much or very much improved on both the patient-rated and clinician-rated Clinical Global Impressions scale, compared to only 24% of those on placebo (35). A daily dose of 250 mg/day was targeted, with subjects receiving active drug reaching an average end-of-study dose of 188 mg/day. Approximately 25% of subjects receiving active drug experienced liver function abnormalities following naltrexone dosing, and these enzyme level elevations returned to normal following discontinuation of the drug. The findings are consistent with a black box warning regarding a dose-dependent relationship between naltrexone and liver function abnormalities. The authors report that this association seems most prominent for individuals taking concurrent non-steroidal antiinflammatory drugs (36).

Mood Stabilizers

Successful responses to lithium and carbamazepine were described in two early case reports. Three subjects who were treated with lithium 1800 mg/day reported cessation of gambling (37). A case report also found carbamazepine resulted in improvement in pathological gambling (38).

In a 14-week single-blind trial, 14 (60.9%) of 23 patients taking lithium and 13 (68.4%) of 19 patients taking valproate were responders based on a Clinical Global Impressions scale (39).

A more recent double-blind, placebo-controlled, flexible-dosing randomized clinical trial examined sustained-release lithium carbonate versus placebo in 29 bipolar-spectrum pathological gamblers over 10 weeks (40). Bipolar spectrum disorders were defined as including DSM-IV diagnoses of Bipolar II disorder, Bipolar Disorder NOS, and cyclothymia, and mood swings that occurred at times unrelated to gambling urges/behavior. Drug was dosed targeting therapeutic blood levels of lithium, with subjects on active drug receiving on average a daily dose of 1,170 mg/day at the end of the study. Patients taking medication improved significantly more than those assigned placebo as measured by PG-YBOCS and control over gambling behavior. Participants receiving active as compared with placebo drug also demonstrated

a statistically significant improvement in manic symptoms as assessed by the Clinician-Administered Rating Scale for Mania (CARS-M).

Atypical Antipsychotics

Atypical antipsychotics have been explored as augmenting agents in the treatment of non-psychotic disorders and behaviors, including obsessive-compulsive disorder. One case report demonstrated efficacy of olanzapine 10 mg/day in the treatment of pathological gambling with comorbid schizophrenia (41). Olanzapine has been examined in the treatment of video poker pathological gamblers but failed to demonstrate any statistically significant benefit when compared to placebo (42).

KLEPTOMANIA

Although mentioned in the medical literature for the last two hundred years, kleptomania was only designated a psychiatric disorder in 1980 in DSM-III. The DSM-IV-TR diagnostic criteria essentially reflect an understanding of "kleptomania" (stealing madness) dating back to 1838, the time in which the term was coined: 1) recurrent failure to resist an impulse to steal unneeded objects; 2) an increasing sense of tension before committing the theft; 3) an experience of pleasure, gratification or release at the time of committing the theft; and 4) the stealing is not performed out of anger, vengeance, or due to psychosis (1).

Clinical Characteristics

Kleptomania begins most often in late adolescence or early adulthood (4,43–45). The course of the illness is generally chronic with waxing and waning of symptoms. Although men suffer from kleptomania, the majority of patients with kleptomania are women. Women appear twice as likely to suffer from kleptomania as men (43–46).

Most patients with kleptomania try unsuccessfully to stop stealing. In one study all patients reported increased urges to steal when trying to stop their behavior (43). The inability to stop a behavior that a person does not want to perform may lead to feelings of shame and guilt, which was reported in 77.3% of the subjects. Of married subjects, less than half (41.7%) had told their spouses about their behavior due to the shame and guilt (43).

Although patients with kleptomania may steal from several different places, the majority steal from stores. Items stolen generally vary between patients. In one study, 68.2% of patients reported that the value of stolen items had increased over the duration of the disorder (43), suggesting

an aspect of tolerance. Patients frequently keep, hoard, discard, or return stolen items (44).

Many patients with kleptomania (64% to 87%) have been apprehended at some time due to their stealing behavior (44,45), and 15% to 23% report having been jailed (43). Although the majority of the patients who were apprehended reported that their urges to steal were diminished after the apprehension, their symptom remission generally lasted only for a few days or weeks (44).

Comorbidity and Family History

High rates of other psychiatric disorders have been found in patients with kleptomania. Rates of lifetime comorbid affective disorders range from 59% to 75% (43,47) to 100% (44). The rate of comorbid bipolar disorder, however, is unclear, with results of lifetime comorbid bipolar disorder ranging from 9.1% (43) to 27% (47) to 60% (44) having been reported. Studies have also found high lifetime rates of comorbid anxiety disorders (60% to 80%) (44,45,48), impulse control disorders (20% to 46%) (45,49), and eating disorders (60%) (44). Large, nationally representative epidemiological studies investigating the relationship between kleptomania and other psychiatric disorders have yet to be performed.

Patients with kleptomania are more likely to have a first-degree relative with a psychiatric disorder compared to normal controls (49). In particular, high rates of mood and substance use disorders have been reported in the first-degree relatives of individuals with kleptomania. In fact, 20% to 35% of first-degree relatives appear to suffer from mood disorders, while 15% to 20% suffer from substance use disorders (44,49).

Pharmacological Treatment

Only case reports, two small case series, and one open-label study of pharmacotherapy have been described for kleptomania. One study reported treatment response in 10 of 20 patients with the following single agents: fluoxetine, nortriptyline, trazodone, clonazepam, valproate, and lithium (44). Other agents used successfully as monotherapy for kleptomania include fluvoxamine (50), paroxetine (51), topiramate (52), and naltrexone (53). Combinations of medications have also been effective in case reports: lithium plus fluoxetine (54), fluvoxamine plus buspirone (55), fluoxetine plus alprazolam (44), fluvoxamine plus valproate (56), and fluoxetine plus imipramine (44).

The only formal trial of medication for kleptomania published to date involved 10 subjects in a twelve-week, open-label study of naltrexone. Using a mean dose of 150

mg/day, medication resulted in a significant decline in the intensity of urges to steal, stealing thoughts and stealing behavior (57).

Not all medication options, however, have shown effectiveness. In fact, seven cases of fluoxetine, three cases of imipramine, two cases of lithium as monotherapy and two cases of lithium augmentation, four cases of tranylcypromine, and carbamazepine in combination with clomipramine all failed to reduce kleptomania symptoms (44). Together, these findings highlight the need for placebo-controlled, large-scale investigations into the efficacy and tolerability of pharmacotherapies for kleptomania.

COMPULSIVE BUYING

Although not specifically recognized by DSM, the following diagnostic criteria have been proposed for compulsive buying: 1) maladaptive preoccupation with or engagement in buying (evidenced by frequent preoccupation with or irresistible impulses to buy; or frequent buying of items that are not needed or not affordable; or shopping for longer periods of time than intended); 2) preoccupations or the buying lead to significant distress or impairment; and 3) the buying does not occur exclusively during hypomanic or manic episodes (5).

Clinical Characteristics

As with other ICDs, the onset of compulsive buying appears to occur during late adolescence or early adulthood, although the full disorder may take several years to develop (58,59). In clinical samples, the disorder shows a female preponderance ranging from 80% to 92% (5,58–60).

Patients report repetitive urges to shop that are most often unprovoked but may be triggered by being in stores. These urges may worsen during times of stress, emotional difficulties, or boredom. Urges are generally intrusive, and most patients attempt to resist the urges, although usually unsuccessfully. The behavior regularly results in large amounts of financial debt, marital or family disruption, and even legal consequences (59). Although the behavior is pleasurable and momentarily relieves the urges to shop, guilt, shame, and embarrassment typically follow the buying episodes.

Patients report purchasing any number of items as it is often the positive interaction with salespeople that drives the excessive shopping behavior. The choices of items vary considerably and might include clothing, jewelry, books and auto parts. Most items are not used or even removed from the packaging. Many items are given away or returned. Hoarding of particular items is common (59).

Comorbidity and Family History

Patients with compulsive buying commonly experience comorbid disorders. Rates of comorbid mood disorders range from 28% to 95% (5,59–61). The mood disorder often precedes the compulsive buying by at least one year in the majority of patients (59). Lifetime histories of anxiety (41% to 80%), substance use (30% to 46%), eating (17% to 35%), and impulse control (21% to 40%) disorders are fairly common (5,59–61).

Patients with compulsive buying tend to report first-degree relatives with mood and substance use disorders (20,61), and it is not uncommon for patients to have a first-degree relative who suffers from compulsive buying (20,61).

Pharmacological Treatment

The effectiveness of pharmacotherapy in treating compulsive buying is unclear, and the results from placebo-controlled, double-blind trials are summarized in Table 1. In a case series, the tricyclic antidepressant nortriptyline, the selective serotonin reuptake inhibitor (SSRI) fluoxetine, and antidepressants in combination with mood stabilizers (bupropion plus lithium, fluoxetine plus valproate, and fluoxetine plus lithium) brought about partial or full remission of compulsive buying behaviors in 9 of 20 psychiatric patients (5). In a separate case report, clomipramine was effective in two patients with compulsive buying (62). In addition to trials of antidepressants, one case series of three patients with strong urges to shop found that naltrexone (mean dose 150 mg/day) was effective in reducing both the urges and the behavior itself (63).

The initial open-label study involved treating ten patients with fluvoxamine over a nine-week period. Nine of ten subjects reported positive results taking a mean dose of 205 mg/day of fluvoxamine (64). More recently, Koran and colleagues tested citalopram in an open-label design. In this study, a mean dose of 35 mg/day demonstrated effectiveness in 17 of 24 patients (71%) treated for 12 weeks (65).

In the first of two double-blind studies to use fluvoxamine, 37 subjects were treated for 13 weeks. Only 9 of 20 patients assigned to medication were responders (mean dose of 215 mg/day), and this did not differ significantly from the placebo group (8 of 17 were responders) (66).

In the second double-blind study using fluvoxamine, Black and colleagues treated 23 patients for 9 weeks following a one-week placebo lead-in phase. Using a mean dose of 200 mg/day, patients on medication failed to respond as well as patients assigned to placebo, although the difference between the groups was not statistically different (67).

A double-blind study also using citalopram has confirmed the earlier open-label findings. In this study, seven weeks of open-label treatment was followed by responders

being randomized to medication or placebo for another nine weeks. Patients taking citalopram demonstrated statistically significant decreases on the Yale Brown Obsessive Compulsive Scale—Shopping Version and the Clinical Global Impression scale (68). In summary, findings from pharmacotherapy trials for compulsive shopping have yielded mixed results, with SSRIs appearing beneficial in some placebo-controlled studies but not others.

CONCLUSIONS

Impulse control disorders have historically received relatively little attention from clinicians and researchers. Despite ICDs having prevalence rates similar to or greater than those for schizophrenia and bipolar disorder, much less research has been performed investigating treatment strategies for these disorders. As a consequence, our understanding of efficacious and well-tolerated pharmacotherapies for ICDs lags significantly behind those for other major neuropsychiatric disorders. Emerging data from controlled clinical trials, however, suggest that ICDs frequently respond to pharmacological intervention.

Approaches reviewed in this article represent significant advances from only several years ago. It is hoped that progress in the treatment of ICDs will continue to be made at the rate recently witnessed. More definitive treatment recommendations await completion of additional, large-scale controlled treatment studies for these disorders and comparative investigations of pharmacological agents. Advances in these areas hold the potential for significantly improving the lives of individuals with ICDs and those directly or indirectly affected by their conditions.

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