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ADDICTIONS (J GRANT, SECTION EDITOR)

Pharmacotherapy for Behavioral Addictions

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Abstract Certain behavioral syndromes (behavioral addictions), such as gambling disorder, stealing, shopping, and compulsive sexual behavior, appear to share clinical and neurobiological parallels with substance addictions. Pharmacological agents, often those used in substance addictions, have shown some benefit for behavioral addictions. This article reviews the double-blind, placebo-controlled pharmacological studies in the field of behavioral addictions. Future work is needed to develop treatment algorithms for people struggling with these disorders.

Keywords Treatment · Pharmacotherapy · Addiction · Impulsivity · Cognition

Introduction

Certain psychiatric disorders characterized by repetitive habits share considerable clinical parallels with substance addictions and have thus been argued to represent "behavioral addictions" [1]. There is, however, considerable debate as to whether the concept of behavioral addiction is valid, and if so, which disorders should be considered within this remit [2]. For the purposes of this article, we focus on gambling disorder, kleptomania (compulsive stealing), compulsive buying, and compulsive sexual behaviors. We refer to these as behavioral addictions for convenience, but with the caveat noted above. Far from being rare disorders, these conditions appear to be relatively common, particularly among young adults and individuals with psychiatric health issues [3, 4]. Collectively, these conditions cause considerable suffering for affected individuals but can also negatively impact families and careers [5]. In response to the need, the last 10 years have seen a considerable research focused on pharmacological treatments for these behaviors.

Chemicals with addictive properties, such as cocaine or amphetamine, affect the brain's reward pathways, particularly the ventral striatum, and thereby implicate the dopaminergic and opioid systems [6, 7]. Core features of substance addiction include impaired control (craving and unsuccessful attempts to reduce intake), impairment (narrowing of interests), risky use (persisting intake despite awareness of longer-term damaging effects), and a physiological response to use or to reducing use (tolerance, withdrawal). It can be argued that behavioral addictions also fit this repertoire. Individuals with behavioral addictions exhibit impaired control, functional impairment, and persisting engagement in the behavior despite negative consequences. In addition, evidence also suggests that people with behavioral addictions experience tolerance (e.g., need to gambling with greater amounts of money over time, having riskier sex to produce the same degree of euphoria) and withdrawal (e.g., agitation, insomnia, irritability) [5].

In substance addictions, drugs directly or indirectly modulate dopamine levels in the neural "reward system." In addition, drug intoxication is associated with strong reinforcement effects, which induce repetitive drug-seeking and heighten salience attribution to the drugs of abuse [8, 9]. Dopamine

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also appears to play a key role in the various aspects of reward underlying the behavioral addictions. Pro-dopaminergic medication has been linked, in some individuals, with new onset (or worsening of) gambling, stealing, excessive spending, and sexual behavior [10, 11]. The literature has particularly focused on new onset behavioral addictions in relation to the use of dopamine agonists in Parkinson's disease [12•], possibly via D3 receptor mechanisms [13].

In the case of gambling disorder, multiple neurotransmitters have been implicated in its etiology. One particular line of inquiry has found positive correlations between dopamine binding and gambling severity and impulsivity [14–17]. In addition, in a PET study measuring dopamine activity during the Iowa Gambling Task, it was found that dopamine release in pathological gamblers was related to excitement and worse behavioral performance [14, 18]. Instead of the diminished dopamine release in response to amphetamine challenges in substance addictions, one study using the [11C] PHNO DA (D3) receptor ligand showed that an amphetamine challenge increased dopamine release in the dorsal striatum in disordered gamblers [19]. While chronic substance misuse is associated with D2 receptor downregulation, this appears not to be the case with chronic gambling [20]. Thus, the dopamine system in disordered gambling could be differently affected than in the case of substance use disorders.

The neural correlates of kleptomania have barely been studied, despite the condition being recognized in the medical and legal literature for centuries. Kleptomania has been associated with reduced platelet serotonin transporter levels compared to controls [21]. In a small neuroimaging study, kleptomania was associated with significantly reduced fractal anisotropy (suggestive of disorganized and/or damaged white matter tracts) in frontal brain regions compared to controls [22].

There are few neuroimaging or neuropsychological investigations of compulsive sexual behavior to date. In a pilot study, people with compulsive sexual behavior showed lower white matter diffusivity in superior frontal brain regions compared to controls, coupled with impulsive behavioral performance on a go/no-go paradigm [23]. Using fMRI, people with compulsive sexual behavior, compared to controls, showed enhanced neural responses to erotic videos in the dorsal anterior cingulate cortex, nucleus accumbens, and amygdala [24••]. Processing bias for sex-related visual stimuli has been found in a separate study using a dot-probe attentional task, in people with compulsive sexual behavior compared to controls [25].

Although no neuroimaging studies focusing on compulsive buying have been published, neurocognitive data suggests that individuals with compulsive buying have impairments in response inhibition (stop-signal task), risk adjustment during decision-making, and spatial working memory [26]. These data suggest that individuals with compulsive buying experience problems in several distinct cognitive domains, supporting a likely neurobiological overlap between compulsive buying and other behavioral and substance addictions.

Pharmacological Treatment

Understanding some of these behaviors as behavioral addictions has resulted in new treatment directions. There are no medications currently approved in any jurisdiction for the treatment of behavioral addictions, but some medications that have shown promise in treating substance addictions have also shown promise in treating behavioral addictions. Here, we focus on data from double-blind, placebo-controlled studies. Open-label studies and case series have produced some intriguing avenues for future research but are still too premature to be used as a basis for treatment recommendations.

Gambling Disorder

Gambling disorder, characterized by persistent and recurrent maladaptive patterns of gambling behavior, is associated with impaired functioning, reduced quality of life, and high rates of bankruptcy, divorce, and incarceration [27].

Different classes of medications have been investigated as treatments for gambling disorder. These have included antidepressants (particularly serotonin reuptake inhibitors [SRIs]), lithium, glutamate agents, neuroleptics, and opioid antagonists. Results of these studies have demonstrated mixed findings in regard to efficacy and tolerability.

Opioid receptor antagonists have to date resulted in the most effective treatment outcomes for gambling disorder. In the first study, naltrexone demonstrated superiority to placebo in 45 subjects with gambling disorder [28]. Naltrexone (mean dose of 188 mg/day) was effective in reducing gambling-related behaviors and thoughts/urges. Naltrexone was particularly effective in gamblers with more severe gambling urges. A second naltrexone study in 77 subjects randomized to either naltrexone or placebo over an 18-week period confirmed the findings of the initial study [29]. Furthermore, two multicenter studies demonstrated the efficacy of the opioid antagonist, nalmefene, in the treatment of gambling disorder. In the first multicenter study, 207 subjects were assigned to receive 16 weeks of either nalmefene at varying doses (25, 50, or 100 mg/day) or placebo. At the end of the study, 59 % of those assigned to nalmefene showed significant reductions in gambling urges, thoughts, and behavior compared to only 34 % on placebo [30]. The second multicenter nalmefene study with 233 participants used compared different doses (20 or 40 mg) or placebo [31]. An intent-to-treat analysis failed to show statistically significant differences from placebo on primary and secondary outcomes, but a post hoc analysis of participants who received a full titration of the medication for at least

1 week demonstrated significantly greater reductions on the primary outcome measure for the 40-mg dose.

Another promising area of research in the pharmacological treatment of gambling disorder has been glutamate agents. *N*-acetylcysteine (NAC), a glutamate-modulating agent, was administered to 27 gambling disorder subjects over an 8-week period with responders randomized to receive an additional 6-week double-blind trial of NAC or placebo. Of those in the open-label phase, 59 % experienced significant reductions in symptoms and were classified as responders. At the end of the double-blind phase, 83 % of those assigned to receive NAC were still classified as responders compared to only 28.6 % of those assigned to placebo [32]. A follow-up 12-week study combining NAC with imaginal desensitization in 28 gambling disorder subjects who were also nicotine dependent found that NAC provided significant benefit compared to placebo on nicotine dependence symptoms during treatment and on gambling symptoms 3 months after formal treatment ended [33•]. Other glutamate agents, however, have not been successful. A 14-week trial of topiramate in 42 subjects failed to show significant treatment effects for topiramate on the primary or secondary outcome measures [34].

The most studied class of medications for gambling disorder has been the antidepressants, particularly selective serotonin reuptake inhibitors (SSRIs), but the results have been mixed. In one study using sertraline, 60 subjects were treated for 6 months (mean dose = 95 mg/day) [35]. At the end of the study, 23 sertraline-treated subjects (74 %) and 21 placebo-treated subjects (72 %) were rated as responders based on the primary outcome measurement. Two studies have examined fluvoxamine [36] with one demonstrating some improvement while the other was no more successful than placebo [37]. Two studies of paroxetine were similarly conflicting [38, 39]. One non-serotonergic antidepressant, bupropion, has also been examined, and it failed to separate from placebo [40].

Only one study has examined lithium for gambling disorder. In a study of 40 subjects with gambling and bipolar spectrum disorders (bipolar type II, bipolar not otherwise specified, or cyclothymia), sustained-release lithium carbonate (mean lithium level 0.87 mEq/L) was shown to be superior to placebo in reducing gambling symptoms during 10 weeks of treatment [41].

Finally, two studies have examined the use of olanzapine in the treatment of gambling disorder. Both studies found that olanzapine was no more effective than placebo in reducing gambling symptoms [42, 43].

Several conclusions can be drawn from the pharmacological treatment studies for gambling disorder. Although several different classes of medication have shown efficacy in treating gambling disorder, only the opiate antagonists have had successful replication in randomized, placebo-controlled studies. Having said that, the creation of a treatment algorithm is difficult given that no comparison studies of medications have been performed in a randomized, placebo-controlled design, and no study has examined whether certain individuals with gambling disorder would benefit differentially from specific pharmacotherapies.

Compulsive Buying

Although not specifically recognized in the *DSM-5*, the following diagnostic criteria have been proposed for compulsive buying: (a) 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); (b) preoccupations or the buying leads to significant distress or impairment; and (c) the buying does not occur exclusively during hypomanic or manic episodes [44].

There have been only three double-blind, placebo-controlled pharmacological studies for compulsive buying, and all three have examined SSRI antidepressants. Two of the studies examined fluvoxamine studies, with neither study demonstrating benefit of the medication when compared to placebo [45, 46]. The third study was a 7-week open-label study of citalopram that randomized the responders to 9 weeks of double-blind medication or placebo [47]. Subjects taking citalopram demonstrated statistically significant decreases in the frequency of shopping and the intensity of thoughts and urges concerning shopping.

There is scant evidence concerning effective pharmacological treatments for compulsive buying. Based on available data, citalopram may offer some benefit for this disorder.

Kleptomania

Kleptomania is characterized by repetitive, poorly controlled stealing of items not needed for personal use. There have been only two randomized, placebo-controlled studies of medication for the treatment of kleptomania. An 8-week, double-blind, placebo-controlled trial was conducted to evaluate the safety and efficacy of oral naltrexone in 25 subjects with kleptomania. Subjects assigned to naltrexone had significantly greater reductions in stealing urges and behavior compared with subjects on placebo. The mean effective dose of naltrexone was 116.7 (+/−44.4) mg/day [48].

In the other study, subjects were treated with the SSRI, escitalopram, for 7 weeks in an open-label design. After that period, those who responded were randomly assigned to 16 weeks of placebo or continuation of escitalopram. Nineteen subjects (79 %) were week 7 responders, and 15 were randomly assigned. Three (43 %) of seven escitalopram subjects relapsed compared with four (50 %) of eight placebo subjects [49]. The high response rate during open-label escitalopram treatment was not maintained during the

double-blind phase, and this suggests that the SSRI medication was no more beneficial than placebo.

Although the data are extremely limited for kleptomania, the single positive study for this disorder suggests that naltrexone may be a beneficial treatment for kleptomania.

Compulsive Sexual Behavior

Compulsive sexual behavior, also known as sex addiction and hypersexuality, is characterized by repetitive and intense preoccupations with sexual fantasies, urges, and behaviors that are distressing to the individual and/or result in psychosocial impairment [50].

There is limited research concerning pharmacotherapy for compulsive sexual behavior. In the only published double-blind, placebo-controlled pharmacological study, 28 gay and bisexual men were treated with citalopram over a 12-week period. The group assigned to medication reported significantly greater reductions in their desire for sex, frequency of masturbation, and hours of pornography use per week [51]. In our experience, close monitoring is needed in the treatment of compulsive sexual behavior: in some cases, individuals may undertake more risky sexual practices in order to compensate for dampening effects of medication on sex drive.

Given that there is only one published controlled study of medication, one cannot make detailed recommendations regarding pharmacological treatment options. In the case of this behavioral addiction, the SSRI citalopram showed some benefit.

Pharmacological Treatment Recommendations for Behavioral Addictions

In the area of behavioral addictions, the systematic study of treatment efficacy and tolerability is in its infancy. Thus, it is not possible to make treatment recommendations with a substantial degree of confidence. No drugs are currently approved by the Food and Drug Administration (FDA) for the treatment of any of these behavioral addictions. Nonetheless, the opiate antagonists appear to offer promise for the effective treatment of gambling disorder and kleptomania. The role of SSRIs appears more complicated across these disorders with some positive response noted for citalopram for sexual behavior and compulsive buying. Future studies may want to examine predictors of response to SSRIs across the behavioral addictions and whether certain SSRIs are more likely to result in beneficial outcomes than others.

Even with some encouraging outcome studies for behavioral addictions, treatment studies for all of these disorders are substantially limited. In addition, most published studies have employed relatively small sample sizes, are of limited duration, and involve possibly non-representative clinical groups (e.g., those without co-occurring psychiatric disorders or those recruited from highly specialized clinical settings). In addition, the definition of "response" remains debated. Heterogeneity of treatment samples may complicate the identification of effective treatments. At present, issues such as which medication to use and for whom or the duration of pharmacotherapy cannot be sufficiently addressed with the available data.

Conclusion

The evidence to date suggests that behavioral addictions may respond, at least to some degree, to pharmacological interventions. This evidence is strongest for gambling disorder and the use of opioid antagonists. In all cases, however, more research is needed to better conceptualize targets (e.g., subtypes of certain behavioral addictions, cognitive vulnerabilities to various behavioral addictions) for medication treatment.

Compliance with Ethical Standards

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Dr. Chamberlain reports consultation for Cambridge Cognition, outside the submitted work.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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