

A review of gambling disorder and substance use disorders

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Abstract: In the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*, gambling disorder was recategorized from the “Impulse Control Disorder” section to the newly expanded “Substance-related and Addictive Disorders” section. With this move, gambling disorder has become the first recognized nonsubstance behavioral addiction, implying many shared features between gambling disorder and substance use disorders. This review examines these similarities, as well as differences, between gambling and substance-related disorders. Diagnostic criteria, comorbidity, genetic and physiological underpinnings, and treatment approaches are discussed.

Keywords: pathological gambling, problem gambling, behavioral addiction, transdiagnostic factors, addiction syndrome

Introduction

Gambling disorder (GD) is a persistent maladaptive pattern of gambling resulting in clinically significant impairment or distress.¹ In order to meet the criteria, individuals must exhibit four or more of the nine symptoms within a 12-month period. GD can present as either episodic or persistent and is rated as mild, moderate, or severe according to the number of symptoms endorsed. In the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM-5)*,¹ pathological gambling was renamed GD and recategorized from an impulse control disorder to an addiction-related disorder, highlighting longstanding conceptualizations of GD as an addiction. The links between GD and alcohol and drug use disorders (AUD/DUD) are numerous and include analogous diagnostic criteria, high comorbidity rates, shared genetic underpinnings, similar neurobiological effects, and common treatment approaches. For the purposes of this review, AUD refers to either alcohol abuse or dependence and DUD refers to any illicit or nonmedical (nontobacco, nonalcohol) drug abuse or dependence disorder unless otherwise noted. In light of GD’s reclassification as the first non-substance behavioral addiction, this paper will provide an overview of the potential links between GD and AUD/DUD from etiology to treatment approaches with emphasis on areas impacted by the *DSM-5* classification.

Diagnostic criteria

Significant construct overlap is present across *DSM-5* GD and AUD/DUD, given that the original *DSM-III* gambling criteria were modeled largely on the substance dependence criteria of the time.² However, important differences do exist across the two diagnostic sets, and, consequently, the *DSM-5* substance use disorder (SUD) Work

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Group recommended adoption of the *DSM-IV* GD criteria with modifications rather than adapting the SUD criteria for GD.³ In Table 1, we list the criteria for GD and AUD, highlighting overlapping or similar content items. Items with the strongest content overlap include tolerance, withdrawal, loss of control, and negative consequences. With respect to the latter construct, GD has one item related to negative impact on social, educational, or work domains; for AUD, four items describe negative impacts to more varied life domains (eg, psychological health, physical health). The AUD/DUD criteria set, including these negative consequences items, is likely to be reviewed for redundancy and possible streamlining in future DSM editions,³ thereby facilitating greater diagnostic consistency between addictive disorders. Conversely, GD's negative consequences item might be expanded to include other relevant domains such as psychological health, which is often negatively impacted in those with the disorder.^{4,5} Specifically, both rates of comorbidity⁶ and risk of suicide ideation and attempts^{7,8} have been shown to be elevated in individuals with GD.

A second relevant shared diagnostic feature is fixation upon the addictive behavior. In GD, this construct is referred

to as preoccupation with gambling, and it concerns reliving past gambling experiences, planning future gambling experiences, and strategizing ways to fund gambling. For AUD, a comparable item pertaining to spending a great deal of time obtaining, using, or recovering from alcohol use corresponds with some of the planning features evident in the GD item. However, the AUD item does not fully address the cognitive component of preoccupation represented in GD. The cravinging item from the alcohol criteria, new to *DSM-5*, may capture a portion of this cognitive construct. A cravinging item was not added to the GD criteria, which do not explicitly address cravingings. Although evidence suggests that cravingings are common among individuals with GD^{9,10} and that they are related to gambling behavior,^{11,12} the question of whether cravingings are central to the diagnosis of GD, as in SUD, remains unanswered. The remaining items, four from GD and one from AUD, do not have a corresponding criterion in each disorder set and highlight unique aspects of each disorder (eg, chasing losses). Questions remain about whether shaping the GD criteria to more closely model the SUD criteria set is advantageous for diagnosis of GD and for diagnostic consistency within the section.

Table 1 Comparison of *DSM-5* gambling disorder and alcohol use disorder criteria

Construct	Gambling disorder	Alcohol use disorder
Tolerance	Gambles with more money in order to achieve the desired excitement	Using increasing amounts of alcohol to achieve intoxication or desired effect; or diminished effect when using the same amount of alcohol
Loss of control	Unsuccessful attempts to control, limit, or stop gambling	Desire or unsuccessful attempts to cut down or control use Using larger amounts or over a longer period than intended
Withdrawal	Restlessness or irritability when trying to control gambling	Alcohol withdrawal symptoms or use of alcohol (or related substances) to relieve or avoid withdrawal symptoms
Negative consequences	Risked or lost a significant relationship, educational, or work-related opportunity because of gambling	Continued use despite social or interpersonal problems related to alcohol use Alcohol use leading to neglect of major role obligations at work, school, or home Gave up important social, occupational, or recreational activities because of alcohol use Continued use despite physical or psychological problems that are likely to have been caused or worsened by alcohol
Fixation	Preoccupation with gambling-related thoughts such as reliving past gambling experiences, planning future experiences, or strategizing ways to fund gambling behavior	Excessive time spent obtaining, using, or recovering from alcohol Cravings, strong desire, or urges to use alcohol
Negative affect	Frequently gambles in response to negative affect	
Chasing losses	Often follows gambling losses by returning another day to recoup money	
Lying	Lies about gambling or gambling-related consequences	
Bailouts	Depends on others for money to alleviate desperate financial situations caused by gambling	
Hazardous situations		Recurrent use in physically hazardous situations

Abbreviation: *DSM-5*, *Diagnostic and Statistical Manual for Mental Disorders*, fifth edition.

Prevalence

AUD exhibits high prevalence rates relative to many other psychiatric conditions. For example, lifetime and past-year prevalence rates of AUD were 30.3% and 8.5%, respectively, in the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC).¹³ These rates are substantially higher than prevalence rates of any nontobacco DUD (lifetime: 10.3%, past-year: 2.0%) and major depressive disorder (lifetime: 13.2%, past-year: 5.3%).¹³ Results from nationally representative samples estimate a substantially lower prevalence for GD with ~1%–2% meeting lifetime criteria and half of that meeting past-year criteria.^{14–17} Young age, male sex, low socioeconomic status, and unpaired marital status (ie, never married, divorced, separated, widowed) are common demographics shared by individuals with GD and AUD/DUD.^{13,15–17}

Diagnostic threshold

In the *DSM-5*, the diagnostic threshold for GD was lowered from five of ten criteria to the current threshold of four of nine criteria.¹ The changes done by the *DSM-5* SUD Work Group to the GD criteria were designed to minimize impact on prevalence rates while improving diagnostic accuracy.¹⁸ Nonetheless, modest increases in GD prevalence rates are likely as *DSM-5* criteria are adopted. For example, in a sample of randomly selected US household residents (N=2,417), prevalence rates of GD increased from 0.1% to 0.2% using the *DSM-5* criteria.¹⁹ Samples from clinical settings that serve high-risk gamblers will also be affected. Prevalence of GD increased from 81.2% under *DSM-IV* to 90.3% using *DSM-5* criteria among West Virginian gamblers (N=2,750) calling a state gambling help line.⁸

Despite a lower threshold, stark differences remain between SUD and GD in terms of diagnostic thresholds and recognition of milder forms of the disorder.^{8,20} For the *DSM-5* SUD criteria, which combine *DSM-IV* substance abuse and dependence items in a single diagnostic set, only two or more of eleven symptoms are required for diagnosis. Severity is indicated with mild (2–3 symptoms), moderate (4–5 symptoms), and severe (six or more symptoms) specifiers, which is incongruent with GD severity specifiers: mild (4–5 symptoms), moderate (6–7 symptoms), and severe (8–9 symptoms).

If GD criteria were to be more directly modeled after SUD criteria with its lower threshold, the prevalence rate of GD would rise significantly, as an additional 2% of individuals endorse subclinical lifetime gambling problems.^{14,15} Consideration of such a change, despite the potential impact on prevalence rates, may be warranted if individuals with subthreshold GD symptoms experience clinically significant

levels of impairment or harm on par with mild AUD/SUD and if they benefit from identification and treatment. Several studies document substantial negative impacts associated with subclinical gambling, including increased risk of comorbidity,^{6,21} financial problems and gambling-related debt,⁸ and suicide ideation and attempts.⁷ Given these significant consequences, as well as the high rates of comorbidity between AUD/DUD and GD (discussed in the following section), consistency among these diagnostic sets may assist clinicians by applying a single set of criteria and severity ratings across disorders.

Comorbidity

GD and psychiatric disorders

Comorbidity with other psychiatric disorders, including other addictions, is common in both AUD/DUD and GD. As many as 96% of individuals with lifetime GD also meet criteria for at least one other lifetime psychiatric disorder.^{6,15} Lifetime rates of many psychiatric disorders are elevated among those with GD,¹⁶ with mood (49%–56%)^{15,16} and anxiety (41%–60%)^{15,16} disorders and AUD (73%)¹⁶ and DUD (38%)¹⁶ being particularly prevalent.¹⁵ Personality disorders are also more common among those with GD¹⁶ and the prevalence of multiple comorbid disorders is increased as well. Specifically, in a cross-sectional study,¹⁵ individuals with GD were 30 times more likely to have multiple (three or more) other lifetime psychiatric disorders compared to those without GD. Moreover, this retrospective study suggests that the majority of this comorbidity (74%) precedes and may be a risk factor for the development of GD rather than GD serving as a risk factor for the development of other psychiatric disorders. However, longitudinal prospective studies,^{22,23} which are advantageous for establishing temporal sequence of disorder onset, suggest that past-year GD is associated with the subsequent development of new psychiatric conditions including mood, anxiety, and AUD. The risk of developing new disorders appears to be associated with the severity of gambling behavior,²³ with diagnosed gamblers at greatest risk for onset of a new comorbid disorder compared to problem or recreational gamblers. Overall, the literature supports a bidirectional relationship with respect to comorbidity such that psychiatric disorders can serve as risk factors in the development of, can serve as maintenance factors in GD, and can arise as consequences of GD.^{15,22,24}

GD and AUD/DUD

GD's association with other addictive disorders is well established. Population-based meta-analytic estimates

suggest high rates of lifetime AUD and DUD comorbidity among lifetime problem and pathological gamblers, with 28% of gamblers reporting an AUD and 17% reporting an illicit DUD.²⁵ These prevalence rates are best understood by comparing the difference in rates of SUD/DUD diagnoses among those with and without GD. For example, in the Welte et al¹⁷ study, 25% of those with GD met criteria for current alcohol dependence, whereas only 1.4% of those without GD were alcohol dependent. Echoing the discussion of multi-comorbidity noted earlier, the presence of dual addictive disorders, such as concurrent AUD and GD, is associated with increased risk of additional psychiatric disorders compared to the presence of GD without AUD.²⁶

AUD and DUD are also more common among treatment-seeking gamblers than in the general population, with as many as 41% meeting criteria for lifetime AUD and 21% meeting criteria for nonalcohol SUDs including nicotine dependence.²⁷ Comorbid DUD impacts gambling outcomes such that those with no lifetime history of DUD are 2.6 times more likely to achieve a 3-month period of gambling abstinence compared to those with lifetime DUD.²⁸ Another study²⁹ suggests that even among those with lifetime AUD/DUD, a majority (58%) of those seeking gambling treatment are actively using alcohol or illicit substances in the year prior to admission for gambling treatment. Fortunately, at-risk alcohol use (more than 14 standard drinks/week or 4/day for males; more than 7 drinks/week or 3 drinks/day for females) does appear to decrease during gambling treatment,³⁰ and these naturally occurring reductions in alcohol use might be enhanced with the incorporation of brief alcohol interventions into gambling treatments. Such treatments may reduce the possibility of progression to disordered levels of alcohol use, the presence of which is associated with gambling relapse.²⁸ These concurrent changes in alcohol use and gambling suggest that these behaviors may influence each other over time.

Given the high prevalence rates and the impact of comorbid DUD and AUD on gambling outcomes, inclusion of AUD and DUD screening procedures in clinical practice is recommended for patients with GD. The converse, screening for problematic gambling among substance abuse treatment seekers, is also warranted. Approximately, 15% of AUD/DUD treatment seekers meet lifetime criteria for GD and 11% meet current criteria for GD.³¹ Among opioid substitution patients, rates of GD may be even higher,³¹ and problem gambling is associated with poorer response to substance abuse treatment among these patients.³² Integration of gambling screening and referral processes into substance abuse treatment may

improve not only the gambling problem, but also AUD/DUD treatment outcomes. In addition, many individuals with AUD/DUD are able to achieve sobriety from alcohol and drugs, but are unable to control their gambling,²⁹ suggesting gambling-specific or integrated treatments may be necessary for treatment success of both disorders in substance abuse treatment settings.

GD diathesis

An individual's genetic makeup can confer significant risk in the development of both SUD and GD. The proportion of variability due to genetic factors ranges from 0.39 for hallucinogens to 0.72 for cocaine.³³ GD's heritability is within this range at 0.50–0.60 and is similar to heritability rates for alcohol and opiates.³⁴ Recent theoretical work³⁵ on the progression from initiation to addiction suggests that genetic contributions play a larger role in the later stages of the addiction process (eg, loss of control), whereas environmental experiences appear to mediate initial exposure and experimentation.^{36,37} These environmental contributions to the variability in risk for developing GD are reported to account for 38%–65% of the variance in problematic gambling behavior³⁸ and represent a significant factor in understanding the development of this disorder. Specific environmental factors identified as risk factors for GD include childhood maltreatment,³⁹ parental gambling behavior and monitoring,^{40–42} cultural acceptance of gambling,⁴⁰ and situational factors such as convenience of gambling establishments and prize characteristics.⁴³

Much of the heritable risk for drug addiction is nonspecific and shared across substances. This shared risk is likely due to broad constructs such as impulsivity and negative affect, which have genetic underpinnings and may serve as risk factors for substance use.⁴⁴ Not only do the risks associated with impulsivity and negative affect cut across substances of abuse, but a burgeoning literature also suggests that these constructs are risk factors for the development of several other externalizing disorders, including GD.^{34,45} For example, a prospective developmental study²⁴ suggested that 1) underlying adolescent impulsivity influences the later development of both problematic gambling and depressogenic features, and that 2) these two sets of symptoms then bidirectionally perpetuate each other across late adolescence and early adulthood. Moreover, with respect to mechanistic investigations, the presence of the Taq A1 allele of the dopamine receptor D2 polymorphism has been linked to both GD and AUD.⁴⁶ This allele has been associated with increases in impulsivity on neurocognitive tasks,⁴⁷ suggesting the possibility that at least

part of the shared genetic variance between GD and alcohol dependence (12%–20%)⁴⁸ is due to a genetic predisposition toward the underlying construct of impulsivity.

Taken together, these findings lend support for the syndrome model of addiction, which posits that different objects of addiction share core diatheses and sequelae.³⁷ Although the end results are variable (eg, uncontrolled gambling versus uncontrolled heroin use), the underlying etiological substrates are highly overlapping, reflecting the phenomenon of multifinality, in which individuals with similar backgrounds in terms of risk and protective factors experience different developmental outcomes.⁴⁹

Neurobiology

The pathway from genes to behavior is hierarchical, reciprocal, and is modulated at an intermediate level by neural circuitry, which is constructed largely by way of genetic activity and which functions to regulate phenotypic behavior. For example, the mesocorticolimbic dopamine pathway modulates the reward value of addictive substances and behaviors.³⁵ A number of studies of GD and DUD have delineated genetic contributions to various aspects of this pathway, including density of D2 receptors and magnitude of dopamine release, which predict the subjective hedonic response.⁵⁰

Just as genetic contributions to behavior are multifaceted, the phenomenon of addiction is far too complex to be mediated by a single neurocircuit. Additional networks involved in the addiction process include the nigrostriatal pathway,⁵¹ the hypothalamic–pituitary–adrenal (HPA) axis,⁵² the insula,⁵³ and multiple prefrontal cortex (PFC) regions.⁵⁴ As a broad neurobiological model of addiction, Koob and Le Moal³⁶ postulated the existence of both 1) within-systems neuroadaptations, characterized by an elevated reward threshold (ie, tolerance) which are mediated by reductions in ventral striatum dopamine activity, as well as 2) between-systems neuroadaptations, in which anti-reward stress systems (eg, HPA axis, extended amygdala) are increasingly activated, causing a negative affect state (ie, withdrawal, craving) in the absence of the substance/behavior. These neuroadaptive changes are consistent with multistep theories of the progression to addiction³⁵ and can be superimposed upon an impulsivity-to-compulsivity spectrum shift in which initial engagement in the addictive behavior results from an impulse-driven desire for the hedonic effects. Subsequent behavior follows the development of tolerance and allostatic changes in key neurocircuitry, and, in contrast to initial engagement, is driven by a habitual, compulsive desire to attenuate anxiety and negative affect (ie, reduce craving,

avoid withdrawal). Evidence suggests that chronic substance use damages PFC networks critical for top-down modulation of behavior, reducing the ability to exercise the inhibitory control necessary for maintaining abstinence.⁵⁵ This residual damage may also help explain why other addictive behaviors can develop after the cessation of an initial addictive behavior and why relapse after a period of abstinence is more likely when GD is comorbid with DUD.

Koob and Le Moal's³⁶ model converges with support for predominant ventral striatal involvement in early drug use, followed by increasing dorsal striatum modulation as conditioned cues begin to supplant hedonic reward as the key motivator of behavior.⁵⁶ In this context, individuals experience cravings following repeated pairings of sensory cues (eg, the “pssht” of a beer can) and addictive behaviors (eg, alcohol consumption), via the process of classical conditioning. Eventually, the attenuation of aversive states (eg, cravings, withdrawal) associated with the addictive behavior becomes the primary driving force for continued engagement in the behavior. Interestingly, damage to a subregion of the insula, which is responsible for assessing internal mood and sensory states, eliminates the experience of craving.⁵⁷

In addition to the insula, another key neurobiological modulator of the anti-reward system is the HPA axis. This neuroendocrine pathway is disrupted with chronic exposure to substances, as well as during engagement in gambling,⁵⁸ which alters its ability to function effectively and efficiently.⁵² The changes to the HPA axis resulting from repeated substance use include increases in circulating adrenocorticotrophic hormone and corticosterone. These changes cause individuals with addictions to experience stress more intensely and for longer periods than others³⁶ and lead to a long-term increase in their susceptibility to the negative effects of stress.⁵⁹ Moreover, such modifications to the HPA axis are considered between-systems adaptations in the context of an addiction process, inasmuch as the recruitment of this stress response system partially mediates the experience of craving and predicts relapse.⁵⁹

Although Koob and Le Moal's³⁶ model was structured around substances, emerging evidence links key constructs involved in drug dependency to GD as well. For example, the impulsive–compulsive spectrum shift that occurs in DUD also takes place in GD.⁶⁰ Additionally, on a molecular level, evidence indicates that dopamine D2 receptors underlie the experience of reward secondary to both gambling⁶¹ and substance use.⁶² With respect to the dopaminergic influence on GD, dopamine agonists, often utilized as pharmacotherapy for individuals with Parkinson's disease and restless leg

syndrome, can lead to engagement in hedonic behaviors such as hypersexuality and gambling, ostensibly through dysregulation of the dopamine reward pathway.⁶³ Conversely, pharmacotherapy with dopamine antagonists has shown some efficacy in treating alcohol dependence,⁶⁴ although evidence has yet to support the efficacy of this approach in GD.^{65,66} Finally, as in DUD, the inability of individuals with GD to exhibit appropriate top-down impulse control may result from combinations of increases in sympathetic nervous system activity during gambling,⁶⁷ coupled with hyperactive stress systems,⁵⁸ as well as decreased activation in crucial PFC networks.⁶⁸

In spite of significant progress in elucidating the neurobiological underpinnings of both GD and DUD, much work remains to be done. Although strides have been made in integrating GD into preexisting models of DUD, the GD literature is still lacking in a complete and thorough understanding of the role of dopamine in the development of the disorder, which prevents its full inclusion in these broad theoretical models of addiction.^{35–36} Moreover, it is clear that neurotransmitters aside from dopamine contribute significantly to the addiction process,⁵⁴ but the empirical evidence involving serotonin, norepinephrine, and glutamate in GD is sparse.⁶⁹

Neurocognition

Addictive disorders are commonly associated with cognitive deficits, although there is significant variability in observed outcomes based upon the specific substance abused, as well as the intensity and chronicity of use. In individuals with alcoholism, deficits occur in the domains of executive functions (EFs) and visuospatial skills, while other abilities such as language and gross motor abilities are relatively spared.⁷⁰ Fortunately, these impairments resulting from chronic use can be at least partially ameliorated with prolonged abstinence. Individuals with GD also exhibit deficits in EFs,⁷¹ including decision making,⁷² inhibitory control,⁷³ and mental flexibility;⁷⁴ however, no studies to date have examined the impact of prolonged abstinence from gambling on these deficits. Another unresolved question in this literature pertains to whether these neurocognitive deficits are present pre-morbidly or whether they represent downstream phenotypic effects of physiological changes due to addictive behaviors. Several studies in GD and alcohol dependence generally support the presence of premorbid impulsivity in the larger population of individuals with addictions, although data from these investigations also indicate greater impairment in EFs such as working memory among individuals with alcohol dependence compared to those with GD,⁷⁵ possibly

suggesting that chronic ethanol ingestion selectively damages PFC circuitry. Moreover, convergent with neurocognitive findings, self-report data show that trait impulsivity tends to be elevated in GD, providing independent, multimodal evidence for preexisting inhibitory control deficits in addictive disorders.^{73,76}

Overall, findings regarding neurocognitive deficits in GD are informative, especially with respect to investigations in which GD is utilized as a behavioral model of addiction in order to address specific research questions.⁷⁵ However, a key limitation that has persisted in this literature is heterogeneity in cognitive tasks employed across studies, which limit direct comparisons and aggregate analyses.⁷⁷ Importantly, this line of research is still in its infancy, and as it continues to develop, more precise comparisons of neurocognitive profiles can be made between individuals with GD and those who abuse substances such as alcohol. Replicating findings across similar tasks and engaging in more prospective longitudinal designs⁷⁴ will solidify our understanding of impulsivity and other important cognitive constructs as they relate to both GD and DUD.

Treatment

Approximately, a third of individuals with GD and about one-quarter of those with alcohol dependence will recover naturally without need for treatment.^{78,79} Others will turn to treatment options including self-help and peer support, brief and motivational interventions, and cognitive-behavioral therapy (CBT) in order to regain control of their addictive behavior. These gambling treatments are largely based on those developed for alcohol and drug addictions, and research suggests that gamblers,⁸⁰ like those with substance-related addictions,⁸¹ benefit from such interventions. However, gambling treatment is not as widely available. In the following section, we briefly discuss common interventions for substance and gambling problems.

12-Step recovery program

Alcoholics Anonymous (AA) is a peer-led support group for those with alcohol use problems. AA meetings are widely available in the US and research indicates that participation is common and associated with improved outcomes. Kelly et al⁸² followed alcohol-dependent patients who were encouraged to engage in support groups following discharge from intensive outpatient treatment and 79% accessed these groups in the first year. Participation declined but remained substantial in the second (54%) and third (54%) years post-discharge and was associated with better drinking outcomes.

Other studies^{83,84} suggest that benefits of AA participation may be optimal when patients engage in AA in concert with professional treatment and that AA participation may be an important component in long-term recovery.

Gamblers anonymous (GA) is based on the 12-step philosophy pioneered by AA, and it espouses many of the principles found in AA, including an abstinence-only orientation, adoption of the disease model of addiction, and conceptualization of addiction as a chronic illness. GA appears to benefit those with greater addiction severity,⁸⁵ but the aforementioned characteristics (eg, abstinence orientation) may reduce its appeal for some individuals. There is relatively little data on GA as a stand-alone treatment, but available studies^{85–87} suggest that GA's benefits as a sole intervention are modest, possibly as a consequence of high drop-out rates. However, GA involvement in concert with professional treatment does seem to enhance treatment outcomes,⁸⁸ and it remains a recommended component of some professionally delivered treatments.⁸⁹

Self-help

Self-help treatments offer many benefits not found in 12-step meetings or professionally delivered approaches such as privacy, cost savings, convenience, and accessibility.⁹⁰ Bibliotherapy for alcohol problems generates small to medium effect sizes compared to no treatment controls, may be equally effective as more extensive interventions, and appears to lead to maintenance of treatment gains over long periods.⁹¹ Bibliotherapy has also been evaluated for problem gambling and is beneficial for gamblers relative to those randomized to wait-list controls.⁹² However, a randomized controlled trial (RCT)⁹³ and its 24-month follow-up⁹⁴ suggest that limited therapist contact may be an important component of effective bibliotherapy for gambling problems.

Motivational interventions

Motivational interventions may be ideal options for those with addictions who are ambivalent about changing behavior or seeking treatment. Meta-analysis of 55 randomized or quasi-randomized studies concluded that motivational interventions for those with AUD/DUD lead to significant reductions in drinking and substance use outcomes relative to no treatment controls and comparable outcomes relative to other active treatments.⁹⁵ Similarly, motivational approaches are an efficacious intervention for gambling problems. An RCT demonstrated that single-session motivational interventions of ~75 minutes can be effective among problem gamblers in reducing gambling frequency and dollars wagered

compared to a control interview, with effects persisting up to a year following the intervention.⁹⁶ More time-limited formats (eg, 10–15 minutes), including brief advice and personalized feedback, show promise for changing some gambling behaviors in those with problem or disordered level gambling.^{97,98} Interestingly, more extensive formats (eg, four sessions) of motivational enhancement combined with CBT do not consistently improve outcomes relative to brief or single-session formats in RCTs of individuals with problem or disordered gambling recruited from community⁹⁸ and college student⁹⁹ populations. This effect may be due to the inclusion of subclinical gamblers in these studies, who may not need or desire extensive treatments. For others, particularly those with GD, professionally delivered treatments of longer duration may be necessary for behavior change.

Cognitive and/or behavioral therapies

Professionally delivered, manual-guided CBT improves outcomes relative to GA or self-directed bibliotherapy in those with GD in RCTs.^{86,88} However, in an RCT¹⁰⁰ that included less severe college student gamblers, a 4- to 6-session CBT condition did not yield improved outcomes relative to a single session of personalized feedback. Other studies examining format (group versus individual) or comparisons of CBT to other active therapies generally find no differences amongst the comparisons groups.^{101–103} These findings mirror evidence from the treatment of alcohol dependence.¹⁰⁴

Although CBT for gambling is very similar to CBT for substance abuse treatment, cognitive therapies that focus explicitly on the distorted cognitions related to gambling are more unique in content. These therapies often involve more therapist contact (eg, up to 20 sessions) and demonstrate robust benefits relative to wait-list controls.^{105,106} However, these results will need to be replicated using larger sample sizes and using intent-to-treat analyses, as these studies^{105,106} excluded individuals who dropped out of treatment resulting in inflated treatment effects. Similar to other studies finding few differences among gambling treatments,¹⁰¹ an RCT¹⁰⁷ that compared cognitive therapy to other active therapies (eg, motivational interviewing, behavioral therapy) and used intent-to-treat analyses found no significant differences in gambling outcomes among the therapies.

Overall, AUD/DUD and gambling treatment research to date suggests that no one format or approach is ideal. Rather, it appears that most treatments are beneficial, with few differences found between active treatments when pitted against one another. Thus, persons with addictions who desire treatment have a wide range of options available to

them based on preferences, needs, and perhaps severity of their disorder. Moving forward, therapies may need to increasingly incorporate content that addresses the high comorbidity between GD and other psychiatric disorders, including, anxiety, mood, personality, alcohol, and drug disorders.²² Evidence suggests that psychiatric symptoms are responsive to and improve during gambling treatment.¹⁰⁸ However, room for further improvement in psychiatric symptoms remains among those with more severe presentations,¹⁰⁹ suggesting that these individuals need specialized and integrated content to address comorbid conditions.

Conclusion

A major challenge in comprehensively delineating links and risks across GD and AUD/DUD pertains to the asymmetrical nature of research on addiction-related disorders in which GD is a nascent field of inquiry with a dearth of funding in comparison to other addictions.¹¹⁰ Nevertheless, recent investigations have begun to elucidate the developmental progression of GD,¹¹¹ suggesting that the etiology of GD is complex, epigenetic, and includes a multitude of both proximal and distal predictors. Moreover, these models are similar in nature to developmental psychopathology models of AUD/DUD, suggesting significant overlap and common risk factors. As evidence accumulates, we are able to integrate decades of research into broad, inclusive models of addiction³⁷ that incorporate behavioral addictions such as GD.

Research addressing questions such as harms and economic costs related to subclinical gambling and whether subclinical gamblers experience negative consequences on par with the milder diagnostic forms of AUD/DUD is needed. These studies will be important for future DSM revisions regarding decisions about whether to model GD criteria and thresholds more closely to those for AUD/DUD. Another research priority is investigation of treatment approaches, particularly integrated treatments that address comorbid disorders or underlying dysfunctions (eg, impulsivity). The high rates of comorbidity suggest that such integrated treatments are an area of high need and have great potential. Unfortunately, the GD treatment literature is less well developed in this respect than other addictions.

In terms of clinical practice, we recommend screening for non-gambling psychiatric disorders among those seeking treatment for gambling problems. Routine screening for psychiatric disorders among treatment-seeking gamblers may help these patients obtain needed treatment for comorbid disorders more quickly and has the potential to improve response to both GD and the comorbid disorder when such

treatment is offered concurrently or in an integrated manner. In addition, within AUD/DUD treatment clinics, the higher prevalence of gambling disorder within this population suggests that systematic screening for gambling problems is warranted.^{31,112}

GD, as the first nonsubstance behavioral addiction, sets the bar for consideration of other disorders as behavioral addictions in the future. As reviewed, GD shares many features across many domains with AUD/DUD, leading some investigators³⁷ to espouse a syndrome model of addiction, which highlights the etiological overlap across the various manifestations of addiction (eg, uncontrolled gambling, alcohol use, or cocaine use). Researchers and clinicians alike should account for the substantial overlap in these conditions when conceptualizing psychopathology for the varied purposes of designing research studies, assessing for clinical symptomatology, and planning treatment.

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