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The Conceptualization and Diagnosis of Disordered Gambling

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Introduction

This is an introductory chapter designed to provide a broad overview of conceptual, diagnostic, and epidemiological topics for the treatment professional who may have limited experience with this population. As such, the chapter discusses competing conceptualizations of disordered gambling in the context of recent empirical work and discusses implications for the upcoming publication of the DSM-5. Research discussed in later chapters is touched on here to provide a foundation. Clinicians with significant experience with this population may wish to bypass this chapter and move directly to more specialized foci.

To be consistent with the DSM-5, we use the term "gambling disorder" or "disordered gambling" throughout this volume rather than the archaic terms "pathological gambling" or "pathological gambler." However, the reader should keep in mind that studies published prior to 2012 used the DSM-IV-TR diagnostic criteria to identify research participants. It is not known at this time how much effect the DSM-5 diagnostic criteria changes will have on the subsequent composition of research samples, although a significant effect is not expected. The vast majority of individuals who would have met the criteria for a DSM-IV-TR diagnosis of "pathological gambling" will also meet the DSM-5 criteria for a diagnosis of "disordered gambling."

Who is a disordered gambler?

Identifying disordered gamblers is a complex task for several reasons. First, disordered gamblers form a heterogeneous group. Although males are more likely to be diagnosed than females, a casual survey of any casino floor will inventory

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a dizzying array of players with varied demographic characteristics. Second, disordered gambling may be viewed as a psychiatric disorder in its own right or as the consequence of another underlying psychological condition. In most cases, the relationship is likely reciprocal – disordered gambling probably owes much to associated psychopathology (e.g., depression, anxiety, and substance use) and gambling can exacerbate comorbid conditions. Third, the diagnostic criteria for disordered gambling require considerable clinical judgment. Although extreme cases may reliably be identified, many cases are not so clear cut. Statistical analyses do not always clearly differentiate disordered gamblers from social or recreational gamblers when simply looking at wagers, bets won/lost, or total money lost (e.g., Braverman, LaBrie, & Shaffer, 2011).

Nonetheless, disordered gamblers may be differentiated from social or recreational gamblers, and non-gamblers, in several ways. These differences are observed across a number of dimensions: behavioral, cognitive, social, and neurobiological. Before discussing the diagnostic criteria for disordered gambling, and considering its evolution to DSM-5, it may be useful to contextualize the diagnostic criteria by identifying many of the defining characteristics of disordered gamblers.

Behavioral dimensions

Disordered gamblers evidence a reliable behavioral course: increased frequency and duration of gambling combined with an increase in the amount of money gambled. For example, LaBrie and Shaffer (2011) compared sports bettors on an internet web site who closed their accounts for gambling-related problems to those bettors who closed their accounts because of dissatisfaction with the gambling service or lost interest in betting. They found that the former group made more and larger bets, bet with a greater frequency, and were more likely to bet bigger amounts soon after joining the site. The tendency to increase the frequency and duration of play, along with the amount gambled, is a function of the gambler's predispositions and pre-existing conditions, game play structure, and the reinforcing effects of wins and near misses. Frequency of play has been shown to be related to overall gambling problem severity, especially for slot machines and video lottery terminal games (Holtgraves, 2009).

Increased play often follows a gambling urge that is precipitated by one or more gambling-related cues. Cues are stimuli that elicit a learned response because of associations made through classical and operant conditioning. Gambling-related cues can elicit changes in both measurable autonomic function (e.g., heart rate) and self-reported gambling urges. Wulfert, Maxson, and Jardin (2009) found that disordered gamblers reported stronger urges to gamble than social gamblers when exposed to videos of a horse race and a car race. In addition to well-known classical conditioning processes, all gambling operates on a variable or random ratio schedule of reinforcement that pays off unpredictably. The surprising nature of unpredictable rewards primes the brain for increased dopamine release. Dopamine is integral to motivated, goal-oriented activity. Because surprising rewards are delivered intermittently, gambling behavior is difficult to extinguish.

Cognitive distortions

Disordered gamblers engage in a variety of cognitive biases and errors compared to normal controls (Toneatto, 1999). Many of these errors are associated with other conditions and are not specific to disordered gambling. For example, when choosing between small, immediate rewards or larger rewards that are delayed, disordered gamblers discount larger delayed rewards and are more likely to choose smaller, more immediate rewards, a phenomenon also seen in substance use populations (Petry & Madden, 2010) and initially observed in animal studies (Madden, Ewan, & Lagorio, 2007). Disordered gamblers are also more likely than normal controls to think they can control independent events (Delfabbro & Winefield, 2000), with illusions of control more likely to occur in gamblers who are depressed (Källmén, Andersson, & Andren, 2008). Illusions of control are heightened when individuals perceive patterns in random events and assume that past outcomes predict future outcomes, a phenomenon euphemistically known as the Gambler's Fallacy. In addition to heuristic errors, the overwhelming preoccupation with gambling that disordered gamblers exhibit interferes with cognitive processing. Cognitive interference from gambling has been found in Stroop colorword studies that have shown disordered gamblers have longer latencies to name colors of gambling-related words compared to drug and neutral words (Boyer & Dickerson, 2003; see also Kertzman, et al., 2006) although not all studies have found an effect (e.g., Cooper, 2002).

Social and cultural factors

Because gambling behavior and other leisure activities are often mutually exclusive (e.g., one cannot simultaneously be at a casino and home with family), this means that the disordered gambler's world becomes increasingly constricted over time until sources of reinforcement may only be found in the gambling context. Increased stimulus control of reinforcement, and its strong association with gambling, means that abstaining from gambling becomes increasingly aversive while engaging in previously reinforcing activities becomes less rewarding. Although this phenomenon may be understood in terms of basic learning principles, what occurs within the individual reflects a biological change. In a recent fMRI study, de Greck and colleagues (2010) found that, relative to normal controls, the bilateral nucleus accumbens and the left ventral putamen cortex of disordered gamblers, both of which are involved in the brain's endogenous reward system, were *deactivated* when subjects viewed stimuli of high personal relevance. In contrast, these areas were activated when viewing gambling-related cues. It is reasonable to hypothesize that many of the social and interpersonal problems that gamblers face reflect underlying neurological sequelae to learning. Social interactions lose their saliency and reward value over time as the gambling addiction progresses.

Cultural influences on disordered gambling are profound and it has long been recognized that the prevalence of disordered gambling is largely a function of both opportunity and the degree to which the culture tolerates the activity. Native American Indian culture, for example, has a long history of gambling that predates the

current investment in casinos on reservation land. Historically, those cultures that have been the most accepting of gambling, despite periodic religious tensions and governmental interventions, have been from the industrial west, China, southern India, sub-Saharan Africa, and the western parts of South America (Binde, 2005). In contrast, Muslim countries, because of prohibitions on gambling in Islamic law, have shown far less tolerance for gambling.

Neurobiological substrate and psychophysiology

Over the past ten years, neuro-imaging research has consistently found that the brains of disordered gamblers respond to gambling-related stimuli in ways that are similar to the response patterns of addicted substance users. Specifically, the pleasure centers of the brain that are mediated by dopaminergic transmission (i.e., the mesolimbic dopaminergic system) appear to be active in both substance and behavioral addictions. PET scans have shown disordered gamblers with dopamine release in the ventral striatum during a gambling simulation task reported higher levels of excitement, and exhibited poorer performance, than normal controls (Linnet, Møller, Peterson, Gjedde, & Doudet, 2011). Studies using fMRI technology have recently reported a number of differences in the brains of disordered gamblers, including activation of the dorsolateral prefrontal cortex and the visual processing centers of the brain with corresponding subjective reports of increased craving (Crockford, Goodyear, Edwards, Quickfall, & el-Guebaly, 2005) and decreased activity in the left ventromedial prefrontal cortex (Dannon et al., 2011; Tanabe et al., 2007; Potenza, Leung, et al., 2003). Unfortunately for gamblers, efficient decision-making may require that the ventromedial prefrontal cortex is activated, not deactivated (Northoff et al., 2006). These findings are consistent with results from studies of depressed and alcohol-dependent individuals that show impaired decision-making and resistance to learning, both of which are linked to deactivation of the left lateral orbitofrontal cortex (Jollant et al., 2010; Claus, 2009). Lower neuronal activity in the cortex may be complemented by increased activity in the ventral striatum during gambling, and it is interesting to note that dopamine agonists enhance this effect (Abler, Hahlbrock, Unrath, Grön, & Kassubek, 2009).

Beyond the fMRI evidence that the brain operates a little differently in disordered gamblers, some studies have shown disordered gamblers also exhibit distinctive autonomic arousal relative to normal controls in response to gambling cues and paradigms (Wilkes, Gonsalvez, & Blaszczynski, 2010), although not all studies have found this effect (e.g., Diskin & Hodgins, 2003). In addition to change in autonomic responding, hormonal changes also take place with salivary testosterone increasing in poker players (Steiner, Barchard, Meana, Hadi, & Gray, 2010) and correlated with choosing the riskiest decks in the Iowa Gambling Task, a computerized measure of executive functioning (Stanton, Liening, & Schultheiss, 2011).

Motivational aspects

Although disordered gamblers show a diversity of motivations to gamble, research consistently shows that individuals with the most severe gambling problems gamble to alleviate, avoid, or cope with aversive emotional states and dysphoric

mood (Stewart, Zack, Collins, & Klein, 2008). It is, therefore, important not just to focus on the specific symptoms and comorbid conditions evidenced by a disordered gambler, but to consider the function gambling plays as a complex avoidance and escape behavior. Thus, disordered gambling effectively functions in the short-term as a regulatory mechanism for unpleasant emotional states (see Ricketts & Macaskill, 2004).

Diagnosis

Diagnostic critieria for Gambling Disorder

Although the DSM-IV-TR (American Psychiatric Association, 2000) classified pathological gambling (PG) as an Impulse Control Disorder not Elsewhere Classified, the revised DSM-5 version of the diagnostic criteria characterizes it as a behavioral addiction called Gambling Disorder. The name change was in response, partly, to Petry's (2010) suggestion that the new label would be less pejorative. According to the DSM-5, in order to diagnose a Gambling Disorder, the clinician must consider at least four of the following criteria to be present:

(1) A need to gamble with increased amounts of money in order to achieve same level of excitement;
(2) Restlessness or irritability when attempting to cut down or stop gambling;
(3) Repeated efforts to control, cut back, or stop gambling have not been successful;
(4) Often preoccupied with gambling (e.g., reliving past gambling experiences, planning one's next gambling experience, thinking of ways to raise funds to gamble);
(5) Often gambles when feeling distressed (e.g., helpless, guilty, anxious, or depressed);
(6) Chasing one's losses – after losing money, returns the next day to win losses back;
(7) Lying to conceal the extent of the gambling problem;
(8) Has jeopardized or lost a significant relationship, job, or other opportunity (educational/career) because of gambling;
(9) Relies on others (e.g., family, friends, acquaintances) to provide money to relieve a desperate financial situation.

Changes from DSM-IV-TR to DSM-5 The DSM-5 (APA, 2013) represents a departure from the DSM-IV-TR in a number of ways. For reasons that will be described later, the symptom "commits illegal acts in order to fund gambling opportunities" was omitted from the diagnostic criteria. Although many gamblers resort to illegal acts to fund their gambling, many do not. Instead, they drain their own financial resources and the resources of others. Although financially catastrophic, no laws are actually broken. Another important change was that the number of symptoms required for a diagnosis to be given was reduced from five to four. Using a questionnaire that measured the DSM-IV diagnostic criteria with 259 men and women admitted to a gambling treatment program, Stinchfield (2003)

found that the reduction from five required symptoms to four improved classification accuracy and reduced the rate of false negatives. The final significant change in the DSM-5 was the migration of the disorder itself from being classified as an impulse control disorder to a behavioral addiction. We discuss this issue in more detail later in the chapter.

DSM-5 criteria for "Gambling Disorder"

Although the DSM-5 version of the disorder is similar, there are some striking differences. A variety of psychometric studies has indicated that the DSM-IV-TR diagnosis includes a symptom constellation that does not optimally represent the disorder. As of October 22, 2012, the American Psychiatric Association has indicated through its website that "Pathological Gambling" will be reclassified as a "Gambling Disorder" and moved from the "Impulse-Control Disorders not Elsewhere Classified" to a new category entitled "Addiction and Related Disorders."

In contrast to the DSM-IV-TR, the DSM-5 version of the disorder includes nine rather than ten symptoms. The eighth DSM-IV-TR symptom, committing illegal acts to support one's gambling, has been removed. The symptom concerning lying to others does not specify individuals to whom a person has lied (e.g., family members, mental health professionals, etc.), rather it emphasizes that the function of lying is to "conceal the extent of involvement with gambling". Course specifiers will include episodic, chronic, and in remission.

Which symptoms represent core features of a gambling disorder?

As with other disorders, a diagnosis of disordered gambling is polythetic in that a variety of combinations of symptoms may be present across individuals, thus allowing significant individual differences in symptom presentation. Given that all of the research to date preceded the DSM-5 redefinition of the disorder, one should keep in mind that future psychometric studies of the criteria for a gambling disorder may yield different results from those employing "pathological gambling" criteria. Although we use the terms "disordered gambling" or "gambling disorder" throughout, this is an important caveat to bear in mind.

Researchers have examined whether some symptoms are better than others in predicting diagnostic presence of disordered gambling. This kind of question is frequently addressed using signal detection methodology in which each symptom is examined for its sensitivity and specificity to diagnosis. In other words, is the presence of a symptom predictive of a subsequent diagnosis? If a symptom is *sensitive*, this means that it is often considered present. It will predict a diagnosis of disordered gambling, but it will be associated with many false positive diagnoses. If a symptom is highly specific to the disorder, the symptom is less frequently considered present. Thus, highly specific symptoms are more likely to accurately identify true cases of disordered gambling than highly sensitive symptoms that have a high false-positive rate. On the other hand, highly specific symptoms may have a

higher miss, or Type II error, rate. Typically, as measures become more sensitive, they become less specific, and vice versa.

Research suggests that some symptoms are better than others at predicting disordered gambling. In fact, there is considerable evidence that simply asking two questions may provide both high sensitivity and high specificity in diagnosis. The Lie/Bet Scale (Johnson, Hamer, Nora, & Tan, 1997) considers a person to be positive for disordered gambling if either of the following two questions is endorsed: "Have you ever had to lie to people important to you about how much you gambled?" or "Have you ever felt the need to bet more and more money?" In a sample of Gamblers Anonymous members and control participants, the Lie/Bet Scale correctly classified all of the GA members as clinical cases, and incorrectly classified only 9% of control group members. Thus, overall sensitivity was excellent (0.99) and specificity was also high (0.91). Subsequent studies (e.g., Johnson, Hamer & Nora, 1998; Götestam, Johansson, Wenzel, & Simonsen, 2004) found similar results although not quite as impressive as in the original study. Across studies, the authors concluded that the Lie/Bet Scale is a reasonably accurate and efficient screening tool for community-based samples.

The development of the NODS-CLiP, a DG screening tool, similarly showed that a small subset of items may be enough to reliably diagnose DG. Toce-Gerstein, Gerstein, and Volberg (2009) administered the NORC Diagnostic Screen for Gambling Disorders (NODS) to 8867 gamblers and found that three questions assessing failed efforts to control or stop gambling, lying to family members or important others, and preoccupation with gambling activities successfully identified virtually all disordered gamblers. Another recent attempt to develop a brief three-item screening scale was reported by Gebauer and colleagues (2010). Using data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), they found that endorsement of any one of the following three items efficiently identified disordered gamblers: Criterion 4, restless/irritable when attempting to cut down or stop gambling; Criterion 7, lying to family members, therapist, or others to conceal the extent of one's gambling; and Criterion 10, borrowing money from others to relieve a desperate financial situation. In total, 78 of 79 disordered gamblers endorsed these three items, compared to only 226 of 10 801 other respondents. Sensitivity for the three-item scale was 0.99, with positive predictive value of 0.26. These results were comparable to those with the Lie/Bet Scale.

Zimmerman, Chelminski, and Young (2006) reported the results of a semi-structured diagnostic interview (i.e., the Structured Clinical Interview for DSM-IV, or SCID) with 1709 psychiatric outpatients, 88 of whom screened positive for the diagnosis. Of those 88, 40 met the DSM-IV diagnostic criteria for a lifetime history. These patients were more likely to be male and were less likely to have graduated from college. No other demographic variables predicted caseness. The authors compared those who ultimately met diagnostic criteria ($n = 40$) to those who did not ($n = 48$) according to the SCID. Individuals meeting diagnostic criteria were significantly more likely to be considered positive for all of the ten diagnostic symptoms than patients who did not meet the diagnostic criteria. Endorsement of the eighth criterion (commitment of an illegal act) was infrequent and occurred only in the most severe cases. Consistent with results regarding the Lie/Bet

Questionnaire, the three criteria that were the most sensitive to the presence of disordered gambling were: Criterion 1, preoccupation with gambling; Criterion 7, lying to family; and Criterion 6, chasing losses.

In summary, research to date suggests that symptoms focusing on preoccupation with gambling, lying to others, betting in successively increasing amounts to achieve the same effect, and finding oneself in dire financial straits appear to be central to the diagnosis. Items measuring criminality (e.g., theft, embezzlement) are likely present in the most severe cases.

Factor Structure

Generalizing across instruments regarding factor structure is hazardous given that factor analyses are content-dependent statistical procedures. To the degree that different measures represent the facets of disordered gambling to varying degrees, factor structures should be expected to vary. Nonetheless, a few general trends have been observed when factor analyses have been conducted on instruments that are explicitly derived from the diagnostic criteria, and these results may inform our understanding of the construct.

The most common result has been a single factor solution on which all ten of the DSM-IV-TR symptoms load. Brooker, Clara, and Cox (2009) examined the factor structure of the Canadian Problem Gambling Index (CPGI) using a sample of 742 moderate-to-high risk problem gamblers. Using principal components exploratory factor analysis, they found evidence of high internal consistency (Chronbach's $\alpha = 0.86$) with loadings ranging from 0.52 (borrowing money or selling belongings) to 0.78 (feeling like one has a gambling problem). Similarly, Arthur et al. (2008) found in a sample of Singaporean students a single factor structure for the CPGI with all factor loadings exceeding 0.44, with the exception of the item measuring family financial problems as the result of gambling. Unidimensionality was also observed in a study done by Orford and colleagues (2010) with a community sample. An important finding from their study was that a questionnaire based strictly on the DSM-IV diagnostic criteria yielded two factors – with gambling-related crime and "chasing losses", two symptoms most likely present in the most severe cases, on the smaller second factor.

Research on the South Oaks Gambling Scale (SOGS) has been less consistent in settling on a clear factor structure. The lack of clarity may be due to low correspondence in item content between the SOGS and the DSM-IV criteria given that the SOGS was developed to conform to the DSM-III diagnostic criteria (Petry, 2007). Richard, Nguyen, and Joyner (2009) noted content validity issues vis-à-vis the DSM-IV diagnostic criteria in that the SOGS over-represented financial problems while other DSM-IV symptoms were not represented at all. Because financial problems often occur in the later stages of problem gambling, the SOGS may not function well as a screening tool. As such, it is not surprising that Orford, Sproston, and Erens (2003) found a two factor structure on the SOGS, described as dependence and gambling-related problems. Finding consistency in the SOGS factor structure has been elusive with other researchers reporting a three factor solution (Oliveira, Silva, & da Silveira, 2002). An adolescent version of the

instrument has evidenced a two factor solution as well, but the factor labels (Control Over Gambling and Other Gambling Consequences) suggest qualitatively different factor content (Wiebe, Cox, & Mehmel, 2000).

Overall, factor analytic results appear to suggest that the construct of disordered gambling is mostly unidimensional with certain symptoms reflecting more advanced pathology possibly forming a smaller second factor. The DSM-5 revision of the disorder addresses this issue with the removal of the criminal activity criterion (DSM-IV-TR item 8).

Subtypes

As noted earlier, disordered gamblers constitute a heterogeneous population with significant behavioral and demographic variability. They may be found in almost any age group, among both sexes, across a diverse range of ethnicities and cultures, and in a plethora of gambling venues and gaming contexts. Although the diagnosis may be useful clinically in providing a shorthand description of a prototypical disordered gambler, its descriptive focus results in a relatively simplistic distillation of (mostly) core features likely to co-vary across individuals. Subtyping gamblers into relevant motivational patterns, however, may have more clinical utility because, unlike a descriptive diagnosis, gambling subtypes that suggest *why* a person gambles may speak to the function of the behavior and have greater heuristic value for treatment.

A review of disordered gambling subtypes by Milosevic and Ledgerwood (2010) traced the effort back to Moran (1970). In Moran's initial typology, disordered gamblers were divided into five motivational subtypes that varied largely in terms of the hypothesized intrinsic and extrinsic causes of the disorder. For example, on the extrinsic pole, a *subcultural* gambler was one who gambled as the result of pressure from family or peers and a *neurotic* gambler was one who gambled in response to stressful life events or life situations. Intrinsic motivational states characterized the *psychopathic* gambler (who gambled because of significant personality disturbance), the *impulsive* gambler who has lost the ability to control his or her gambling, and the *symptomatic* gambler who gambled as a result of another underlying psychological disorder.

Researchers who subsequently subtyped disordered gamblers did so from an array of theoretical perspectives utilizing varying levels of empirical support and a potpourri of statistical procedures. A partial list of hypothesized subtypes included the personality disordered, paranoid, and depressive-anxious (Graham & Lowenfeld, 1986), chronically under-stimulated (McCormick, 1987), boredom prone (Blaszczynski et al., 1990), escape and action seekers (Lesieur, 2001), sensation seekers (Steel & Blaszczynski, 1996; Bonnaire, Bungener, & Varescon, 2009), behaviorally conditioned, emotionally vulnerable, and antisocial impulsivist (Blaszczynski & Nower, 2002), dissociative (Ledgerwood & Petry, 2006), coping and enhancement (Stewart & Zack, 2008; Stewart et al., 2008), and demoralized or hedonic (Vachon & Bagby, 2009).

Recently, Milosevic and Ledgerwood (2010) comprehensively inventoried gambling subtypes that have been proposed over the last thirty years. They concluded

that three subtypes emerged and that the subtypes were consistent with those proposed by Blaszczynski and Nower (2002). The first subtype was characterized by elevated levels of depression or anxiety. Gambling functions for these individuals as an avoidance and/or escape response and corresponds to Blaszczynski and Nower's (2002) "emotionally vulnerable" gambler. The second subtype of disordered gambler identified by Milosevic and Ledgerwood is characterized by high levels of impulsivity and low tolerance for boredom. Gambling functions to stimulate the central nervous system and is one of many activities engaged in by these individuals that might best be described as "sensation seeking" behaviors. The third subtype of disordered gambler is behaviorally conditioned to gamble. These individuals generally do not evidence impulsivity, sensation seeking behavior, or other forms of psychopathology (e.g., depression, anxiety), but they often make significant cognitive errors in how they think about gambling. Interestingly, Milosevic and Ledgerwood's (2010) conclusions are consistent with factor analytic findings reported by Steel and Blaszczynski (1996) who gave eighty subjects a series of personality measures. Four factors emerged and were labeled: psychological distress, sensation seeking, crime and liveliness, and impulsive-antisocial.

It would be difficult to imagine that the subtypes represent orthogonal, or even distinct, profiles within which individual disordered gamblers may neatly fall. Although the defining features of one subtype may predominate in any given case, this is not to say other compelling motivations to gamble, or other forms of psychopathology, will be absent. Clinicians should assess clients vis-à-vis empirically validated subtypes, provided the subtypes show utility in treatment planning.

Given the potential utility of subtyping, it is somewhat surprising that the relationship of subtypes to criterion-related validity and treatment utility is limited. Relatively few studies have examined whether subtyping might help clinicians understand the severity of psychopathology, select treatment, or predict treatment outcome. For example, Stewart and colleagues (Stewart & Zack, 2008; Stewart, Zack, Collins, Klein, & Fragopoulos, 2008) found that individuals classified as "coping" gamblers (i.e., characterized by higher levels of depression and anxiety for whom gambling is negatively reinforced) showed greater severity of gambling problems. They also scored higher on a measure of problem drinking. For these individuals, gambling and alcohol likely function as escape behaviors from negative cognitions and aversive physiological states. The complex relationship with substance abuse is especially important to consider given that other researchers have found greater prior alcohol use and a history of substance abuse treatment have been associated with case severity but not treatment outcome (Stinchfield, Kushner, & Winters, 2005).

Epidemiology

Much of what we know about the epidemiology of disordered gambling comes from large-scale, nationally representative surveys, like the National Epidemiologic Survey on Alcohol and Related Conditions, (NESARC) or the National Comorbidity Survey-Replication (NCS-R) and the subsequent data analyses conducted by several different research groups. These researchers have found that

disordered gambling may be diagnosed in slightly less than 1% of the population while the at-risk population is much larger. In analyzing NESARC data, Blanco, Hasin, Petry, Stinson, and Grant (2006) found that 0.64% of men and 0.23% of women met diagnostic criteria. In addition, subthreshold levels of gambling occurred in 6.79% of men and 3.26% of women. These rates are almost identical to those found by the NCS-R as reported by Kessler and colleagues (2008). They reported that 2.3% of Americans endorsed at least one diagnostic criterion of disordered gambling while 0.6% would be classified as a disordered gambler. Risk factors included youth, male sex, and being non-Hispanic African-American.

For the most part, studies in other countries have found similar prevalence rates. In Switzerland, Brodbeck, Duerrenberger, and Znoj (2009) reported 2% of the population was at-risk for a lifetime gambling problem while 0.3% would be classified as disordered gamblers. Jonsson (2006) found past year prevalence to be identical in a review of survey research conducted in Iceland, Norway, and Sweden. Caution should be exercised in generalizing these findings, however, since higher prevalence rates have been observed in regions with greater liberalization of gambling laws and access to casinos or gaming terminals (Cox, Yu, Afifi, & Ladouceur, 2005).

Race/Ethnicity

As part of the National Epidemiologic Survey on Alcohol and Related Conditions, Alegría et al. (2009) reported prevalence rates of disordered or at-risk gambling in the United States are higher in African-American (2.2%) and Native American populations (2.3%) than in the Caucasian population (1.2%).

Sex Differences

Although seen as a predominantly male problem, researchers have found sex differences both in the types of games played and the speed by which a gambling disorder develops. When looking at the frequency with which certain games were played, Holtgraves (2009) used factor analytic techniques to identify a male-dominated first factor composed of sports betting, internet play, using a bookie, and horse racing; and a second factor composed of games more likely to be utilized by women that included lotteries, raffles, bingo, and video lottery terminals. Gambling severity scores on the Canadian Program Gambling Index (CPGI) were higher for the first than the second factor suggesting sex differences both for the types of games played and the severity of the ensuing gambling problem.

Men and women also differ in terms of the speed in which gambling problems develop. Women generally develop gambling problems more quickly than men. The effect has been termed "telescoping" and has been attributed to both the higher rates of comorbid depression in females relative to males and differences in the structural characteristics in games females prefer (Tavares et al., 2003). Age appears to mediate the effect as it is more pronounced in middle aged and older women (Grant & Kim, 2004). This is of clinical interest because women also tend to be older than men when they start gambling, when problems develop, and when

they initiate treatment (Ladd & Petry, 2002). Women are also more likely than men to have a lifetime anxiety or mood disorder (Blanco, Hasin, Petry, Stinson, & Grant, 2006). Men meeting the diagnostic criteria, or just short of the diagnostic threshold, are significantly more likely than women to smoke two or more packs per day, drink heavily, and have a history of substance use.

Age

Disordered gambling is a diagnosis with higher prevalence rates at the opposite poles of the age continuum. Specifically, research has accumulated that teens, college-age gamblers, and elders may show the highest rates of disordered gambling. Winters, Bengston, Dorr and Stinchfield (1998) found that 2.9% of college students were probable disordered gamblers. Among student-athletes, this rate balloons to 4.3% in men but does not deviate from adult population rates in female athletes (Huang, Jacobs, Derevensky, Gupta, & Paskus, 2007). With elders, DG prevalence may increase partly because of diseases that require the prescription of dopamine agonists. Combined with life and health stressors that typically occur later in life, this may explain the increased estimated prevalence of DG in seniors to between 1 and 3.2% of the population, depending on the study (Barns, Rickards, & Cavanna, 2010; Philippe & Vallerand, 2007). The types of games people gamble on also vary as a function of their age. Stevens and Young (2010) found that gamblers who played games with a high chance component (e.g., slot machines) were more likely to be over 35 years of age. In contrast, younger players preferred games characterized by higher levels of required skill (e.g., poker).

Comorbid Conditions

Although comorbidity will be discussed in more detail in another chapter in this volume, disordered gambling is frequently comorbid with substance use and mood disorders. Using NESARC data, Petry, Stinson, and Grant (2005) reported a variety of comorbid conditions that occurred in high frequency with disordered gambling: alcohol use disorder (73.20%), personality disorder (60.80%), nicotine dependence (60.40%), mood disorder (49.60%), anxiety disorder (41.3%), and a drug use disorder (38.10%). It is rare to observe a disordered gambler without a comorbid condition, and it is often the comorbid condition that ultimately leads the individual to treatment (Afifi, Cox, & Sareen, 2006).

The DSM-5 Conceptualization: Why An Addiction Model Prevailed

As noted above, there were two different, but related, types of problems with the DSM-IV-TR criteria for disordered gambling: problems at the symptom level and problems concerning the conceptualization of the disorder and how it is categorized with respect to other disorders. These problems have stimulated work aimed at

reconceptualizing, and reclassifying, disordered gambling either in terms of extant obsessive-compulsive or substance abuse models.

Symptoms and classification

The DSM-IV-TR ten symptom disorder evidenced construct validity problems. Nelson, Gebauer, LaBrie, and Shaffer (2009) concluded that the diagnostic facets were not sensitive enough to pick up on lower levels of problem gambling. Some diagnostic criteria (e.g., lying about gambling, illegal activities) have been found only in the most severe cases, suggesting that the current diagnostic criteria are insensitive in detecting emergent or at-risk gamblers. Researchers also discovered that not all symptoms are stable over time, as measured by symptom count over the last year and prior to the last year. This could suggest either temporal inconsistency in the diagnostic criteria or a nonlinear course in the development of DG symptoms.

Zimmerman, Chelminski, and Young (2006) contended that when diagnoses are made based on a minimum number of criteria, each symptom should be present in at least half of the people with the disorder. They found that criterion 8, committing illegal acts to gamble, and criterion 10, relying on others to finance gambling, did not meet this requirement. These symptoms are highly specific to disordered gambling and may be better indicators of disorder severity than of disorder presence.

In the DSM-IV-TR, pathological gambling was categorized as an Impulse Control Disorder-Not Otherwise Specified, along with Trichotillomania (TTM), Intermittent Explosive Disorder, Kleptomania, and Pyromania. Impulse control disorders in the DSM-IV-TR were characterized by the inability to resist an urge to engage in a behavior, arousal and discomfort experienced before the behavior occurs, and perceived relief, although transient, shortly after completing the behavior (Dell'Osso, Altamura, Allen, Marazziti, & Hollander, 2006). In many ways, these characteristics parallel models of Obsessive Compulsive Disorder (OCD). However, the diagnostic criteria for gambling also mirror substance abuse models (Westphal, 2007b).

Competing Models

DSM-5 workgroups considered two competing models for disordered gambling: the obsessive-compulsive spectrum model and the substance abuse model. Ultimately, the substance abuse model, which conceptualized a gambling disorder as a behavioral addiction, won out and the diagnosis was included in a new category entitled "Addiction and Related Disorders." In addition to reclassifying disordered gambling as an addiction-related disorder, the DSM-5 version of the diagnostic criteria supports lowering the severity threshold for diagnosis. This will be done, in part, by removing criterion 8 (i.e., committing an illegal act to support one's gambling behavior). In addition, only four symptoms are now necessary to make a diagnosis. In order to help the reader understand the distinctions between these competing conceptualization models, a brief review of both the Obsessive-Compulsive Spectrum model and the Substance Use Disorder model is provided below.

The Obsessive-Compulsive Spectrum Model

Conceptual argument The Obsessive-Compulsive Related Disorders Work Group was one of two groups charged with reviewing and revising the diagnosis in 2006, as impulse control disorders share many features with the Obsessive-Compulsive pathology (Westphal, 2007b). Members of the committee contended that OCD should be removed from the anxiety disorders section and placed in its own autonomous category called Obsessive-Compulsive Spectrum Disorders (OCSD; Bartz & Hollander, 2006). A number of disorders would be included within this category along a continuum ranging from compulsive to impulsive. Disordered gambling was among the disorders considered for inclusion within the spectrum. The rationale for placing disordered gambling in the Compulsive-Impulsive (C-I) cluster was based on the observation that gambling reflects impulsive behavior that functions to relieve stored anxiety, a supposed common feature between OCD and disordered gambling (Bartz & Hollander, 2006). C-I shopping, C-I sexual behaviors, C-I internet use, and C-I skin picking were among the disorders also considered for inclusion within the cluster (Dell'Osso et al., 2006). Bartz and Hollander (2006) further contended that OCD and ICDs should be combined within a single diagnostic category because of greater comorbidity and behavioral similarities with OCD and Impulse Control Disorders than OCD and Anxiety disorders.

Serotonergic system comparisons An OCSD conceptualization of disordered gambling has been supported by research showing dysregulation of serotonin pathways in both OCSD and DG. This is not surprising, as serotonin depletion is implicated in impulse control and risky decision-making in both humans and animals (Long, Kuhn, & Platt, 2009). Because selective serotonin reuptake inhibitors (SSRIs) have been used to treat OCD, it should follow that disordered gamblers would also experience similar benefits. Reviews of SSRIs have been mostly favorable. Grant, Kim, and Potenza (2003) concluded that SSRIs may be effective in the short term reduction of DG symptoms, are well tolerated, require higher doses relative to the treatment of depressive disorders, and appear to exert treatment effects on DG that are independent of treating underlying depressive symptoms. High placebo response rates, however, warranted caution in interpreting results. Methodological problems, including the low number of female participants, high attrition rates, and variability in the magnitude of the placebo response have led some researchers to consider the evidence of efficacy mixed at best (see, for example, Iancu, Lowengrub, Dembinsky, Kotler, and Dannon, 2008). In addition, it is debatable whether treatment effects reflect evidence of a primary serotonergic pathway for OCSD and disordered gambling. Given that both serotonergic and dopaminergic pathways are implicated in disordered gambling, and given that serotonin effectively modulates other neurotransmitters like dopamine, it may be the case that SSRIs indirectly modulate dopamine by inhibiting serotonin reuptake. Further, low levels of serotonin may be generally related to states that make risk-taking more likely (e.g., impulsivity) and gambling is just one manifestation of a biological predisposition to be impulsive.

Behavioral pattern comparisons Although researchers have found a strong relationship between obsessive-compulsive traits and DG, the same cannot be said of Obsessive Compulsive Disorder and disordered gambling (Durdle, Gorey, & Stewart, 2008). In fact, individuals with disordered gambling evidence lower rates of comorbid OCD than other impulse control disorders, like trichotillomania and kleptomania. This discrepancy suggests that an OCD model alone is not sufficient to account for disordered gambling (Dell'Osso et al., 2006). Individuals with OCD and disordered gamblers differ most notably in their risk-taking tendencies. Those with OCD often avoid and overestimate risk, whereas those with DG ignore the consequences of their gambling behavior and continue to gamble despite bad odds and dangers to their interpersonal and financial well-being.

Differences in risk assessment have been preliminarily demonstrated in fMRI studies that examine ventromedial prefrontal cortex functioning in participants with DG and OCD. Potenza, Leung, et al. (2003) found that disordered gamblers showed reduced ventromedial prefrontal cortex (vmPFC) activation when viewing scenes that induced gambling urges, suggesting less cortical inhibition of impulses. In contrast, OCD patients often show the opposite pattern – increased vmPFC activation in similar tasks (Saxena & Rauch, 2000). Thus, OCD patients may have increased anticipatory apprehension with corresponding activation of cortical inhibitory control centers, whereas disordered gamblers show deactivation in the same structures, a consequence of which is increased impulsivity and risk-taking.

Genetic comparisons Lochner et al. (2005) used cluster analysis and genetic monoaminergic genotype comparisons to study patterns of OCD and Obsessive Compulsive Spectrum Disorders. They found that DG fits within the "reward deficiency" cluster, along with trichotillomania, Tourette Syndrome, and hypersexuality, but there were no discernible genetic links within or between OCSD clusters. In an earlier study, Black, Goldstein, Noyes, and Blum (1994) did not find increased prevalence of disordered gambling in first degree relatives of 32 individuals diagnosed with OCD. Again, these findings suggest that an OCSD model for DG is not adequate to account for DG symptomatology (Lochner et al., 2005; Westphal, 2007b).

A Better Fit

The Addiction Model Potenza (2006) has contended that the paradigm for substance use disorders (SUDs) should be broadened to include the term "addiction" as the term "substance use" or "substance abuse" limits pathology to only substance-related problems. Accordingly, the DSM-5 includes a new "Addiction and Related Disorders" category within which "Gambling Disorder" will be included. The rationale for this change is based on a variety of research results that show the typography and course of disordered gambling is profoundly similar to substance use disorders with high levels of comorbidity (Potenza, 2006; Petry, 2006; Petry, 2010).

Casting disordered gambling within an addiction paradigm has been facilitated by emergent models of addiction that emphasize psychosocial components and

common neural pathways shared by substance-based and behavioral addictions. In models like the biopsychosocial component model advocated by Griffiths (2005), behavioral addictions (e.g., gambling, sex, exercise, work) are viewed as existing on a severity continuum with regard to the behavior's salience (the degree to which it becomes the most important activity in the person's life), mood modifying properties, tolerance development, withdrawal symptoms, interpersonal conflict, and relapse potential.

Research has correspondingly focused on identifying gambling symptoms that are consistent within an addiction framework. Cunningham-Williams, Gattis, Dore, Shi, and Spitznagel (2009) assessed the extent to which withdrawal-like symptoms were present in a sample of disordered gamblers. They found that 40.9% of their sample reported feeling at least one withdrawal-like symptom: disappointment, guilt, loss of control, or hopelessness when attempting to quit. A regression analysis indicated that these symptoms could not be attributed to comorbid depression (Cunningham-Williams et al., 2009). With disordered gamblers and alcohol dependent individuals, Blaszczynski, Walker, Sharpe, and Nower (2008) found that both groups endorsed questionnaire items indicating tolerance and withdrawal. An important question for gamblers, however, is whether tolerance can truly be demonstrated by increased bet sizing across gambling sessions. Whereas alcohol dependent individuals may increase alcohol consumption in order to achieve the same subjective effect (e.g., relaxation, euphoria), increased bet sizes do not appear to operate similarly for gamblers (i.e., bigger bets do not necessarily increase the magnitude of reported arousal). Instead, bet sizing may be a function of a probabilistic computation, correct or not, regarding the likelihood of achieving a subsequent win – a judgment that has no parallel in substance dependent individuals. Although disordered gambling may very well show symptoms of withdrawal and tolerance, much more work needs to be done to establish equivalence to drugs of addiction.

Reward system comparisons As noted above, all addictions operate on the mesolimbic dopaminergic pathway system and disordered gambling appears to function similarly. This pathway serves to regulate behavioral responses to both induced substances and conditional stimuli, is sensitive to surprising or unpredictable rewards presented by the environment, and fosters motivated, goal-oriented behavior. In short, the dopaminergic reward system mediates the likelihood that an instrumental behavior will recur if reward is contingent upon the behavior. Interestingly, recent research has shown that, in gambling, the reward system is activated not just during "wins" but when individuals experience losses or near misses. In disordered gamblers, it is also slower to deactivate in response to losses.

For example, Linnet, Peterson, Doudet, Gjedde, and Møller (2010) examined dopamine responses to net gains and losses on the Iowa Gambling Task (IGT). A net loss in the IGT indicates a proclivity to choose decks that provide high immediate rewards, but larger long-term losses. A PET scan assessed changes in binding potential at dopamine receptor cites in the ventral striatum. A negative change in binding potential suggests an increase in dopamine release. The researchers found that disordered gamblers had significantly lower binding-potentials after a net loss than the healthy controls, suggesting that their dopamine responses were more

sensitive to occasional moderate wins than to overall long term losses. This finding was consistent with a neurological explanation for the tendency for disordered gamblers to delay discount (i.e., show preference for smaller, immediate rewards over larger, delayed rewards). It also suggests that disordered gamblers may be less able to learn from losses because dopamine release is not inhibited in response to losing situations (Linnet et al., 2010). Similarly, Hewig et al. (2010) found that disordered gamblers showed a more pronounced dopamine response after winning a risky hand in black jack than non-gambling controls, indicating that disordered gamblers are more sensitive to unexpected rewards. Researchers corroborated this notion with the finding that disordered gamblers were more likely to make risky decisions in black jack hands following a losing hand than controls, thus suggesting that they may learn more readily from their enhanced dopamine release following wins than the negative consequences following losses (Hewig et al., 2010).

In keeping with the research on dopamine sensitivity, De Greck and colleagues (2010) found diminished deactivation of the reward centers in disordered gamblers during monetary loss events, suggesting a neural deficiency to distinguish the difference between wins and losses relative to controls. These findings suggest that a decreased ability to learn from errors, coupled with an increased sensitivity to rewards associated with gambling, could help to explain gambling perseveration. Parke and Griffiths (2004) may have explained this phenomenon in an earlier paper that found gamblers may consider near misses (e.g., a slot machine sequence that was almost a winner) to erroneously provide information about the likelihood of future wins. For example, a near miss may raise hopes that a future win is coming, thereby reaffirming the gambler's strategy and enhancing the value of the "miss" information. While there is no objectively meaningful difference between a near miss and a loss in a game where trials are completely independent of one another, disordered gamblers make the mistake of thinking the information is predictive of future events. Thus, the brain's reward system is activated not just when a win occurs, it is also activated when an individual perceives a win to have been narrowly out of reach or when the environment presents information that the individual errantly thinks may be useful in terms of securing a future reward (i.e., utility). In support of this notion, Chase and Clark (2010) found that gambling severity, as measured by the SOGS, predicted more pronounced dopaminergic responses in the substantia niagra and ventral striatum following a near win on a computer-simulated slot machine.

Not surprisingly, compensatory models of substance use and disordered gambling emphasize the possibility of a deficient reward system in the brain mediated by impaired dopaminergic transmission (Reuter et al., 2005). These individuals are often described as "sensation-seekers." Consistent with this model were findings by Reuter et al. (2005) who showed that disordered gamblers showed lower levels of mesolimbic, specifically ventral striatum activity than normal controls during a guessing task that was previously shown to activate the brain's reward system in the normal population. These results, and others already discussed in this chapter, suggest that disordered gamblers may have pre-existing deficiencies in the brain's reward circuitry that predispose one to excessive gambling. Similar patterns of deficient activation of reward centers have been observed in

drug-addicted individuals, suggesting a common pathway involving dopaminergic deficits.

Genetic and familial comparisons In contrast to the lack of findings between disordered gambling and obsessive-compulsive spectrum disorders, a study by Black, Monahan, Temkit, and Shaw (2006) found a familial link between disordered gambling and substance use. Researchers assessed 31 DG probands, 31 control probands, and 335 first degree relatives, using various structured interviews to assess gambling, substance use disorders, obsessive compulsive disorder, and impulse control disorders. Gambling problems and substance use disorders were significantly more common in first-degree relatives of disordered gamblers than normal controls. Impulse control disorders or OCD in first degree relatives were not overrepresented, suggesting that there is a stronger link between SUDs and disordered gambling than OCD and other impulse control disorders. Future research should study common genetic markers between probands and their relatives to explore the extent to which this familial link is genetic, environmental, or both. For a more exhaustive review of the extant literature on genetic aspects of disordered gambling and other similar disorders see Lobo and Kennedy (2006).

Serotonergic system comparisons Serotonergic systems are also implicated in both disordered gambling and substance use disorders. Pallanti, Bernardi, Quercioli, DeCaria, and Hollander (2006) tested the reactions of substance users and disordered gamblers to the serotonin agonist meta-chlorophenyliperazine (m-CPP). Substance users tend to experience a euphoric high when administered the serotonin agonist, whereas the normal population does not. Disordered gamblers reported experiencing a high more frequently than controls and placebo groups, suggesting that they and substance users share similar serotonin deficiencies and sensitivities to serotonin agonists. These results, coupled with similarities in dopaminergic reward deficiency, suggest common neurochemical pathways for substance abuse and disordered gambling.

Risk taking deficiencies and impulsivity When grappling with the issue of whether disordered gambling is an impulse control disorder, an obsessive compulsive spectrum disorder, or an addiction-like disorder, it is important to also address the cognitive processing deficits that may be specific to disordered gambling. As previously mentioned, disordered gamblers tend to have decreased activation in brain regions that mediate cognitive processing of risk and response inhibition, whereas OCD patients have increased activation in these regions (Potenza, 2008). There are some similarities in cognitive processing between disordered gamblers, substance users, and obsessive-compulsive individuals, but there are also fundamental differences.

Potenza (2008) found patterns of decreased activation in frontal lobe regions in disordered gamblers and substance users which could mediate a person's ability to inhibit drug use or gambling behavior (Lubman, Yücel, & Pantelis, 2004). Lack of inhibition coupled with a deficient reward system could contribute to patterns of delay discounting (choosing smaller immediate rewards over larger

long term yields) and other cognitive deficits observed in both disordered gamblers and substance users. Like substance users, disordered gamblers perform poorly on tasks that require higher cognitive functioning mediated by the prefrontal cortex (Lubman, Yücel, & Pantelis, 2004). Evidence of this deficit comes from studies exploring how gamblers perform on the Iowa Gambling Task, a computerized task in which participants must select cards from one of four decks, each of which is associated with a different schedule of reinforcement (i.e., wins and losses). On the IGT, they found that disordered gamblers consistently chose disadvantageous decks characterized by moderate wins and big losses (and large losses over the long run) as opposed to decks that presented small wins and small losses but were advantageous over the long run. Poor performance on the IGT has been shown to be associated with ventromedial prefrontal cortex (vmPFC) damage. In addition to disordered gamblers and substance users, there is a subset of OCD patients that do not respond to SSRIs and who also evidence similarly skewed choice patterns on the IGT. This subset tends to show higher levels of compulsivity, and may, therefore, represent the portion of the continuum that links OCSD, substance users, and disordered gamblers (Cavedini et al., 2002).

Another study by Goudriaan, Oosterlaan, de Beurs, and van den Brink (2005) assessed decision-making in disordered gamblers, Tourette's syndrome patients, a substance abuse group, and normal controls. Using the Iowa Gambling Task, the Card Playing Task, and the Go/No-Go discrimination task, they found that the disordered gamblers and alcohol dependent groups performed worse than normal controls on the IGT and Card Playing Tasks. These groups also performed worse than the Tourette's group on the IGT, suggesting that the cognitive deficits associated with disordered gambling and substance use are distinct from the impulse control deficits found in Tourette's patients. Only the disordered gamblers tended to respond faster and remain on disadvantageous decks after a larger loss than normal controls. Disordered gamblers may exhibit unique patterns of cognitive inflexibility and perseveration with regard to punishment and reward, a finding that might help explain why they frequently chase bets without regard to further consequences (Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2005). Taken together, these results suggest that similar cognitive deficits exist with disordered gambling and alcohol dependence that are not demonstrated in other impulse control disorders.

The Addiction Model Prevails

The results from the above studies painted a picture of disordered gambling that looked much closer to an addictions-based model rather than one characterized by obsessive-compulsive behavior. That being said, individual clients will vary tremendously, especially in the sub-clinical range, with regard to their presentation. Further, the wise clinician will always keep in mind that addictions reflect not only an individual's propensity to become addicted, but also the schedule of reinforcement. As such, a gambling addiction should be conceptualized as a highly interactive engagement between an individual (and all of his or her strengths and shortcomings) and the reinforcement contingencies within which gambling occurs.

Looking ahead

This chapter has provided an overview of the major nosological and conceptual challenges when considering disordered gambling. Subsequent chapters in this volume will expand on the foundation presented here. Technological advances in neuroscience have shed light on the brain mechanisms involved in disordered gambling and their relationship to rewards and schedules of reinforcement. Exciting developments within the field are certain to occur in the next few years as a result. These advances have already served to clarify whether disordered gambling is more appropriately conceptualized as an impulse control disorder or an addictive disorder with the latter conceptualization receiving the greater degree of support from an array of published reports.

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