CHAPTER 22

Gambling and Impulse Disorders

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DESCRIPTION OF THE DISORDER AND CLINICAL PICTURE

The *DSM-IV-TR* (American Psychiatric Association [APA], 2000) category of *impulse control disorders not elsewhere classified* includes five disorders characterized by recurrent behaviors that result in harm either to the self or to others: *pathological gambling, trichotillomania, kleptomania, intermittent explosive disorder,* and *pyromania.* Impaired control is defined by the recurrent failure to resist an urge to carry out a behavior that is, on the one hand, appetitive in that it provides positive or negative reinforcement but, on the other, is associated with severe deleterious consequences in personal, social, familial, employment, and/or legal functioning. Clinically, individuals describe an increasing tension or arousal prior to the commission of an act, a sense of gratification, and/or tension reduction on its completion, followed by an immediate sense of guilt or remorse.

Pathological Gambling

Gambling involves risking an item of value (usually monetary) on the outcome of a chance event. Primary forms of gambling include wagering (betting on horses), lotteries, electronic gaming machines (slot machines), and sports betting.

Epidemiological studies indicate that most individuals gamble responsibly within financial limits. The terms *pathological, problem, compulsive,* or *disordered gambling* are all used to describe the minority of individuals who exhibit impaired control over their behavior as evidenced by the presence of persistent and recurrent maladaptive gambling behaviors that disrupts personal, social, family, or vocational pursuits (APA, 2000, p. 671). To meet a formal psychiatric diagnosis, individuals must endorse 5 of 10 symptoms of disorder: (1) a *preoccupation* with gambling; (2) need to gamble increasing amounts to achieve desired excitement (*tolerance*); (3) repeated, unsuccessful efforts to reduce or cease gambling (*loss of control*); (4) restlessness or irritability attempting to reduce or cease (*withdrawal*); (5) gambling to escape problems or relieve dysphoric mood (*escape*); (6) continued gambling to recoup losses (*chasing*); (7) lying to conceal involvement in gambling; (8) commission of illegal acts to finance gambling; (9) jeopardizing significant relationships, employment, or educational opportunities; and/or (10) relying on money from others to relieve desperate financial circumstances caused by gambling (*bailouts*). The behavior cannot be accounted for by a manic episode.

The evolution of the *DSM* criteria highlights the paradoxical nature of the disorder, which is typically compared to substance abuse but otherwise classified with other nonsubstance use disorders that are characterized primarily by impulse dyscontrol. Unlike the other disorders "not elsewhere classified," which require endorsement of all criteria, pathological gamblers are required to endorse only 5 of 10 symptoms. Those symptoms are unweighted, though studies have shown that only some of the indicators are able to differentiate pathological gamblers from those with gambling problems (e.g., betting increasing amounts of money, lying about gambling; Stinchfield, 2003). As a result, an individual may endorse five symptoms that do not, individually, indicate pathology yet receive the same diagnosis as another gambler who endorses the most severely predictive symptoms. Accordingly, Stinchfield (2003) has cautioned that clinicians should be careful in classifying individuals who endorse three or four symptoms because tests of reliability, validity, and classification accuracy of the *DSM-IV* criteria indicate there is only a 50/50 probability that those individuals may require treatment. The clinical picture is further confounded by the fact that gamblers may meet diagnostic criteria at one point and not another, gambling in "binges" or fluctuating between subclinical and clinical levels of disorder (Nower & Blaszczynski, 2003).

Clinical studies suggest that 90 percent of pathological gamblers in treatment commence gambling prior to age 20 (mean age around 12 to 15 years), maintain control for 1 to 10 years, before losing control and gambling excessively

for a similar length of time prior to entering treatment at an average age of 35 to 39 (Blaszczynski & McConaghy, 1986; Petry, 2005). Rapidity of onset of pathological gambling varies extensively, however females reported shorter periods than males of intense (1.0 years versus 4.6 years) and problem gambling (1.8 years versus 8.6 years; Tavares, Zilberman, Beites, & Gentil, 2001). Electronic gaming devices account for a disproportionate percentage of problem gamblers, particularly among females. Although life events are considered important precipitants, factors that contribute to the transition from controlled to pathological gambling remain poorly understood.

The gender distribution for pathological gamblers seeking treatment is 60 percent males and 40 percent females (Petry, 2005). Females show a tendency to gamble predominantly as a means of escaping emotional distress in contrast to males who are motivated for factors related to winning, excitement, and chasing losses (Custer & Milt, 1985; Mark & Lesieur, 1992).

Trichotillomania

Trichotillomania or repetitive hair pulling, first identified by the French dermatologist Hallopeau, is a chronic, neglected psychiatric disorder, characterized by the failure to resist impulses to pull out one's hair, resulting in noticeable hair loss. A diagnosis of trichotillomania requires: (1) recurrent, episodic hair pulling resulting in noticeable hair loss; (2) increased tension immediately prior to hair pulling or when trying to resist hair pulling; (3) pleasure, gratification, or relief when hair pulling; (4) the absence of another mental health disorder or medical condition to better account for the behavior; and (5) clinically significant distress or impairment in social, occupational, or other important areas of functioning (APA, 2000). Though classified as an impulse control disorder, some have argued that trichotillomania would be better classified as an affective (Christenson, MacKenzie, & Mitchell, 1991) or obsessive-compulsive spectrum disorder (Swedo & Leonard, 1992).

Compared to earlier versions of the diagnostic criteria, *DSM-IV* added the requirement that hair pulling result in clinically significant distress (Criterion E) and included "pleasure" (Criterion C) as an emotion that accompanies the act of hair pulling.

Despite a high degree of individual variation in those with the disorder, there are some phenomenological consistencies among clients (for a review, see Diefenbach, Reitman, & Williamson, 2000). Hair pulling occurs at sites throughout the body, though the most common sites are the scalp, followed by lashes, brows, and pubic hair (Christenson, Mackenzie, et al., 1991; Schlosser, Black, Blum, & Goldstein, 1994). Episodes, lasting a few minutes to a few hours, are often prompted by negative affective states and sedentary activities like reading, watching television, driving, or talking on the phone (Christenson, MacKenzie, et al., 1991; Schlosser et al., 1994). In addition, the behavior can be automatic or result from focused intention (Christenson, MacKenzie, et al., 1991). Du Toit, van Kradenburg, Niehaus, and Stein (2001) have suggested that hair pulling varies with clinical subtypes, characterized by the presence or absence of automatic/focused hair pulling, comorbid self-injurious habits, and/or oral habits. In addition, comorbid obsessive-compulsive disorder and negative versus positive affective cues may reflect greater severity in symptomatology. Following hair pulling behavior, about 48 percent of individuals with trichotillomania will perform oral behaviors such as chewing or eating the hair, which can lead to dental erosion and medical complications (Christenson, MacKenzie, et al., 1991; Diefenbach et al., 2000).

Kleptomania

Kleptomania (Greek for "stealing madness") is characterized by (1) a recurrent failure to resist impulses to steal objects that are not needed for personal use or their monetary value. Individuals with the disorder experience (2) an increasing sense of tension immediately before the theft and (3) pleasure, gratification, or relief during the theft. In addition, (4) the stealing is not merely an expression of anger or vengeance or a response to a delusion or hallucination and (5) is not better accounted for by conduct disorder, a manic episode, or antisocial personality disorder (APA, 2000).

Initially introduced in the first edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders,* kleptomania was omitted from the second version only to return in 1980 in *DSM-III* and its subsequent versions. Revisions of the criteria in *DSM-IV* in 1994 added "gratification" to the sense of pleasure and relief that accompanies stealing and added as exclusionary criteria theft that occurs in response to a delusion, hallucination, or manic episode. However, despite its long tenure among psychiatric disorders, kleptomania is a poorly understood and underdiagnosed disorder. Because it often presents in comorbidity with other psychiatric disorders, it is conceptually unclear whether the disorder presents in clusters with different symptomalogical expressions or, rather, whether it is merely a nonspecific symptom of alternate, underlying, primary psychopathology (Presta et al., 2002).

Intermittent Explosive Disorder

Individuals with intermittent explosive disorder characteristically report (1) several discrete episodes of the failure to resist aggressive impulses that result in serious assaultive acts (e.g., physical assaults, verbal threats) or property destruction (e.g., purposefully breaking an object of value; APA, 2000). In addition, (2) the level of aggressiveness during episodes must be grossly out of proportion to any precipitating psychosocial stressors and (3) is not better accounted for by other mental disorders (e.g., antisocial or borderline personality disorder, psychotic disorder, and bipolar disorder). The current criteria eliminated the requirement in *DSM-III-R* of an absence of generalized impulsiveness or aggressiveness between episodes.

The current diagnosis is primarily one of exclusion, as aggressive or explosive behavior is also symptomatic of psychotic, conduct, and personality disorders, as well as a variant of panic disorder and a possible feature of depression (Opdyke & Rothbaum, 1998). *DSM-IV-TR* (APA, 2000) cautions that the diagnosis should be made only if other diagnostic possibilities are ruled out. Accordingly, there is little consensus as to whether the disorder should be an independent diagnostic category related to mood disorders or a nonspecific group of symptoms that often presents in a wide range of psychiatric and medical conditions (McElroy, 1999; McElroy, Soutullo, Beckman, Taylor, & Keck, 1998). In addition, the current criteria have been criticized for lacking an emphasis on the concept of an irresistible impulse and the highly ego-dystonic and largely uncontrollable nature of the range of outbursts (McElroy, 1999).

Though reliable data are lacking, onset for the disorder, between childhood and early 20s, is typically abrupt and absent a prodromal period; it is most prevalent in children who exhibit temper tantrums, impaired attention, hyperactivity, and other behavioral difficulties like stealing and fire setting (APA, 2000). Individuals with narcissistic, obsessive, paranoid, or schizoid traits are the most likely to exhibit episodic explosive outbursts (APA, 2000). In addition, the course of the disorder varies from chronic to episodic. First-degree relatives of individuals with intermittent explosive disorder are more likely than others in the general population to suffer from mood, substance use, intermittent explosive, and other impulse control disorders (APA, 2000).

Prior to aggressive outbursts, individuals with the disorder report experiencing physiological symptoms such as tingling, tremor, palpitations, chest tightness, head pressure, or hearing an echo (APA, 2000). They often feel (1) intense urges toward aggression prior to initiating the act; (2) irritability or rage, increased energy, and/or racing thoughts during the acts; and (3) depressed mood and fatigue when the aggressive acts are completed (APA, 2000; McElroy et al., 1998).

Neurological symptoms might include nonspecific EEG findings or evidence of abnormalities on neuropsychological testing, such as difficulty with letter reversal (APA, 2000). In addition, altered serotonin metabolism (e.g., low mean 5-hydroxindoleacetic [5-HIAA]) has been noted in the cerebrospinal fluid of some aggressive individuals, but studies have yet to clarify the nature of the relationship of these findings to intermittent explosive disorder (APA, 2000).

Pyromania

Pyromania is characterized by (1) deliberate and purposeful fire setting, preceded by a feeling of (2) tension or affective arousal and accompanied by (3) a fascination with, interest in, curiosity about, or attraction to fire and its situational contexts. Individuals with pyromania report (4) pleasure or gratification when setting fires or when witnessing or participating in their aftermath. Fire setting cannot be undertaken (5) for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one's living circumstances, in response to a delusion or hallucination, or as a result of impaired judgment. Nor may the fire setting be (6) better accounted for by conduct disorder, a manic episode, or antisocial personality disorder, an exclusionary criterion added in *DSM-IV* (APA, 1994).

Historically, the term *pyromania* preceded its classification as an impulse control disorder. Lewis and Yarnell (1951) were the first to explore the disorder using psychiatric and police records of 1,300 arsonists and enumerated five classification types: accidental or unintentional, delusional, erotically motivated, revenge-motivated, and children who light fires. Under the current diagnostic criteria, only the "erotically motivated" offenders would qualify for diagnosis if they met other criteria for the disorder.

Accordingly, pyromania is particularly difficult to diagnose because of the numerous exclusionary symptoms such as alcohol abuse, retaliatory motives, or delusional thinking that typically accompany fire-setting behavior. Accordingly, the majority of research into fire setting or arson either fails to differentiate between offenders who meet the diagnostic criteria for pyromania or applies the label colloquially to individuals who would not meet criteria in a clinical setting. The latter may be due, in part, to the fact that the diagnostic label is applied infrequently by psychiatrists but commonly by law enforcement officers who actually have a poor understanding of the parameters of the disorder. Laubichler and Kuhberger (1997) have also theorized that diagnostic criteria

are unnecessarily limited by the exclusion of single fire settings, comorbid alcohol usage, and aggressive motives, which often characterize adolescent fire setters who act out of anger against peers or family members.

PERSONALITY DEVELOPMENT AND PSYCHOPATHOLOGY

Pathological Gambling

A number of scientific studies have reported that a significant percentage of pathological gamblers demonstrate evidence of mood or personality disorders (Blaszczynski & McConaghy, 1989; Vitaro, Arseneault & Tremblay, 1999), neurobiological dysfunction (Potenza & Winters, 2003), and/or genetic abnormalities (Comings, Rosenthal, Lesieur, & Rugle, 1996) that predispose them to pleasure seeking. They may also exhibit high levels of impulsivity (Vitaro, Arseneault, & Tremblay, 1997), sensation seeking (Powell, Hardoon, Derevensky, & Gupta, 1999), substance use (Ladouceur, Boudreault, Jacques, & Vitaro, 1999; Stinchfield, Cassuto, Winters, & Latimer, 1997), compulsive eating and/or working (Shaffer, LaBrie, LaPlante, Kidman & Korn, 2002), sexual risk taking (Petry, 2000), and attention deficit (Rugle & Melamed, 1993). In a majority of individuals, these disorders may preexist problem gambling behavior; however, in other cases they may result from stress caused by problem gambling (for a discussion of pathways leading to problem gambling, see Blaszczynski & Nower, 2002).

Developmentally, it is theorized that a large proportion of individuals who become problem gamblers have experienced deprivation or abuse in childhood and premorbid substance abuse and symptoms of depression or anxiety (Gupta & Derevensky, 1998a, 1998b; Jacobs, 1986; Nower & Blaszczynski, 2003). In addition, they often score higher than their nonproblem gambling counterparts on measures of impulsivity and sensation seeking, report parents who gamble problematically or abuse substances, and, particularly among males, use gambling as an avoidant stress coping strategy (Nower, Derevensky, & Gupta, 2004).

A substantial percentage of disordered gamblers have comorbid mood disorders, including depression (Becona, Del Carmen Lorenzo, & Fuentes, 1996), bipolar disorder (McCormick, Russo, Ramirez, & Taber, 1984), anxiety (Black & Moyer, 1998), and severe insomnia (Bergh & Kuehlhorn, 1994). In addition, several studies have suggested that gamblers are particularly vulnerable to suicide because of high levels of comorbid depression coupled with the severe financial, legal, and psychosocial consequences of problem gambling (Bergh & Kuehlhorn, 1994; MacCallum & Blaszczynski, 2002; Nower, Gupta, Blaszczysnki, & Derevensky, 2004). Reported rates of suicidality in these studies vary widely from 17 percent to 80 percent for suicidal ideation and 4 percent to 24 percent for reported suicide attempts, depending on the population sampled and methodology employed. However, it is important to note that studies have failed to clarify whether affective symptoms, officially diagnosed or not, predated the onset of problematic gambling behavior.

In addition to comorbid affective disorders, up to 50 percent of disordered gamblers also report a substance use disorder (MacCallum & Blaszczynski, 2002). Petry (2001b) has suggested that pathological gambling and substance abuse have an additive effect, manifesting in a preference for immediate gains with higher levels of punishment and overall net losses than either substance abusers alone or subjects with no addictions.

Trichotillomania

Though no single diagnosis is consistently associated with the disorder, a number of studies have found higher rates of personality, affective, and eating disorders in individuals with trichotillomania as compared to the general population (Christenson, MacKenzie, et al., 1991; Diefenbach et al., 2000; Schlosser et al., 1994; Swedo & Leonard, 1992). For example, prevalence estimates suggest 25 percent to 55 percent of clients with trichotillomania also have a personality disorder such as histrionic, borderline, or obsessive-compulsive personality disorder (Christenson, Chernoff-Clementz, & Clementz, 1992; Swedo & Leonard, 1992). Similarly, Christenson, Pyle, and Mitchell (1991) noted a 20 percent lifetime prevalence rate for eating disorders and an 82 percent lifetime prevalence of Axis I disorders.

Kleptomania

Though kleptomania has long been recognized as a disorder, there is relatively little research into its nature and course. Most of the published reports in the area are either derived from case studies and/or family histories of a small number of individuals in treatment or provide merely demographic information about the client population. For example, Sarasalo, Bergman, and Toth (1996) sought to explore personality characteristics, psychiatric disorders, and somatic illness among 37 individuals with kleptomania recruited through newspaper ads. As a whole, the participants scored extremely low on measures of socialization. More than half of the sample reported receiving treatment for another psychiatric disorder, most commonly depressive, anxiety, and sleep disorders, and

had a family history of psychiatric illness. Nearly half of the sample reported a history of chronic somatic diseases, including asthma/allergy, lower back and neck pain, hearing deficiency, heart disease, Crohn's disease, and other neurological disorders. In addition one-third of subjects reported either an alcohol problem or a need to cut back on alcohol consumption.

Another study compared individuals with kleptomania (n = 11), alcohol abuse/dependence (n = 60), and psychiatric disorders other than impulse control or substance-related disorders (n = 29) on various psychopathological dimensions (Bayle, Caci, Millet, Richa, & Olie, 2003). Findings indicated that those with kleptomania reported significantly higher rates of impulsivity and sensation seeking, comorbid psychiatric disorders (particularly mood disorders), other impulse-control disorders, and substance abuse or dependence (mainly nicotine) as compared to other groups.

Other studies have also found a high prevalence of both unipolar and bipolar mood disorders and obsessive-compulsive disorder in individuals with kleptomania (Presta et al., 2002), suggesting possible inclusion of the disorder with so-called obsessive-compulsive spectrum disorders (OCSD). Presta and colleagues (2002) noted that about 60 percent of the 20 participants met diagnostic criteria for bipolar I or II disorder, however the participants reported feeling distinct subjective differences between kleptomanic impulses and abnormal behaviors occurring exclusively in the course of mood polarity. In addition, unlike individuals with obsessive-compulsive disorder, participants in the study reported a variable degree of resistance to the stealing behavior, despite the pleasurable deriving action (Presta et al., 2002).

Limited evidence from studies with small, nonrepresentative samples exists to suggest a familial link between kleptomania and other psychiatric disorders, including mood disorders, obsessive-compulsive disorder, and substance use disorders. Presta et al. (2002) found that 35 percent of first-degree relatives of individuals diagnosed with kleptomania suffered from a mood disorder, 25 percent had obsessive-compulsive disorder, and 15 percent reported a substance use disorder. McElroy, Pope, Hudson, Keck, and White (1991) likewise reported a significant prevalence of mood, anxiety, and eating disorders in first-degree relatives of participants with kleptomania. A third study, which compared self-reported individual and familial psychiatric histories of 31 clients with "stealing behavior" with unmatched controls, failed to find any relationship between mood disorders and stealing, though subjects were not required to meet diagnostic criteria for kleptomania (Grant, 2003). The study did, however, note a correlation between stealing and both comorbid impulse control disorders and the presence of a first-degree relative with alcoholism and/or another psychiatric disorder.

Intermittent Explosive Disorder

Little is known about the developmental history of individuals with intermittent explosive disorder. In one study, childhood histories were notable for hyperactivity, impaired attention, attention-deficit/hyperactivity disorder, problematic temper tantrums, stealing, and fire setting (McElroy et al., 1998). The study also found that 96 percent (n = 26) of individuals surveyed met criteria for one or more and 70 percent (n = 19) for three or more comorbid Axis I disorders, most commonly a mood disorder (e.g., bipolar disorder), anxiety disorders (panic disorder, post-traumatic stress disorder, phobias, and obsessive-compulsive disorder), substance use disorder (alcohol abuse), eating disorders (binge eating), and/or other impulse-control disorders (McElroy et al., 1998).

Pyromania

Most of the research in the area focuses on individuals charged with arson or children and adolescent fire setters who may or may not meet criteria for pyromania. The *DSM-IV-TR* (APA, 2000) indicates that more than 40 percent of those arrested for arson-related offences are younger than 18, but most of those offenders do not suffer from pyromania; rather, the fire setting is associated with conduct disorder, attention-deficit/hyperactivity disorder, or adjustment disorder. Impulsive fire setters (with or without pyromania) often have a current or past history of alcohol dependence or abuse (APA, 2000). In addition, authors have noted that fire setting often bears a complex and complementary relationship with sexuality, which may serve as an additional motive for the behavior (Fras, 1997). Lejoyeux, Arbaretaz, McClaughlin, & Ades (2002) also found a significant history of depression among clients with pyromania as compared to subjects with other impulse control disorders.

Developmentally, research in the area of adolescent fire setting (without regard to pyromania) has reported a high correlation between fire setting and shyness and aggression as a reaction to perceived rejection by peers (Chen, Arria, & Anthony, 2003); extreme drug use and antisocial and suicidal behavior (Martin, Bergen, Richardson, Roeger, & Allison, 2004); and depression, alienation and poor reality testing (Moore, Thompson-Pope, & Whited, 1996). Most of these findings would preclude diagnosis for pyromania.

EPIDEMIOLOGY

Pathological Gambling

A variety of studies internationally have attempted to estimate the lifetime prevalence rate for adult pathological gambling, though it should be noted that findings often vary widely depending on location, survey methods, classification schemes, and ease of accessibility to gambling over time. A meta-analysis of 120 prevalence studies in the United States and Canada found that an average of 1.6 percent of adults met criteria for pathological and 5.5 percent for problem gambling disorder (Shaffer, Hall, & Vander Bilt, 1997). For other countries, reported rates vary between 1.2 percent for Australia, 1.7 to 3.2 percent for Spain, 0.8 percent for Switzerland, 1.8 percent for Hong Kong, 0.6 percent for Norway, and 2.7 percent for New Zealand. Rates of subthreshold problem gambling are generally double those for pathological gambling; however, there is significant conceptual uncertainty regarding the actual status of gamblers "in transition," who may be moving toward or away from pathology (Shaffer & Hall, 1996) or "binge gamblers" who may meet clinical criteria at some times but not at others (Nower & Blaszczysnki, 2003).

Rates of disordered gambling may also vary by geographical location and by socioeconomic status. Volberg (1996) has noted that U.S. states with a long history of legalized gambling reported higher rates of pathological gambling with a propensity for more pathological gambling to come from lower socioeconomically disadvantaged groups.

Adolescents characteristically report higher rates of both problem and pathological gambling than adults, due in large part to phases of development that correlate with heightened risk taking and disinhibition. Studies have reported that 24 percent to 40 percent of adolescents gamble weekly, 10 percent to 14 percent are at risk for gambling problems, and 2 percent to 9 percent meet diagnostic criteria for pathological gambling (for reviews of youth gambling, see Hardoon & Derevensky, 2002; Shaffer & Hall, 1996). The mean prevalence rate for adolescent pathological gambling is estimated at 5 percent—three times the 1.5 percent average for adults (National Research Council, 1999).

Trichotillomania

There have been no epidemiological studies to determine the prevalence of trichotillomania in the general population. One study of more than 2,500 college students reported that 1.5 percent of males and 3.4 percent of females reported chronic hair pulling though only 0.6 percent of either gender met diagnostic criteria for disorder (Christenson, Pyle, et al., 1991). Another study found that 10 percent to 13 percent of college students surveyed in the United States reported chronic hair pulling, though only 1 percent resulted in clinically significant hair loss and/or distress (Diefenbach et al., 2000).

The majority of diagnosed hair pullers are female, though studies suggest males also suffer from the disorder and present with similar phenomenological features though they may be less likely to seek treatment (Christenson, MacKenzie, & Mitchell, 1994; Christenson, Pyle, et al., 1991; Swedo et al., 1989).

There have been no longitudinal studies to detail the progression of trichotillomania over time. However, it is clinically accepted that symptoms may manifest in benign form in early childhood then remit with little or no therapeutic intervention (Swedo & Rapoport, 1991) or present in adolescence around the age of 13 years in a late-onset form that is more resistant to treatment and associated with comorbid psychopathology (Christenson, Pyle, et al., 1991; Diefenbach et al., 2000; Swedo & Leonard, 1992).

Kleptomania

Kleptomania is rarely diagnosed, occurring in fewer than 5 percent of identified shoplifters (APA, 2000). The overall prevalence rate in the general population is unknown, though evidence from clinical samples suggests that two-thirds of individuals with the disorder are female.

Intermittent Explosive Disorder

In the absence of prevalence studies, *DSM-IV-TR* (APA, 2000) notes that the disorder is "rare" and more common in males as compared to females. The disorder needs to be adequately differentiated from aggression linked to other causes such as an organic brain damage, drug-induced aggression, other psychiatric disorder or in response to deliberate provocation by others.

Pyromania

Little is known about the actual prevalence of pyromania except that it occurs more frequently in males and is "rare" (APA, 2000). In one unconventional study designed to ascertain the prevalence of the disorder in a small number of arsonists, a group of Canadian psychiatrists evaluated 236 arson cases and determined that only 2.9 percent ($n = 7$) met the clinical criteria (Crossley & Guzman, 1985). In another study, Ritchie and Huff (1999) examined the mental health records and/or prison files of 283 arsonists and found that, whereas most had psychiatric histories, only three offenders

were diagnosed with pyromania. The authors indicated that the majority of fire setters were either angry or delusional, two criteria that would exclude them from a diagnosis of pyromania. Of note, 36 percent of the sample had been diagnosed with schizophrenia or bipolar disorder and 64 percent were abusing alcohol at the time of the fire setting.

ETIOLOGY

Pathological Gambling

It is generally agreed that the etiology of gambling disorder is multifactorial, dependent on a variety of biopsychosocial and environmental risk factors. Blaszczynski and Nower (2002) have proposed that all gamblers are initiated into gambling as a result of common ecological factors such as accessibility, availability, and accessibility of gambling. In addition, subsequent exposure to a variable ratio reinforcement schedule and other cues in the gambling environment provide behavioral conditioning needed to habituate and maintain the behavior. However, despite these commonalities, the pathways that lead certain individuals to progress toward the disorder while protecting others depends on a variety of factors including age of onset, childhood experiences, familial exposure to addictive behaviors, sensation-seeking and impulsivity traits, gender, ethnicity and socioeconomic status, age, and biobehavioral abnormalities (Blaszczynski & Nower, 2002).

Early onset and familial participation in gambling behavior are significant risk factors for future problems in youth. A number of studies have found that adolescents with gambling problems began gambling before the age of 11 (Griffiths, 1990; Gupta & Derevensky, 1998a). In addition, a significant percentage of youth problem gamblers report they first gambled with parents or other relatives or had parents with gambling problems (Gupta & Derevensky, 1998b). A recent meta-analysis of 17 family and 2 twin studies found a stronger familial effect for sons of problem gambling fathers and daughters of problem gambling mothers—strongest for high severity problem gambling in males (Walters, 2001).

Problem gambling behavior is also common in individuals with high levels of intensity seeking and impulsivity. Because gambling involves a high degree of mental and sensory stimulation, it is not surprising that a majority of studies have noted a strong empirical association in both youth and adults between problem gambling and the pursuit of intense stimulation, one form of sensation seeking (Anderson & Brown, 1984; Coventry & Brown, 1993; Kuley & Jacobs, 1988; Nower et al., 2004). Findings have been mixed (see, Blaszczysnki, Wilson, & McConaghy, 1986; Dickerson, Hinchy, & Fabre, 1987) due in large part to sampling bias and other methodological inconsistencies.

Severe gambling problems have also been noted in highly impulsive individuals (Nower et al., 2004; Petry, 2001a, 2001b; Steel & Blaszczynski, 1996; Vitaro, Brendgen, Ladouceur, & Tremblay, 2001). With few exceptions (Allcock & Grace, 1988; Petry, 2000), studies with both adult and youth gamblers have consistently noted a positive relationship between problem gambling and high levels of impulsivity, particularly the subtype associated with an inability to act with forethought and deliberation.

Male gender has long been a strong predictor of gambling problems. The majority of early studies on pathological gambling focused on men from Gamblers Anonymous and the Veterans Administration hospital system, limiting the number of women sampled (Mark & Lesieur, 1992). But recent surveys have noted that an increasing number of women are developing gambling problems due in part to a preference for gaming machines that prolong play while offering a highly addictive reinforcement schedule and low rate of monetary return. Recent studies comparing male and female gamblers have reported that females prefer lower denomination slot machines and longer sessions of play (Hing & Breen, 2001); a later age of onset (mean age 34.2 years versus 20.4 years) and shorter periods of intense (1.0 years versus 4.6 years) and problem gambling (1.8 years versus 8.6 years; Tavares, Zilberman, Beites, & Gentil, 2001); fewer problems with drugs or gambling-related arrests (Potenza et al., 2001); and higher rates of affective disorders and histories of physical abuse (Ibanez, Blanco, de Castro, Fernandez-Piqueras, & Saiz-Ruiz, 2003). A majority of those studies found similar rates of gambling severity, overall psychiatric comorbidity, and indebtedness in both males and females.

Though research is limited, it appears that gambling problems may be more prevalent in ethnic cultures that sanction the behavior. Zitzow (1996) has reported that American Indian adolescents, who also report higher rates of substance abuse than their non-Indian peers, also endorsed an earlier onset of gambling problems. Similarly, Blaszczynski, Huynh, Dumlao, and Farrell (1998) found that 2.9 percent of individuals in the Chinese community in Sydney were pathological gamblers. Among African Americans in the United States, Welte, Barnes, Wieczorek, Tidwell, and Parker (2002) noted lower overall past-year rates of gambling participation, however, those who did gamble bet more frequently and reported larger wins and losses than other groups. Likewise, another study reported that nearly 17 percent of 80 elderly African Americans living in senior centers with bus trips to

casinos were "heavy to pathological" gamblers (Bazargan, Bazargan, & Akanda, 2001).

Increasingly, researchers are concerned that advanced age is an additional risk factor for problem gambling, particularly when individuals live in residential facilities that provide transportation to gambling venues. As indicated in the Bazargan et al. (2001) study, elderly African American gamblers surveyed reported a prevalence rate of pathological gambling about 15 percent higher than that of the adult population in general. McNeilly and Burke (2000) have also reported that 24 percent of seniors who actively gambled frequented casinos, 17 percent played the numbers, and 41 percent played bingo once a week or more.

Finally, an increasing number of studies have indicated abnormalities in biobehavioral functioning and genetic mechanisms that may contribute to the development or maintenance of gambling disorder (for a comprehensive review, see Goudriaan, Oosterlaan, de Beurs, & van den Brink, 2004). Neuropsychological studies indicate deficits in executive functioning, such as delay discounting, fluency and interference control, and impulsiveness/disinhibition. Neuroimaging studies have identified abnormalities in braining functioning, including decreased hemispheric lateralization and/or temporal lobe function, and abnormal activation in specific subcortical frontal regions. Research into neurochemistry has likewise identified deficits in neurotransmitter mechanisms in pathological gamblers, particularly dopamine, serotonin and norepinephrine in reward-pathway areas. Finally, genetic studies have identified allele variants of dopamine receptors (DRD2, DRD3, DRD4), deficits in the serotonin transporter (5-HTTLPR) and monoamine oxidase A (MAO-A) genes, and shared genetic factors that suggest a heritable vulnerability to factors that contribute to the development of pathological gambling disorder.

Trichotillomania

A variety of approaches have attempted to explain the etiology of trichotillomania: psychoanalytic, biological, and behavioral (see Diefenbach et al., 2000). However, to date, none is comprehensive and supported by a large body of empirical evidence. Psychoanalytic theorists suggested that the disorder is a symbolic expression of unconscious conflicts, or the result of childhood trauma or poor object relationships (Singh & Maguire, 1989). In contrast, biological theories maintain that trichotillomania is an obsessive-compulsive spectrum disorder (OCSD), characterized by intrusive and obsessive thoughts and/or uncontrollable, repetitive behaviors (Hollander, Skodol, & Oldham, 1996; Swedo & Leonard, 1992). Studies to test this association have been largely unsuccessful, reporting instead significant phenomenological, neuropsychological, and neurological differences between the two disorders (see for review, Diefenbach et al., 2000). Some recent studies have noted a possible overlap with Tourette's syndrome, also an OCSD (Diefenbach et al., 2000). Both disorders are characterized by repetitive behaviors prompted by sensory cues and urges, share structurally similar morphometric findings, and respond well to neuroleptic drugs, and the presence of comorbid Tourette's syndrome appears to amplify hair-pulling behavior (Diefenbach et al., 2000; Stein & Hollander, 1992).

Finally, several potentially complementary theories have attempted to explain the etiology and maintenance of trichotillomania from a behavioral perspective. One theory proposes that hair-pulling behavior is initiated in response to stress, reinforced by its capacity for tension reduction, and, ultimately, maintained by association with environmental cues by virtue of operant conditioning (Diefenbach et al., 2000). A second theory maintains that hair pulling results from modeling, as children imitate the behavior of their caregivers (Christenson, MacKenzie, & Reeve, 1992; Diefenbach et al., 2000). Other models assert an integrative approach, suggesting that a combination of biological, cognitive, affective, behavioral, and environmental factors serve to initiate and maintain pathological hair-pulling behavior over time (Diefenbach et al., 2000).

Kleptomania

Historically, kleptomania was initially viewed as a "feminine sickness," resulting from hysteria, insanity, menstruation, and pelvic and uterine diseases (for a review of early theories on etiology, see Murray, 1992) These theories were later replaced by psychoanalytic interpretations, which viewed the act of stealing as an ego defense against anxiety and a manifestation of sexuality in which a female, out of desire to obtain a penis, stole objects that represented the symbolic byproducts of anal fixation.

Subsequent conceptualizations of the disorder have viewed kleptomania as an adaptive response to underlying depression in which an individual adopts the stealing behavior in response to aversive mood (Presta et al., 2002). During these periods, individuals experience a sense of elation concurrently with the kleptomanic urge, relieving the ego-dystonic nature of the behavior. The behavior is often followed by feelings of depression, guilt, and fatigue, though individuals report that, overall, the act of stealing has a therapeutic effect on the severity of depressive symptoms (Presta et al., 2002). Most patients do not exhibit other antisocial behavior (Goldman, 1991).

Intermittent Explosive Disorder

Few studies have explored the etiology of individuals who meet criteria for intermittent explosive disorder apart from other psychiatric conditions with aggressiveness as an associated feature. Commonalities in one early study included a history of coma-producing conditions (e.g., meningitis, febrile convulsions, head injuries), a high incidence of family violence and alcoholism, aggressive eruptions with alcohol usage, and comorbid pyromania (Bach-y-rita, Lion, Climent, & Ervin, 1971). In addition, clients in that study reported chronic anxiety and insecurity, poor coping skills, and childhood deprivation (Bach-y-rita et al., 1971). Other researchers have asserted that PTSD symptoms, precipitated by early childhood exposure to violence, are subsequently triggered by situations that evoke feelings of being trapped, criticized or rejected. One study found that individuals with intermittent explosive disorder had at least one first-degree relative with a mood disorder (56 percent, n = 14), another impulse-control disorder (56 percent, n = 14), a substance use disorder (80 percent, n = 20), and an anxiety (8 percent, n = 2) or eating disorder (8 percent, n = 2; McElroy et al., 1998).

Mounting evidence suggests that the disorder may be related to mood disorders and a form of affective spectrum disorders (for summary, see McElroy et al., 1998). First, mood disorders are common in alcoholic violent offenders and impulsive fire setters, who also commonly meet criteria for intermittent explosive disorder. Second, studies have reported abnormalities in central serotonergic neurotransmission and circadian rhythm disturbances common to individuals with mood disorders. Third, individuals with intermittent explosive disorder have been found to respond to treatment with antidepressants, mood stabilizers, and antiepileptic drugs with mood stabilizing properties (McElroy et al., 1998).

Pyromania

The etiology of pyromania is unknown. Limited information from case histories suggests a possible familial tendency toward fire setting and frontal lobe dysfunction (Calev, 1995) and/or varying degrees of mental retardation (Geller, 1987). Adolescent fire setters, not necessarily diagnosed with pyromania, have also reported families characterized by absent fathers, parental drug and alcohol abuse, physical abuse, and neglect (Showers & Pickrell, 1987).

COURSE, COMPLICATIONS, AND PROGNOSIS

Pathological Gambling

Pathological gambling is commonly viewed as a spectrum disorder, progressing from social gambling for entertainment to pathological gambling that meets clinical criteria for disorder. The majority of problem gamblers report participation in adolescence with fluctuating degrees on involvement over adulthood. Problem gamblers who exhibit some gambling-related difficulties may be in transition, moving either toward or away from the serious end of the spectrum (Shaffer & Hall, 1996). Gambling disorder progresses through three or four stages (for discussions of gambling stages, see Custer & Milt, 1985; Rosenthal, 1992). During the *winning phase,* gamblers play for fun and excitement, however an early big win or other accomplishment fuels a desire to gamble more frequently for greater profit. Heightened preoccupation with gambling, increases in gambling frequency and mounting losses herald the *losing phase,* in which gamblers increase the frequency and amount of bets in a frantic attempt to win back lost funds. As losing continues, gamblers begin lying, borrowing, ignoring bills, begging for financial bailouts, and exploiting relationships to hide the extent of gambling losses. This behavior leads ultimately to the *desperation phase,* a period characterized by engaging in previously inconceivable behaviors like embezzlement, fraud, and stealing as a necessary means of financial and psychological survival. Some gamblers progress to the *hopelessness phase,* in which they abandon all hope of winning to gamble frantically for excitement alone (Rosenthal, 1992). As gambling increases in severity and frequency, pathological gamblers find themselves with an increasingly limited spiral of options (Lesieur, 1979), leading to serious adverse financial, legal, and psychosocial consequences.

The primary complications of pathological gambling are the development of major depressive symptoms including risk of suicidality and marital problems. The direction of causality is varied with some cases in which depression leads to gambling and vice versa.

Trichotillomania

Trichotillomania manifests itself in infancy, childhood, and early adolescence and runs a chronic and debilitating course of variable intensity through adulthood. As noted by Swedo (1993a), the onset of trichotillomania in early infancy, that is, prior to age 5 may constitute a separate group and represent a habit disorder that is benign, relatively self-limiting and remits by school age following maturation. The behavior occurs when the infant is tired or bored and increases during separation. Accordingly, it is viewed as a self-soothing behavior comparable to rocking or thumb sucking. In early as compared to later onset cases, there is a greater proportion of males; almost twice as many.

Males tend to have a slightly earlier onset in the prepubescent phase of maturation as compared to females who

display hair-pulling behaviors in early adolescents. Symptoms may be exacerbated during menstruation and under conditions of emotional stress taking on a focused and ritualistic form of repetitive behavior that is associated with tension reduction. In about three-quarters of sufferers, there does not appear to be any perceptible relationship between trichotillomania and stresses; the behavior appearing to be entirely habitual or automatic, and associated with sedentary conditions such as reading, watching television and use of computers. Individuals attempt to conceal the disorder from family and others, for example, through use of scarfs, hats, wigs, or concealing hairstyles or, if limited to eyebrows or lashes, may tolerate the social opprobrium that may emanate from peers or family members. Given the embarrassment, shame, and lowered self-esteem associated with the cosmetic effects of unseemly bald patches or thinning hair, patterns of social avoidance and isolation are common. Typically, the behavior persists into adulthood before presentation for treatment in the late 20s and early 30s.

Physical complications may arise from secondary behaviors associated with trichotillomania. Slightly fewer than a half of people with trichotillomania show a tendency to chew and/or swallow the hair (trichophagia). This behavior presents risk of dental erosion or serious medical complications caused by the development of hair balls (trichobezoar) in the stomach and intestines (Diefenbach et al., 2000).

With respect to comorbid disorders, there is no single psychiatric diagnosis or personality traits inherently linked to trichotillomania, although affective and anxiety disorders are commonly present, with 50 percent to 65 percent reporting mood disturbances, 23 percent current major depression, and 32 percent history of major depression. Approximately half experience lifetime anxiety and 18 percent current/past panic. A small percentage (10 percent) report a current obsessive-compulsive disorder, 5 percent a history of such, and 18 percent to 27 percent have obsessive-compulsive symptoms that do not meet criteria for the diagnosis of the disorder.

Addictive behaviors and eating and body dysmorphic disorders have been reported in 20 percent of cases in addition to a range of Axis II personality disorders including the dramatic Cluster B, histrionic and borderline, obsessive-compulsive, and passive-aggressive disorders (Diefenbach et al., 2000). The casual relationship between these disorders and trichotillomania remains open to speculation. Concurrent self-cutting/harm occurs in approximately 3 percent to 5 percent of cases.

Methodological problems and mixed results reported in treatment outcome studies preclude any definitive comment on prognosis (Diefenbach et al., 2000). However, pharmacological and behavioral treatments do lead to symptomatic improvement in the short term but there are difficulties maintaining gains in the long term with high rates of relapse occurring.

Kleptomania

There is little published information or systematic study on the course of the disorder. Presta et al. (2002) note this is due in large part to two factors: (1) the secretive nature of the disorder and associated shame that dissuades individuals from seeking treatment until they are arrested or in treatment for a comorbid psychiatric disorder and (2) the lack of sophisticated, widely used assessment instruments resulting in underdiagnosis and small sample sizes for research.

The *DSM* suggests the course is variable, beginning in childhood, adolescence, or adulthood and, in rare cases, in late adulthood and may continue for years despite multiple convictions for shoplifting (APA, 2000). The typical course is thought to follow one of three patterns: (1) sporadic with brief episodes and long periods of remission; (2) episodic with protracted periods of stealing and periods of remission; or (3) chronic, with some degree of fluctuation (APA, 2000). Limited information from empirical research suggests individuals with the disorder tend to be older (mean age 35 to 40) when sampled, evade diagnosis for years, and report a stealing history that exceeds 10 years in duration and an age of onset in late teens or early 20s (McElroy, Hudson, Pope, & Keck, 1991; Sarasalo et al., 1996).

Intermittent Explosive Disorder

The nature of the disorder suggests that aggressive behaviors are intermittent and, therefore, unpredictable, largely determined by eliciting events that trigger rageful internal states. This is further complicated by the fact that triggers for explosive behaviors are often undetectable by direct observation (Opdyke & Rothbaum, 1998). The course of an explosive episode begins with perceived threats, rejections or criticisms, which produce aversive stimulation in the form of internal unrest. This, in turn, precipitates a need for escape through erupting in a violent outburst, which serves to dissipate the pool of negative emotion (Opdyke & Rothbaum, 1998). Furthermore, the number of perceived threats, rejections, or criticisms may accrue, increasing the reoccurrence of outbursts or increasing the overall level of volatility.

Subjects in a study by McElroy et al. (1998) reported that their aggressive acts were very brief, lasting an average of 22 to 23 minutes. Most indicated that outbursts were precipitated by psychosocial stressors, typically conflicts with other people, though a majority of the subjects also reported they also had spontaneous aggressive outbursts. Aggressive impulses began with an urge to attack or defend themselves or,

simply, with an "adrenaline rush," were often accompanied by autonomic symptoms and associated with some degree of loss or change in awareness, and were followed by a sense of relief and/or pleasure (McElroy et al., 1998). Most of the individuals in the study indicated they also experienced difficulty with chronic anger and frequent subthreshold episodes, which they managed to either suppress or channel into less aggressive behaviors such as screaming or punching walls with no damage.

Pyromania

There are no longitudinal or other empirical studies investigating the course of the disorder or the relationship between fire setting in childhood and adulthood (APA, 2000). It is generally accepted that fire setting is episodic and may wax and wane in frequency (APA, 2000).

ASSESSMENT AND DIAGNOSIS

Pathological Gambling

The South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987) is the most widely utilized screening instrument in treatment and research with adults. Based on the *DSM-III-R* (APA, 1987), the SOGS categorizes individuals who endorse five or more items as pathological gamblers. Despite its popularity, the SOGS has been criticized for generating a high number of false positives in general population samples, diagnosing based on outdated criteria for pathological gambling, providing only lifetime estimates of pathological gambling behavior, and failing to discriminate adequately between subclinical and pathological gambling groups (see, e.g., Stinchfield, 2002).

Other adult screening instruments include the Canadian Problem Gambling Index (CPGI; Ferris & Wynne, 2001), a 31-item measure used for general population surveys; the two-item Lie/Bed Questionnaire (Johnston, O'Malley, & Backman, 1997), a two-item screen measuring two *DSM-IV*-based questions found particularly sensitive in identifying pathological gamblers; and the NODS (NORC, 1999), a *DSM-IV*-based measure used in U.S. prevalence studies. Adolescent studies typically administer the adolescent version of the SOGS, called the SOGS-RA (Winters, Stinchfield, & Fulkerson, 2003), or the *DSM-IV*-based measures by Fisher, *DSM-IV*-J (Fisher, 1992), or, more recently, the *DSM-IV*-MR-J (Fisher, 2000).

In addition to formal screening measures, 20 questions devised by members of Gamblers Anonymous, the GA 20 Questions, have long been utilized as an informal self-screening mechanism, and some clinicians merely adapt the 10 *DSM-IV-TR* criteria to question format. Despite the variety of screening tools, it is important to note that few treatment programs routinely screen for gambling disorder, and many treatment providers know little, if anything, about pathological gambling (Rowan & Galasso, 2000; Volberg, 2002). Shepherd (1996) suggested that some professionals may resist screening for gambling because they are reluctant to accept changes in protocol, have inadequate education and training to administer instruments properly, are unaware that gambling can serve as a relapse trigger for substance abuse, or perceive screening as "intrusive to the therapeutic agenda" (p. 27). Medical professions may also lack resources and training to encourage proper implementation of screening tools (Rowan & Galasso, 2000).

Trichotillomania

The diagnosis is typically established through clinical interview and scalp examination and biopsy to rule out organic factors. Accurate scalp examination and biopsy are critical, though the process is often complicated by patient and family denial or a lack of knowledge about the disorder in general (Walsh & McDougle, 2001). Characteristic scalp biopsies for the disorder feature trichomalacia, pigment clumps, peribulbar hemorrhage, and hair canal pigment casts; a lack of lymphocytic infiltrates is typically observed in individuals with alopecia areata (Walsh & McDougle, 2001). Methods of clinical assessment include: (1) clinical interview (e.g., Minnesota Trichotillomania Assessment Inventory-II [MTAI-II]; Christenson, Mackenzie, et al., 1991); (2) clinical rating scales (e.g., Yale-Brown Obsessive-Compulsive Scale modified for trichotillomania [Y-BOCS TM]; Stanley, Prather, Wagner, Davis, & Swann, 1993); Psychiatric Institute Trichotillomania Scale (PITS; Winchel, Jones, Molcho, Parsons, & Stanley, 1992); (3) self-report measures such as the Massachusetts General Hospital (MGH) Hair-Pulling Scale (Keuthen et al., 1995); (4) self-monitoring; and (5) collateral report.

Kleptomania

Diagnosis is typically made through self-report, according to *DSM-IV-TR* criteria. Care must be taken to differentiate the disorder from behaviors associated with an antisocial personality and where theft is motivated for personal economic gain or due to peer-group pressure or deviant subculture membership.

Intermittent Explosive Disorder

There is no empirical research on the assessment and diagnosis of intermittent explosive disorder as defined by the *DSM-IV-TR* (APA, 2000) criteria, though there are studies that examine episodic dyscontrol and explosive rage. In one study, McElroy (1999) recruited subjects with self-reported rage outbursts that resulted in serious assaultive acts or destruction of property. The participants were assessed using a combination of measures including a structured interview based on the *DSM* criteria, a semistructured interview to elicit demographic data, phenomenology and course of the disorder, and the Structured Clinical Interview of *DSM-IV* Axis I Disorders (SCID-I/P; First, Spitzer, Gibbon, & Williams, 1996) to rule out outbursts that resulted solely from another mood or psychotic disorder.

Pyromania

Diagnosis is primarily made through exclusion, using the *DSM-IV-TR* (APA, 2000) criteria. Assessment instruments for general child and adolescent fire-setting behavior are also available (for a review, see Wilcox & Kolko, 2002).

IMPACT ON ENVIRONMENT

Pathological Gambling

Though estimating the actual social costs of pathological gambling is problematic due to the multifactorial nature of the disorder and difficulty in establishing causality, it is generally accepted that pathological gamblers are at increased risk for committing crimes, accruing debts and filing for bankruptcy, and defrauding employers (NRC, 1999). In addition, they are at high risk for separation or divorce, unemployment, mental and physical health problems, and are likely to pass on intergenerational patterns of addictive behavior to their children (NRC, 1999).

It is estimated that 21 percent to 85 percent of pathological gamblers commit crimes like fraud, stealing, embezzlement, forgery, robbery, assault, and blackmail, and 25 percent to 39 percent are convicted and may serve prison terms for those offenses (for a review of the social cost literature, see Nower, 2003). In a national study in the United States, one-third of problem and pathological gamblers reported arrests, compared to 10 percent of social gamblers and only 4 percent of nongamblers (NORC, 1999). In addition, about 23 percent of pathological gamblers and 13 percent of problem gamblers had been imprisoned, compared to 4 percent of social gamblers and 0.3 percent of nongamblers (NORC, 1999). Another study found that one in five identified callers to a gambling helpline admitted to committing illegal acts to finance their gambling, and those gamblers were more likely to be younger, report suicidality secondary to gambling, use alcohol or drugs, and require mental health treatment (Potenza et al., 2000).

Several investigators have attempted to estimate the relative costs of gambling-related crimes, though methodologies differ and fail to clarify the causal relationship between gambling and crime (Shaffer & Korn, 2002). Derived from studies in the United States and depending upon location and survey methodology employed, estimates of gambling-related theft and misappropriation range from $6,000 to $61,000, suggesting a total annual cost of about $1.7 million in police, probation, incarceration, and judicial administration costs (NORC, 1999; Thompson, Gazel, & Rickman, 1996b).

A high percentage of gamblers will also face bankruptcy, defaulting on financial obligations at alarming rates. The Gambling Impact Behavior Study (GIBS), a national survey of gambling in the United States, estimated that 19 percent of pathological gamblers, compared to 5.5 percent of social gamblers and 4.2 percent of nongamblers, filed for bankruptcy in 1998 and owed an estimated $1.80 for every $1 of income (NORC, 1999). Similarly, a Canadian study reported that one-third of problem gamblers had either filed for bankruptcy or owed debts ranging from $75,000 to $150,000 each (Ladouceur, Boisvert, Pepin, Loranger, & Sylvain, 1994).

Burdened with pressure from creditors, escalating rates of debt, mounting social stressors, and interaction with the court system, pathological gamblers also typically experience serious problems in the workplace. In a survey of the social cost literature, Lesieur (1998) reported that 69 percent to 76 percent of pathological gamblers were often late to work or absent in order to gamble. Others gambled on company time, reported moodiness and irritability, borrowed from fellow employees, stole from employers, and arranged for advances on their paychecks. In addition, 25 percent to 30 percent of pathological gamblers were likely to lose their jobs due to gambling—one-third due to theft—costing employers indirectly in unemployment or severance benefits, extended health benefits, and retraining costs (Ladouceur et al., 1994; Lesieur, 1998). The GIBS study in the United States found that pathological gamblers reported nearly triple the rates of unemployment of social and nongamblers (NORC, 1999). Additional job-related social costs also include search and training costs to employers as well as health and welfare payments (Thompson, Gazel, & Rickman, 1996).

Most pathological gamblers require mental health treatment and, possibly, medical attention for a range of stress-related medical problems at a cost of $722 to $1,000 per

gambler for outpatient (NORC, 1999; Thompson et al., 1996) and $20,000 to $28,000 for inpatient treatment services (Vatz & Weinberg, 1995). Studies have found that, even after discontinuing gambling, adverse medical and psychological symptoms persist, including depression, ulcers and heartburn, bowel disorders, insomnia, excessive weight gain, high blood pressure, back or neck pain, and headaches (Ladouceur et al., 1994; Lorenz & Yaffee, 1986).

Other environmental consequences include the long-term adverse effects on the gambler's family system. Discord between gamblers and their partners are common. Studies have reported that 23 percent of pathological gamblers engaged in affairs, 33 percent separated from their spouses, and 54 percent divorced (Lorenz & Yaffee, 1986; NORC, 1999).

Family turmoil may have dire implications for children. Jacobs et al. (1989) theorized that certain family environments breed "intergenerational effects wrought by highly stressed, preoccupied, inconsistent, and often absent parents who have provided seriously flawed parenting, sex, social and occupational role models for children" (p. 266). As a result, children of disordered gamblers reported feeling angry, hurt, isolated, depressed, and abandoned as a result of their parent's gambling problems (Lesieur & Rothschild, 1989). They are also more likely to develop dependencies on gambling, food, cigarettes, alcohol, and drugs. In addition, children of problem gamblers may experience physical abuse resulting from the displaced anger of one or both parents (see Darbyshire, Oster, & Carrig, 2001; Lesieur & Rothschild, 1989).

Trichotillomania

Embarrassment caused by hair-pulling behavior may lead to social isolation. Other environmental consequences are in need of further exploration.

Kleptomania

Little is known about the environmental impact of kleptomania, except that individuals typically enter treatment only after one or more arrests for shoplifting. In one study, more than 80 percent of participants reported they had been arrested, many on several occasions (Sarasalo et al., 1996).

Intermittent Explosive Disorder

Due to the nature and unpredictability of aggressive behavior, the disorder often results in job loss, school suspension, divorce, difficulties with interpersonal relationships or other impairments in social and occupational functioning, accidents (e.g., in vehicles), hospitalization (e.g., due to fights or accidents), financial problems, incarcerations, and other legal problems (APA, 2000). In one study of 27 individuals diagnosed with the disorder, 41 percent (n = 11) admitted to attempting homicide during an episode, and most had both destroyed property and seriously assaulted another person in the past, 37 percent (n = 10) with a weapon (McElroy et al., 1998). In addition, most of the subjects viewed their outbursts as highly problematic, resulting in distress as well as social, vocational, legal, and financial problems.

Pyromania

Individuals with pyromania are often indifferent to the personal or economic consequences caused by the fire or may derive satisfaction from the results of their behavior. Fire setting may lead to property damage, legal consequences, or injury or loss of life to the fire setter or to others (APA, 2000).

TREATMENT IMPLICATIONS

Pathological Gambling

Treatment for pathological gambling disorder typically includes some combination of attendance at GA support groups, counseling, hotline services, cognitive and behavioral therapies, and/or pharmacological interventions (for a review, see Petry & Armentano, 1999). Early therapeutic interventions involved psychodynamic treatment, attendance at GA and nonspecific professional treatment services. Studies found that the combination of GA attendance and therapy (Russo, Taber, McCormick, & Ramirez, 1984) were more effective than GA alone (Stewart & Brown, 1988), though abstinence rates for the combination were still only 50 to 60 percent.

The few controlled studies currently available have evaluated the efficacy of cognitive behavioral therapy and imaginal desensitization, a form of visualization combined with relaxation. Three studies found that imaginal desensitization was more effective than other behavioral treatments at decreasing gambling urges in treatment-seeking gamblers from one month to nine years following treatment (McConaghy, Blaszczynski, & Frankova, 1991).

Several studies have provided empirical support for cognitive behavioral therapy, particularly when combined with social skills training and cognitive restructuring that targets the notion of randomness (Bujold, Ladouceur, Sylvain, & Boisvert, 1994), though studies are traditionally hampered by small, nongeneralizable sample sizes, insufficient data on dropouts, and inconsistent administration of treatments (i.e.,

variability in treatment time; for reviews see Blaszczysnki & Silove, 1995; Toneatto & Ladouceur, 2003).

Similarly, an increasing number of studies are demonstrating promising results using pharmacological agents, including carbamazepine, naltrexone, clomipramine, paroxetine, fluvoxamine, and lithium (for a review, see Grant, Kim, & Potenza, 2003).

It is important to note that drop-out rates in treatment programs for pathological gambling approach 50 percent, due largely to the fact that individuals miss the thrill of gambling or gain increased confidence they could win and relieve their financial burdens (Grant, Kim, & Kushwoski, 2004). Predictors of treatment completion include a positive response to treatment within eight weeks and having a supportive environment (Grant et al., 2004). Another study found the level of impulsivity to be the single biggest predictor of drop-out rates (Leblond, Ladouceur, & Blaszczynski, 2004).

Trichotillomania

Until the 1990s, trichotillomania received little interest from the research community apart from case studies or descriptive reports of hair pulling and associated disorders and behaviors. Treatment consists primarily of pharmacotherapy combined with behavioral interventions. Case studies have reported mixed results using a variety of medications including lithium, chlorpromazine, amitriptyline, buspirone, isocarboxazid, fensluramine, progestin, and selective serotonin reuptake inhibitors (SSRIs; for a review, see Diefenbach et al., 2000). The tricyclic antidepressant clomipramine has proven the most efficacious in controlled studies. However, it is often difficult for clients to tolerate the medication at adequate doses, which severely limits compliance (Walsh & McDougle, 2001). More benign medications are generally ineffective, though topical preparations and psychotropic medication may help some clients who are experiencing difficulties with treatment or relapse (Walsh & McDougle, 2001). Studies suggest that long-term success of pharmacotherapy alone may be limited, reporting a significant reduction in symptoms for the first few months followed by a high rate of relapse long term, particularly when dosages are lowered or the drugs are discontinued (Alexander, 1991; Iancu, Weizman, Kindler, Sasson, & Zohar, 1996; Swedo, 1993b).

Increasingly, optimal treatment includes a combination of pharmaco- and behavioral therapies. Clinicians have attempted to treat the disorder with therapies including biofeedback, covert sensitization, aversion therapy, positive practice, extinction, overcorrection, and response prevention (Diefenbach et al., 2000). However, one treatment—habit reversal training (HRT; Peterson, Campsie, & Azrin, 1994)—has received the strongest empirical support. HRT, initially formulated with nine components, is designed to identify conditioned cues associated with hair pulling, interrupt hair-pulling behavior, and replace the behavior with adaptive coping strategies (Diefenbach et al., 2000). It is most effective when combined with cognitive therapy (Rothbaum, 1992). However, HRT is a highly specialized and intensive treatment modality, practiced by a limited number of therapists, thereby limiting accessibility to many clients (Walsh & McDougle, 2001).

Kleptomania

The few reports that address effective treatment for kleptomania consist of case studies or small clinical samples, often to ameliorate shoplifting behavior rather than to address kleptomania as a disorder. Studies utilizing covert, systematic, and imaginal desensitization (see Opdyke & Rothbaum, 1998) have reported moderate success. In addition, other studies have noted a remission of symptoms in patients treated with regular pharmacological treatment, including antidepressants (most commonly SSRIs), benzodiazepines, mood stabilizers, thymoleptic, and opioid receptor antagonists, often as adjuvants to cognitive behavioral therapy (Durst, Katz, Teitelbaum, Zislin, & Dannon, 2001; McElroy et al., 1991; Sarasalo et al., 1996).

Intermittent Explosive Disorder

No protocol exists for the treatment of intermittent explosive disorder. Individuals in one study indicated they received the most help from insight-oriented psychotherapy, which assisted them in increasing control over aggressive impulses (McElroy et al., 1998). Other subjects in the study reported undergoing behavior modification or couples, group or family therapy with little success.

Preliminary investigations have suggested that individuals with explosive disorder benefit from treatment with tricyclic antidepressants, serotonin specific reuptake inhibitors (SSRIs), and mood stabilizers such as lithium or carbamazepine (see McElroy, 1999; McElroy et al., 1998). In one study, subjects with a highly compulsive or impulsive presentation with a unipolar component were given SSRIs, whereas patients with affective instability or bipolarity were administered the mood stabilizer, divalproex (McElroy, 1999). The researchers noted that 60 percent of the subjects receiving pharmacotherapy reported a moderate response to SSRIs and a marked response to mood stabilizers, as measured by a reduction in aggressive impulses and explosive acts.

Opdyke and Rothbaum (1998) have suggested adapting therapeutic strategies used for other forms of behavioral out-

bursts as treatment for intermittent explosive disorder. One treatment, commonly used with mentally retarded inpatients, involves blocking violent sequelae with the use of physical restraint while simultaneously presenting escape-provoking stimuli. In theory, this procedure should extinguish aggressive behavior by removing the negative reinforcement that often encourages the behavior (i.e., to avoid the explosive outburst, the "victim" complies with the client's wishes). Alternatively, the differential reinforcement of other behaviors (DRO) approach, successful in treating self-injurious behavior, could be adapted so that clients are differentially reinforced or rewarded for failing to display explosive outbursts during a designated time period until the behavior is extinguished.

Pyromania

There are no existing treatment interventions for pyromania in the literature, though several authors have suggested optimal treatment strategies for adolescent fire setting behavior (for a review, see Soltys, 1992). Those interventions include parenting training, overcorrection/satiation/negative practice with corrective consequences, behavior contracting/token reinforcement, special problem-solving skills training, relaxation training, overt sensitization, cognitive behavioral therapy, fire safety and prevention education, individual and family therapy, and medication. It is, however, important to note that adolescent fire setting behavior typically differs from the idiosyncratic nature of pyromania, which would arguably require greater emphasis on pharmacological interventions, cognitive behavioral therapy, social skills, covert sensitization, relaxation and response cost (Opdyke & Rothbaum, 1998). Treatments to diffuse the buildup of tension, increase awareness of negative consequences of the behavior, and substitute healthy stress coping strategies should prove most effective.

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