PART 4

Assessment and Treatment of Impulse Control Disorders

This page intentionally left blank

CHAPTER

22 Assessment and Treatment of Pathological Gambling

Hermano Tavares

Abstract

As gambling becomes more popular, more people will be exposed to it; thus, the prevalence of and demand for gambling-related treatments are expected to increase. Pathological gambling (PG) is the most severe level of gambling compromise, characterized by unrestrained gambling to the point of financial and psychosocial harm. Classified among the impulse control disorders, PG resembles other addictive disorders. A host of scales for screening and diagnosing PG are available for both the specialist and the general practitioner. The diagnosis of PG, like that of other addictions, is based upon signs of loss of control over the target behavior (i.e., gambling), dose escalation (increasing amounts wagered to get the same excitement as in previous bets), withdrawal-like symptoms, psychosocial harm, persistent desire, and persistent betting despite the negative consequences. Its treatment requires thorough assessment of psychiatric related conditions, motivational intervention, gambling-focused psychotherapy, relapse prevention, and support for maintenance of treatment gains. Psychopharmacological tools to treat craving and gambling recurrence are an incipient but promising field.

Keywords: gambling, pathological gambling, assessment, gambling progression, comorbidity, personality, related medical conditions, risk behavior, psychotherapy, psychopharmacology

Introduction

Over the last decades, the cultural perspective on gambling has changed from a morally questionable behavior to a legitimate entertainment. Prevalence reports estimate that more than 80% of the North American population has participated in some sort of betting in the last year, making gambling as common as drinking and smoking and certainly more prevalent than drug taking (Welte et al., 2002). Along with gambling popularization has come the usual concern that, due to this broader access, more vulnerable people will be exposed to the harmful aspects of gambling: its addictive potential, loss of control over betting, indebtedness, psychological distress, and social maladjustment. Several risk factors have been associated with problem gambling, such as male gender, low socioeconomic status, unemployment, and low education level (Kessler et al., 2008).

Shaffer et al. (2002) proposed a classification of gambling behavior divided into three categories: Level 1 comprises gamblers who gamble without experiencing adverse consequences, the so-called social gamblers; Level 2 gamblers are those who have experienced some adverse symptoms corresponding to problem gambling; Level 3 includes those who fulfill criteria for pathological gamblers as defined in the DSM-IV-TR (APA, 2000). A fourth group, pulled out of Level 3, comprises Level 4, consisting of treatment-seeking pathological gamblers with particularly severe gambling behavior (Shaffer et al., 2002).

In the psychiatry scenario, pathological gambling (PG) is a diagnosis still looking for a proper place. It is currently classified among the Impulse-Control Disorders Not Elsewhere Classified of the DSM-IV-TR (APA, 2000), but its approach and

treatment are mostly based on previous knowledge derived from addictive disorders (Potenza, 2009). Indeed, several claims have been made to classify PG among a broader category including both behavior and substance addictions.

Problem gambling and PG have been deemed more prevalent among ethnic and religious minorities, but this association is controversial, with other authors claiming that rather than being a culturally specific vulnerability, a tendency toward disordered gambling could be related to impaired social insertion (Cunningham-Williams et al., 2007; Tavares et al., 2010). A host of individual factors may contribute to the development of PG. There are reports of family aggregation, and studies point to approximately 50% genetic heritability for PG (Lobo & Kennedy, 2009). Personality issues, particularly impulsivity, seem involved in the initiation and development of problem gambling (Pagani et al., 2009), as well as ineffective coping and cognitive styles (Petry et al., 2007). When assessing pathological gamblers, one has to bear in mind the multiple factors and their complex interactions in the origin and maintenance of harmful gambling.

PGambling Assessment

Screening for Problem Gambling and PG

The term *pathological gambling* was proposed in 1980 at the publication of the DSM-III (APA, 1980), and it summarized in operational criteria what popular culture already knew as compulsive gambling. In the revision of the third edition (DSM-III-R; APA, 1987), the diagnostic criteria for PG underwent extensive review (Lesieur, 1988). From the DSM-III-R to the current DSM-IV-TR, little change has occurred in the conceptualization of disordered gambling and its operational criteria, and despite the structural differences between the DSM-III and the subsequent versions, it seems that PG has been diagnosed in similar ways since 1980 regardless of the successive reviews of the diagnostic procedures (Petry, 2006).

The South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987) is a screening tool for PG based on the DSM-III criteria. Because it was one of the first gambling scales and is flexible in nature (it can be administered as a semistructured interview or as a self-report scale), the SOGS is still the most popular instrument for the assessment of problematic gambling behavior. It can be easily administered while patients are seated in the waiting room for a consultation. Lesieur and Blume (1993) revised the instrument for application in different settings, and concluded that the initial questions about gambling preferences can be modified to suit the games available in a particular jurisdiction and that instructions may undergo slight modifications to cover specific time frames, such as the past 12 months.

The SOGS has been translated and adapted to many cultures and languages, including Spanish, Italian, German, Turkish, Japanese, Hebrew, and Portuguese (de Oliveira et al., 2009; Lesieur & Blume, 1992). Although simple, the SOGS requires attention when scored because a few questions from the introductory section are not counted in the total score. They provide brief but valuable information on game preferences and amounts wagered by the gambler, as well as a family history. Questions 12, 16(J), and 16(K) are not scored either. Question 12 serves as a lead-in to question 13, and subitems J and K from question 16 provide information on the existence of credit lines held by the respondent at bookies' locations and gambling venues. Each positive scoring item adds 1 point to the final score, which totals 20 points. A cutoff of 5 or more is indicative of probable PG. Scores between 1 and 4 may indicate problem gambling. The questions are formulated in a lifetime fashion. Individuals scoring 5 or more must go through clinical evaluation and diagnostic confirmation with the DSM-IV-TR criteria, in which the diagnostic status, whether current or in remission, must be ascertained (Blume & Tavares, 2004).

The original purpose of the SOGS was to investigate PG in alcohol- and drug-addicted patients, at-risk populations, and other groups in which frequent gambling is common behavior (mental health and general medical patients, college and university students). However, the SOGS has been used for several purposes beyond its original conception, including general population surveys. A concern is that the psychometric properties of the scale could vary, depending on the type of sample surveyed. Indeed, Stinchfield (2002) stated that compared to the DSM-IV criteria, the SOGS showed high agreement (.96), high sensitivity (.99), and modest specificity (.75) in a gambling treatment sample. The numbers for a sample drawn from the general population showed lower sensitivity (.67) and a tendency to overestimate the number of pathological gamblers (false-positive rate of .50).

Another easy way to screen for PG is the Lie/Bet Questionnaire (LBQ). The LBQ was derived from the DSM-IV diagnostic criteria. Johnson and colleagues (1997) developed a case-control study comparing 191 pathological gamblers and 171 controls.

The conclusion was that questions deriving from criteria 2 and 7 best discriminated pathological gamblers from nonpathological gamblers. The final format comprised only items that showed very high sensitivity (.99) and high specificity (.91). The questions are: "Have you ever lied to family and friends about how much money you have spent on gambling?" and "Have you ever felt that you needed to gamble for more and more money?" However, the PG rate in the sample was above 50% of the whole sample; the precision of the questionnaire may not be the same if it is applied to samples drawn from the general population, in which the PG baseline rate is close to 1%. Indeed, in a study investigating the psychometric properties of the LBQ in two Norwegian community samples, one comprising adult subjects ($N = 2014$) and the other adolescents ($N = 3237$; Götestam et al., 2004), the LBQ retained its high sensitivity and specificity rates, but the positive predictive value (i.e., the proportion of individuals who have the diagnosis among those who tested positive) was comparatively lower. In a Brazilian population survey in which 3007 home interviews were conducted, the positive predictive value of the LBQ was estimated at 58% (Tavares et al., 2010). Thus, the high sensitivity of the LBQ suggests that very few pathological gamblers may pass undetected through it, but the diagnosis, as with other screening tools, will require further confirmation through the DSM diagnostic criteria.

A small fraction of pathological gamblers seek help for their gambling problems. It is estimated that between 7% and 10% of pathological gamblers have ever sought treatment for PG in North America (Suurvali et al., 2009). However, problem gambling is related to poor health, and the presence of problem and pathological gamblers in general practice settings is disproportionate to the frequency of PG in the general population (Pasternak & Fleming, 1999). This means that they do seek medical attention for other problems likely related to the distress caused by gambling and an unhealthy lifestyle (Morasco et al., 2006; Potenza et al., 2002). Thus, general practitioners (GPs) are well positioned to recognize and approach problem and pathological gamblers. Nevertheless, GPs rarely ask about betting habits and may not be comfortable with the idea of asking patients if they gamble "out of the blue." Tolchard and colleagues (2007) have emphasized the role of primary care professionals in early intervention to prevent a habit from escalating to a serious gambling problem. They pictured such intervention in a flowchart that begins with the patient's report of stress-related complaints (headaches, depression, anxiety, and sleeping problems). This gives the GP an opportunity to ask about stressors, including alcohol and drug intake. In this context, questions about gambling will not seem awkward or misplaced. If the patient reports regular gambling, the GP can suggest applying the Early Intervention Gambling Health Test (EIGHT; Sullivan, 2000). The EIGHT is an eight-item self- or clinician-administered questionnaire with yes/no answers. Each positive answer adds 1 point to the score. If the score is 4 or more, gambling is affecting the patient's well-being and he or she should be referred for specialized treatment. If the score is below 4, the patient should receive information to raise his or her awareness of gambling risks and how to minimize them. The GP will then take a note to reassess the patient's gambling status at the next meeting.

Other target populations in which problem and pathological gambling are disproportionately high include those who seek treatment for substance use disorders and adolescents. In substance abuse patients, the prevalence of PG can be 4 to 10 times higher than in the general population (Spunt, 2002). The SOGS may be the preferable screening instrument in this case because it was originally developed for this purpose (Lesieur & Blume, 1987).

Screening for and diagnosing PG among adolescents may be a challenge because excessive gambling does not include observable signs of intoxication. Financial problems, which are the most easily observable signs of disordered gambling, are not common in this age group because adolescents do not have direct access to credit. Authors have previously criticized the DSM diagnostic criteria for being too centered on financial damage, thus hampering the identification of PG in youth. An adaptation of the original criteria was proposed: the DSM-IV Juvenile criteria (DSM-IV-J; Fisher, 2000). In the DSM-IV-J, criteria 2 (tolerance), 5 (escape), and 6 (chasing) were kept unchanged, while criteria 1 (preoccupation), 4 (withdrawal), and 7 (lies) received minor adjustments. Criteria 3 (loss of control), 8 (illegal acts), and 9 (risking job/education) underwent greater changes in order to better reproduce relevant features of adolescents' milieu. After these adaptations, criteria 8 and 10 partly overlapped, so the former was excluded. In the DSM IV-J, the subject is diagnosed as a pathological gambler if at least four out of nine criteria are positive.

The SOGS was also adapted for adolescents following the same rationale. The SOGS Revised

Adolescents (SOGS-RA; Winters et al., 1993) uses a 12-months time frame, with items reworded to adjust for adolescents' reading level. Items with low content validity that did not reflect adolescents' gambling behavior were removed, and only 1 point is given for having any source of borrowed money, instead of the possible 9 points provided by each separate item from the original version. The final version has 12 items; a score of 4 or higher indicates a probable pathological gambler, 2 to 3 an at-risk gambler, and 1 or 0 no gambling problem (Winters et al., 1995). The SOGS-RA exhibited good reliability (alpha = .80) and a significant correlation with several measures of gambling involvement activity (Winters et al., 1993). The SOGS-RA was compared to the DSM-IV-J, and the scales showed rather high agreement (Derevensky & Gupta, 2000).

A host of other screening tools with moderate to excellent psychometric properties are available to the clinician; they will not be reviewed here because the subject goes beyond the scope of this chapter. The reader is referred to a comprehensive review provided in the report "A Critical Review of Screening and Assessment Instruments for Problem Gambling" by the Ministry of Health and Long Term Care, Substance Abuse Bureau (Canada) at http://www.gamblingresearch.org/download.sz/Critical_Review.pdf?docid=4120.

Other population segments of interest are women and the elderly. Men still make up the majority of problem and pathological gamblers, but female gambling is steadily increasing (Welte et al., 2004). Women's progressive emancipation makes it less likely for them to be entirely dependent on a male partner. However, financial independence is not a reality for all of them; thus, the previous concern that diagnostic criteria focused excessively on financial problems could cloud the diagnosis of PG. Moreover, several reports describe gender differences in gambling motivation and behavior. Female pathological gamblers are less likely to commit crimes and report less indebtedness than males. Conversely, they may demonstrate a faster progression to PG and higher comorbidity with anxiety and depression (Martins et al., 2002; Potenza et al., 2001; Tavares et al., 2001). They also report gambling to escape from psychological distress more often than men (Ledgerwood & Petry, 2006a). Nonetheless, the potential gender bias in the diagnosis of PG remains uninvestigated. The same holds true for the elderly; since fewer people depend upon their income compared to younger age groups, there may be little awareness of their excessive commitment to gambling. When investigating gambling harm in the elderly, clinicians should keep in mind the potential compromise between the use of retirement savings and the narrowing of leisure and coping strategies, which at the moment of assessment may not be perceived as a threat by the patient (Ladd et al., 2003).

Problems in identifying PG in particular populations stem partly from the DSM. Starting with DSM-III, a choice was made to base the diagnostic process on clearly observable behavior using so-called operational criteria. The obvious advantage is that a set of criteria provides greater diagnostic uniformity. The disadvantage is that if contingencies surrounding one special population change, then there may be variations in the target behavior. A potential solution is the adoption of adapted criteria, as in the case of adolescent gambling, but it is possible to foresee an unpractical proliferation of alternative criteria sets (for women, the elderly, ethnic groups, etc.). Another solution would be to combine observable behavior-based criteria with psychopathological analysis of core features that are shared by all individuals with problem gambling (Shaffer & Kidman, 2003). Thus, the assessment of a clinician specializing in PG should not be limited to the investigation of standard criteria, but should also include features of the structure that sustains gambling symptoms, such as cognitive distortions about randomness (Toneatto & Gunaratne, 2009), bias in affective regulation and decision making (Bechara, 2003), impulsivity, and a craving-eliciting process (Tavares et al., 2005), among others yet to be determined.

Diagnosing PG and Psychiatric Comorbidities

In the DSM-IV-TR (APA, 2000) PG is classified as a disorder of impulse control, but its diagnostic criteria are modeled on substance addiction (Cusack et al, 1993). It is defined as recurrent maladaptive gambling behavior that is not better accounted for by a manic or hypomanic episode. In order to be diagnosed as a pathological gambler, the subject has to fulfill at least 5 out of the10 following criteria:

1. "[I]s preoccupied with gambling (e.g., preoccupied with reliving past gambling experience, handicapping or planning the next venture, or thinking of ways to get money with which to gamble)." The pathological gambler's life is centered on gambling. Numbers that casually cross an individual's sight—car plate numbers,

someone's birthday date, or any other random sign— become cues that the gambler interprets as hints of numbers to pick on a lottery ticket or to bet on in roulette or other games.

2. "[N]eeds to gamble with increasing amounts of money in order to achieve the desired excitement." This phenomenon parallels the tolerance seen in substance dependence and requires careful investigation because bets escalate with gambling progression for a number of reasons, sometimes because the gambler is trying to recoup previous losses (criterion 6). Gambling tolerance is best ascertained when the gambler reports that betting small amounts, as he or she did in the past, does not provide the same pleasure as before, and that it only feels like real gambling when the stakes are high.

3. "[H]as repeated unsuccessful efforts to control, cut back, or stop gambling." Regarding control during gambling sessions, Dickerson & O'Connor (2006) describe three types of behavioral response: One response does not involve any conscious effort: in this case, after gambling starts, the gambler ceases in a few hours or less when he or she feels satiated. Another behavioral response requires a commitment to cease gambling when reaching a limit that the gambler sets for him- or herself before the session begins (e.g., a maximum gambling expenditure per day or a maximum number of hours spent in a casino). When the gambler is approaching the previously set boundary, there may be some internal struggle, but overall, the self-established limit is respected. The third response occurs when the gambler is constantly reviewing the initial limit, consecutively building new boundaries that enable gambling to keep going until the initial limit is largely overcome (e.g., staying overnight at a gambling venue) or betting is externally interrupted (e.g., money is over, someone comes to rescue the gambler, or the venue is about to close). When one is a pathological gambler, this third possibility is the most frequent outcome of gambling sessions.

4. "[I]s restless or irritable when attempting to cut down or stop gambling." This phenomenon parallels the withdrawal symptoms seen in substance dependence. Recently, Cunningham-Williams and colleagues (2009) studied a sample of 312 gamblers from the community. They verified that restlessness and irritability were positively associated with gambling pathology. However, three additional withdrawal-like symptoms were experienced by 41% of the sample when attempting to quit or control gambling: feelings of anger, guilt, and disappointment. This association held true even when the overlap with comorbid substance withdrawal and depressive symptoms were considered. Besides these emotional withdrawal phenomena, one study described several bodily ones such as insomnia, headaches, upset stomach, loss of appetite, physical weakness, racing heart, muscle aches, difficulty breathing, sweating, and chills among 65% of pathological gamblers attempting to stop gambling (Rosenthal & Lesieur, 1992).

5. "[G]ambles as a way of escaping from problems or relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)." Escapism is a common motivation for addictive behaviors. However, it is so frequent among pathological gamblers that it is regarded as a diagnostic criterion, which does not happen in the diagnosis of other addictions. Gambling to escape a negative mood is particularly associated with female pathological gamblers (Ledgerwood & Petry, 2006a; Martins et al., 2002).

6. "[A]fter losing money gambling, often returns another day to get even ('chasing' one's losses)." Chasing is one of the earliest and most common symptoms reported by pathological gamblers. It also may occur in people who gamble once in a while without fulfilling diagnostic criteria for PG (Toce-Gerstein et al., 2003). Nevertheless, it represents a watershed between recreational and dysfunctional gambling. One who chases losses in subsequent betting has lost sight of gambling as leisure and is confusing it with a measure of his or her competitive skills and self-worth (Lesieur, 1984).

7. "[L]ies to family members, therapist, or others to conceal the extent of involvement with gambling." As pointed out in a study that investigated sensitive and specific criteria for PG screening (Johnson et al., 1997), along with loss of control, lying about gambling is a hallmark of problematic gambling, underscoring that one's betting has become socially undesirable.

8. "[H]as committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling." Evidence from community-based samples suggests that pathological gamblers can be divided into two levels of severity. In a Brazilian sample, a severity cutoff was established at 7 positive criteria (5–6 criteria defining the lowest level of PG and 7–10 the highest level; Tavares et al. 2010). Data from two U.S. samples placed

the severity cutoff at 8 positive criteria for PG (5–7 and 8–10 positive criteria, respectively, encompassing the lowest and highest levels of gambling severity). The majority of the highest-level gamblers reported engaging in illegal acts (Toce-Gerstein et al., 2003); thus, criminal offenses are an indication of severe gambling compromise. Such offenses usually involve white-collar crime but seldom violence (Lesieur & Rosenthal, 1991; Rosenthal & Lorenz, 1992). Despite the association between PG and antisocial behavior, the endorsement of criterion 8 far outweighs the prevalence of antisocial personality disorder (ASPD) in pathological gamblers, which is estimated at around 15% in clinical samples (Pietrzak & Petry, 2005). In other words, even law-abiding individuals may start appropriating other people's money when they become pathological gamblers. Their need for money overpowers their moral standards, and they begin rationalizing this activity as temporary borrowing, hoping that successful betting will allow them to return this "lent" money.

9. "[H]as jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling." Marital and family ties are the first relationships to be affected. It is no wonder that, cross-culturally, PG is associated with spouse separation, divorce, and loneliness (Park et al., 2009). As the gambler becomes more and more preoccupied with gambling activity, other relationships and activities will be damaged.

10. "[R]elies on others to provide money to relieve a desperate financial situation caused by gambling." Although indebtedness and bailouts alone do not confirm the presence of PG, they are the most easily observable external signs, pointing to the inability of gambling to allow the gambler to meet his or her financial obligations.

As described above, the diagnosis of PG follows an addiction's general rationale. It is preferable to understand that this rationale that has not changed over the last 40 years (Edwards, 1986) than to memorize the DSM criteria for PG, which are often revised in the next edition of the DSM. The fitness of PG in the addiction model has been questioned, but so far, no alternative model has proved better (Tavares & Gentil, 2007). The addiction model predicts that the potentially addictive stimulus changes the individual's subjective state by altering brain activity, in general terms stimulating or depressing neuronal activity. Gambling is regarded as a stimulating activity that in many ways can mimic the effects of stimulant abuse (Zack & Poulos, 2009b). When such action meets an individual's need to compensate for arousal that is either above or below the ideal level, and/or to restore a damaged self-image by means of temporary emotional empowerment and alienation, the contact with the stimulus is strongly reinforced (Brown, 1987; Jacobs, 1986). Recurrent exposure to the stimulus will cause two types of brain adaptation: compensatory mechanisms that lessen the stimulus impact in order to protect the brain activity and sensitization that prompts readily available behavioral responses to the stimulus and other related stimuli. The sensitization process establishes an association with environmental cueing signs, making the stimulus-seeking behavior progressively dominant in the individual's conduct.

In clinical terms, this cycle translates into three clusters of signs and symptoms: (1) tolerance to the stimulus effect and withdrawal symptoms if the stimulation is abruptly ceased or decreased, which does not allow for a timely readaptation of the compensatory mechanisms described above (corresponding to DSM-IV-TR criteria 2 and 4 for the diagnosis of PG); (2) loss of control over the self-administered behavior caused by progressive sensitivity to the stimulus (criteria 1 and 3); and (3) negative consequences that stem from the behavior's persistence (criteria 7 to 10). This three-cluster logic applies to any addictive behavior, whether a substance addiction or a behavioral addiction like gambling. Still, PG includes a fourth cluster encompassing features that are more closely related to the gambling behavior: escapism and chasing (criteria 5 and 6, respectively).

The criticism has been raised that basing a diagnosis of PG on its consequences establishes a circular logic that builds an apparent but false internal coherence (it is pathological because it is harmful, then it is harmful because it is pathological; Dickerson & O'Connor, 2006). In particular situations, the presence or absence of gambling harm may not indicate gambling pathology. In an extremely gambling-intolerant social environment, betting will bring harm even if the individual does not show other features of PG. In the opposite situation, a lenient family with enough money to spare may not bring pressure on an individual who may demonstrate all other signs of PG. The real issue is not the harm done by gambling, but the fact that gambling is self-reinforced despite any other negative consequence. Indeed, gambling persistence in the face of

mounting harm is both the most baffling and the most common phenomenon in PG clinics.

Psychiatric comorbidity is the rule for pathological gamblers assessed in clinical settings (Tavares et al., 2003a) and in the community (Petry et al., 2005). Neglecting other psychiatric diagnoses when assessing a patient may compromise treatment from the start. Hence, when assessing a patient complaining of gambling-related problems, it is of the utmost importance that the clinician take time to search for psychiatric symptoms beyond gambling behavior.

Gambling severity correlates with the severity of psychiatric problems in general (Desai & Potenza, 2008); therefore, the rates of psychiatric comorbidity in clinical samples should be higher since these samples include the most severe pathological gamblers. Interestingly, studies on psychiatric comorbidity in PG in clinical and community samples have reported roughly the same numbers. Classically, PG is more frequently associated with substance abuse disorders, as well as mood, anxiety, and personality disorders. The National Epidemiological Survey on Alcohol and Related Conditions study (NESARC) reported that in a sample of 195 pathological gamblers from the community, 73.2% had a lifetime alcohol use disorder, 60.4% had nicotine dependence, 38.1% had a drug use disorder, 49.6% had a mood disorder, 41.3% had an anxiety disorder, and 60.8% had a personality disorder, among which the strongest associations were reported for antisocial, histrionic, paranoid, and dependent personality disorders (schizotypal, borderline, and narcissistic personality disorders were not assessed in this first wave of the survey). All associations remained strongly significant when pathological gamblers were compared to nongamblers even after controlling for variations in demographic profile and socioeconomic status (Petry et al., 2005). Associations between alcohol and nicotine dependence, drug use disorders, major depression, generalized anxiety disorder, and PG were significantly stronger for female than male pathological gamblers. This one point contradicts a previous report concerning a large sample of treatment-seeking gamblers, in which men had more problems with substance addictions, while women had more problems with behavioral addictions (Nelson et al., 2006). This contrast is probably due to the different origins of the samples. Another point of interest is that the associations between psychiatric disorders and substance use are usually stronger for drug disorders than alcohol disorders, whereas in PG the opposite pattern is shown. This may reflect similarities in the environmental and social milieus of drinking and gambling, since both are nowadays legal and normative behaviors; alternatively, both addictions may share specific genetic and biological features yet to be discovered.

An important though neglected disorder in comorbidity with PG is attention deficit hyperactivity disorder (ADHD). In the adult form, the hyperactivity facet of ADHD tends to disappear, but deficits in the more complex components of attention, particularly executive functions, remain. Neuropsychological assessments of pathological gamblers classically display deficits in executive functions, especially inhibitory control, cognitive flexibility, and planning; the same profile is observed in drug-dependent individuals (Goudriaan et al., 2006). Moreover, a history of childhood ADHD is a risk factor for the development of addiction during adolescence and adulthood (Rodriguez-Jimenez et al., 2006). Unfortunately, the comorbidity between ADHD and PG has not been explored in community surveys and is limited to a few clinical reports: Specker and colleagues (1995) found a 20% rate of adult ADHD in a sample of pathological gamblers, and Rodriguez-Jimenez and colleagues (2006) reported that roughly 30% of a sample of adult pathological gamblers without a current substance disorder (except for nicotine and caffeine) presented criteria for childhood ADHD. Pathological gambling and ADHD seem to share deficits in inhibitory control and planning, but pathological gamblers usually perform satisfactorily on continuous performance tests and tests of working memory (Goudriaan et al., 2004). This dissociation among the executive functions in PG (Rodriguez-Jimenez et al., 2006) has been regarded as a segregate involvement of ventromedial structures (associated with inhibition, decision making, and temporal integration of information) within the prefrontal cortex, apart from its dorsolateral portion (related to sustained attention). The application of neuropsychological evaluation in PG assessment and treatment remains to be explored, but it seems promising. The efficacy of therapeutic strategies aimed at improvement of executive functions could be assessed by neuropsychological testing. For instance, it has been suggested that pathological gamblers with comorbid ADHD, or ADHD traits indicated by some degree of executive function deficits, may profit from treatment with bupropion, which could simultaneously act on comorbid depression, craving, and attention-related aspects of PG (Black, 2004). The same may hold true for the future of psychostimulants in PG treatment (Zack & Poulos, 2009a).

In the realm of psychosocial interventions, gamblers with reduced executive functions will profit more from treatment with structured goals and practical examples of the application of verbal information provided during therapeutic sessions (Goudriaan et al., 2006).

Psychopathology of PG and Assessment of Related Phenomena

Craving is a central phenomenon in addiction syndromes that presents in two forms. One is self-limited and secondary to the withdrawal syndrome; it ceases when withdrawal symptoms are resolved. The other form, the primary craving, is a challenge to addictions' treatment. It consists of a persistent desire that arises in spikes usually elicited by external cueing established by classical conditioning, and it may last for years after the addictive behavior is interrupted (Drummond, 2001). But craving is also elicited by internal cueing, usually related to the need for emotional regulation. Koob (2000) defined craving as the memory of a pleasant, rewarding experience superimposed on a negative affective state. Indeed, in two independent studies, gambling craving was associated with depression and decrease of positive affects during abstinence in the first weeks of treatment; this craving was different from alcohol craving, which was associated with rebound of negative affects in the same period (de Castro et al., 2007; Tavares et al., 2005). In both studies, pathological gamblers' cravings were more frequent, more intense, and harder to resist than the cravings of alcohol-dependent individuals. Thus, craving in PG is an important phenomenon that can lead pathological gamblers to relapse (Hodgins & el-Guebaly, 2004).

Studies that investigate gambling craving usually resort to adaptations of craving scales developed for substance addictions, but specific gambling craving scales have been recently validated. The Gambling Symptom Assessment Scale (G-SAS) was developed to measure treatment efficacy in pharmacological trials for PG. It is a 20-item self-report scale with a one-dimensional structure, and almost all items address gambling craving during the preceding week. It showed significant test-retest reliability and cross-validation with other scales of reference (Kim et al., 2009). Another option is the Gambling Craving Scale (GACS), a nine-item self-report scale. Factor analysis revealed a three-factor structure: anticipation, desire, and relief. Gambling craving as measured by the GACS predicted gambling severity, positive and negative affect, and persistence of play even with continued loss (Young & Wohl, 2009).

Another important characteristic of PG is a bias on the process of making decisions. The pathological gambler's unrelenting wagering is fostered by the hope of winning in the short term a significant amount of money with little effort (e.g., just placing few coins in the machine) or reversing a desperate situation at once with a turn of luck. The fact that in the long run small losses outweigh larger occasional wins—hence the pathological gambler's dire financial situation—is overshadowed by the possibility of an instantaneous gain. Pathological gamblers make decisions based on immediate consequences while ignoring long-term outcomes not only in gambling, but also in other aspects of life, a feature that is shared with substance-addicted individuals. This is what Bechara (2005) calls "myopia for the future." He proposes that an imbalance between two related systems, the amygdala-striatum–based impulsive system and the ventromedial cortex–based reflective system, is responsible for the impairment in decision processes, which in colloquial terms is reported by the patients as lack of willpower. This model predicts two possibilities: either a hyperactive, impulsive system hijacks the reflective system and establishes an oversensitivity to reward, or a hypoactive, reflective system is incapable of providing a time perspective when environmental contingencies require a behavioral choice.

Two tests have generally been used to investigate the decision-making process in PG and other addictive behaviors: the Iowa Gambling Task (IGT; Bechara, 2003) and the Delay Discount Test (DDT; Petry, 2001). In the IGT, subjects are given four decks of cards and have to draw cards from them at their choice. The cards may either give or take points represented as hypothetical money. Two decks (A and B) give high rewards but even higher losses. The other two decks (C and D) give lower rewards but even lower losses. It is expected that after several trials, the subject will learn that decks A and B are disadvantageous and that decks C and D, despite their lower winnings, are better options. The performance of pathological gamblers on the IGT resembles that of patients with lesions in the ventromedial cortex: unlike normal individuals, who shift during the test from decks A and B to C and D, pathological gamblers tend to persevere with decks A and B, thus privileging large winnings and larger losses. In the DDT, the subject is invited to choose between an immediate but smaller monetary gain and a later but larger gain—for example $999 now or $1000

a month later. Then options are presented in which the present choice is progressively lowered until the subject chooses to wait for the higher and later reward. Typically, pathological gamblers and patients with other addictions accept steeper discounts favoring a present reward than nonaddicted controls do. As described above, it is a clinical fact that pathological gamblers also apply this *time discount* to punishment; that is, the aversive relevance of being heavily indebted because of continuing betting diminishes if this will happen only in the future. In other words, beyond variations in the sensibility to reward or punishment, pathological gamblers have difficulty processing time itself; this problem contributes to the deficits in planning and making decisions from a temporal perspective.

But lack of reflection and time perspective may not fully account for bad decisions. Another possibility is that pathological gamblers observe gambling outcomes and jump to conclusions based on false cognitive premises. In the mid-1970s, researchers began to point out how chance events might influence cognitions. Langer (1975) demonstrated how a game as simple as coin tossing could yield an illusion of control by means of specific sequences of outcomes. Recently, Monaghan and colleagues (2009) found that undergraduates playing at an electronic gambling machine (EGM) developed irrational thoughts, erroneous perceptions of chance, and superstitious beliefs, depending on the outcome; those who lost showed less distorted thinking than those who won. Sharpe and Tarrier (1993) propose that cognitive distortions are the bridge between the conditioning processes at play in recurrent gambling. First, operant conditioning factors (winning, excitement, relief from a negative mood, etc.) prompt initial persistence at gambling. Through recurring betting, occasional winnings will be associated with events that occurred simultaneously or close in time (e.g., a certain hour of the day, a specific combination of betting numbers, or pressing the machine's button with the thumb). Such an association can be strengthened by a larger than usual financial reward or by a few chance repetitions that establish two kinds of distorted thinking (Toneatto & Gunaratne, 2009): primary illusion of control (the gambler thinks he or she can manipulate gambling and environmental features in order to produce the desired outcome) and secondary illusion of control (the gambler thinks he or she can read gambling and environmental cues in order to predict the outcome). These cognitive distortions foster unrealistic expectations of winning and more investment in gambling. Finally, the frequent reexperience of the psychophysiological effects of gambling consolidates a classical conditioning process establishing environmental gambling cues that will trigger the cravings, as mentioned above. However, if such a model was infallible, any frequent gambler would have the misfortune of once being fortunate and experiencing a "lucky streak." Then, sooner or later, he or she would turn into a pathological gambler. The truth is that there are regular gamblers who will never develop significant gambling problems. Compared to social gamblers, problem gamblers develop more superstitious beliefs, expectancies of winning, and attribution biases (i.e., positive outcomes are due to the gambler's control and predictive abilities, while negative ones are due to chance or the undesired interference of others; Joukhador et al. 2003). Thus, the question remains whether cognitive distortions are the result of one's history of a gambling chain of reinforcements, or if there could be a trait predisposition to develop superstitions and a wishful pseudorational logic.

Toneatto and colleagues (1997) cataloged several variants of cognitive distortions related to gambling: superstitious beliefs (including lucky charms, luck rituals, and particular states of mind that could favor victory) and biased analyses of gambling outcomes. Gamblers deal with good luck and bad luck as distinct units, both subject to transmission and contagion (e.g., rubbing the back of a player who has just won or avoiding contact with someone who has a losing streak). The gambler's fallacy and entrapment are two examples of interpretative biases; in both cases, the player fails to acknowledge that each gambling outcome is independent of previous and future ones. In the *gambler's fallacy*, the gambler analyzes preceding outcomes and compares them to the expected probability. If a given outcome occurred less often than expected—for example, even numbers have come out fewer times than odd ones on roulette—the gambler concludes that it is time to start betting on even numbers because they are more likely to come out in order to maintain the predicted proportion between all possible outcomes. In the *entrapment distortion*, the gambler believes that after successive losses, he or she must keep on gambling because a winning streak will occur to maintain a balance between wins and losses. Several studies have demonstrated a direct correlation between the intensity, frequency, and conviction of cognitive distortions and the severity of problem gambling (Källmen et al., 2008); alas, most of them involved "home-made" scales lacking proper validation. The Gamblers'

Beliefs Questionnaire (GBQ; Steenbergh et al., 2002) and the Drake Beliefs about Chance Inventory (DBC; Wood & Clapham, 2005) are two exceptions. The GBQ is a 21-item self-report scale designed to measure cognitive distortions related to gambling. The items are framed in 7-point Likert-type scales. The GBQ's factorial structure consists of two closely related factors, Luck/Perseverance and Illusion of Control, which can be summed to obtain a single score. The scale has good internal consistency and adequate test-retest reliability. The GBQ score is related to gambling severity and the duration of gambling sessions. The DBC is a 22-item scale framed in 5-point Likert-type scales. Like the GBQ, it has a dual structure, Illusion of Control and Superstition, with good internal consistency. Both scales were validated on gamblers from the community and treatment centers, proving reliable for players of different games. The DBC had the additional advantage of discriminating different sets of cognitive distortions according to different types of games.

Natural History and Progress of PG

Gamblers Anonymous (GA), early clinical reports, and the DSM state that PG is a chronic and progressive disorder. However, more recent contributions point to the existence of natural recovery among some pathological gamblers and suggest that an intermittent course is more frequent for problem and pathological gamblers (Nelson et al., 2009).

In the 1970s, Dr. Robert Custer established the first treatment unit for pathological gamblers at the Brecksville, Ohio, Veterans Administration Hospital (Custer & Milt, 1985). Based on his experience at that unit, he proposed a progressive course of PG. Gambling progression was divided into three stages: winning, losing, and desperation. Later, Rosenthal added a fourth stage: giving up (Lesieur & Rosenthal, 1991). Usually, a pathological gambler reports frequent wins at the beginning of his or her gambling career. In the case of Custer's patients, this could be explained partly by the fact that almost all of them were male, and many betted on horse races or card games in which experience and skill played a small part. Skill-based wins enhanced the enthusiasm for gambling and the gambler's confidence in his or her ability to predict or produce desired outcomes, thus boosting the betting that afterward would no longer follow any rationale. Another possibility is that pathological gamblers are bettors who experienced a big win or several small wins by sheer luck at the beginning of their gambling, predisposing them to increase their commitment to gambling (Lesieur, 1984). Finally, losing or defeat may prevail in the gambler's early gambling career, but several small losses will not lead to unbearable debts in this initial stage and are likely to be shadowed by fewer but bigger wins, which will create a memory bias toward the gambler's luck and ability at wagering. However, PG is considered a heterogeneous disorder with at least two types of gamblers: the action seeker, who is more likely to be a male, and the escape seeker, who is more likely to be a female (Ledgerwood & Petry, 2006a). Escape gamblers are not oriented to winning, but rather relief and alienation, which again are more easily achieved in the initial stage of gambling, when financial losses have not accumulated to the point where worries start rivaling oblivion. In both cases, the typical pathological gambler will refer to the initial wagering as a profitable and/or pleasurable activity, with a sense of nostalgia and a desire to go back to the time when betting was a positive experience.

The losing phase often begins with a losing streak that sooner or later is experienced by any frequent gambler or a bizarre incident that turns an anticipated win into a loss. Older gamblers refer to those happenings as *flukes*, poker players and younger gamblers as *bad beats* (Rosencrance, 1986). Sometimes, it does not take a specific gambling episode to enter this second stage, but merely the realization that one's bank account balance is negative and that gambling seems less profitable and more financially burdensome than it used to be. Either possibility is a harsh blow to the gambler's narcissism that will trigger a pattern of larger bets and chasing losses in an attempt to restore the battered self-image (Lesieur, 1984). Patients describe this deterioration of gambling behavior as *going on tilt*, when loss of control becomes frequent and lying to cover losses deepens the chasm between the gambler and his or her family. At this point, the pathological gambler turns to others to deal with urgent financial obligations (bailouts), promising to stop gambling in return.

The pathological gambler enters the desperation phase when the money from several bailouts is gambled away and no more credit is available. The uncovering of his or her reckless behavior approaches, and the gambler may think of suicide as an honorable escape (Petry & Kiluk, 2002). Treatment seeking is more expected in this phase than in the other phases; sadly, so are gambling-related suicide attempts. Help-seeking gamblers call this moment *hitting rock bottom*. At this point, the gambler faces three possibilities: trying to cut down or stop

gambling by him- or herself, looking for specialized help (GA, counseling, psychological or medical treatment), or giving in to the desperation phase. This last phase, proposed by Rosenthal (Lesieur & Rosenthal, 1991), represents the chronification of gambling when winning is no longer a goal; the aim is just to stay in action, and the gambler's social ties are disrupted.

However, in light of new evidence, the chronification of PG seems an unlikely event. Indeed, a recent study based on a large sample of gamblers from the community found that less than 30% of those who had ever fulfilled criteria for PG in previous years were still classified as pathological gamblers in the year of the survey (Nelson et al., 2009). Based on two nationally representative community samples of pathological gamblers, Slutske (2006) concluded that the course of PG could be described in three possible ways: variable, chronic, or episodic. In the NESARC sample, the most common pattern identified was one PG episode in a lifetime (61%) lasting for a year or less. Gamblers with more than one episode of PG reported variable durations ranging from a month to decades. Slutske and colleagues (2003) followed a sample of young problem gamblers (ages 18 to 29) and found that the incidence, past-year prevalence, and lifetime prevalence of problem gambling remained stable across adolescence and into young adulthood, but that at the individual level, problem gambling proved to be transitory and episodic. Thus, Custer's phases could still hold clinical meaning, but problem gamblers may alternate periods of abstinence and gambling, going back and forth through the winning, losing, and desperation stages. The inexorable move toward chronic PG (if not treated) predicted by Rosenthal's desperation phase may apply only to a minority.

Additionally, natural recovery (i.e., curbing gambling problems without specialized help) seems much more common than was previously thought: about one-third of the pathological gamblers from two nationally representative American samples did not experience any gambling problem in the 12 months before the survey (Slutske, 2006). An even higher rate of natural recovery was described for a community-based Australian sample. Men (92%) were more likely than women (57%) to experience recovery without any treatment. The overall rate was 82%, suggesting that natural recovery could be the rule rather than the exception (Slutske et al., 2009).

Not only is the diagnosis of PG unstable, but the diagnostic criteria also vary in nature. In a large sample from the Vietnam Era Twin Registry, Sartor and colleagues (2007) found that increases and decreases in PG symptoms over time were equally common. Nelson and colleagues (2009) used data from NESARC to investigate the prevalence of PG DSM criteria in the past year and in the years prior to the past year. Out of 2276 participants who endorsed at least one criterion for PG, 30% reported the same criterion in the past year and in the years prior to the past year, which means that 70% of problem and pathological gamblers either ceased or changed the expression of PG criteria. Indeed, about 40% of respondents who endorsed at least one lifetime criterion for PG did not report any criteria in the past year. Moreover, stability varied across PG criteria, and their frequency depended somewhat on the gambling severity status. Preoccupation (criterion 1) was the lifetime symptom most often reported (12.1%), followed by chasing (criterion 6, 7.1%), tolerance (criterion 2, 6.4%), and escape (criterion 5, 6.0%). Lying (criterion 7) and loss of control (criterion 3) were endorsed by roughly 3% of respondents. About 1% endorsed reliance on others to cover their gambling debts (criterion 10), withdrawal (criterion 4), and jeopardizing relationships. Participating in illegal acts to finance gambling (criterion 8) was endorsed by only 0.4% of the respondents. Dividing respondents into problem and pathological gamblers revealed that lying, loss of control, and withdrawal were more frequently reported by pathological gamblers. Preoccupation, chasing, tolerance, and escape were more common among problem gamblers. However, endorsing escape in the prior years caused a sevenfold increase in the probability of fulfilling criteria for PG in the past year, making criterion 5 the best predictor of progression to PG. Besides, escape was the most stable of all 10 criteria (53% of respondents who endorsed criterion 5 in the prior years also did so in the past year). Although not frequent, reliance on others (criterion 10) was the best predictor of progression from problem to pathological gambling. Illegal acts (criterion 8) and jeopardizing relationships were among the least stable criteria. In sum, authors propose that preoccupation, chasing, and tolerance (the last two translating into stakes escalation) could be gating criteria that help to identify at-risk individuals, inviting further investigation for PG. Escape gambling could be a symptom of vulnerability to PG that remains even in times of remission. Lying, loss of control, and withdrawal symptoms indicate severe gambling and probable PG. The low frequency and low stability of reliance

on others, jeopardizing relationships, and illegal acts cast serious doubt on the diagnostic utility of these symptoms. Previous studies (Toce-Gerstein et al., 2003; Zimmerman et al., 2006) have also raised concerns about these PG criteria. Jeopardy and illegal acts may work better as markers of high severity than as diagnostic criteria. If gambling is a behavior that moves like a pendulum, going back and forth from abstinence to unrestrained betting, then reliance on others, despite being an infrequent symptom, seems to be the best indicator of a specific direction, that is, from problem gambling to PG.

Another aspect of the course of PG that has been under investigation is how quickly an individual progresses from social to problem gambling. It is important to identify both stages because understanding what prompts a rapid progression helps to identify at-risk individuals, who should receive early attention, since the time span to prevent and remediate emerging gambling problems is shorter. In this case, efforts should be directed to strong and prompt interventions. Recognizing slow progress in individuals is also important. Analyzing those individuals may be helpful in finding factors associated with resilience to gambling problems. Besides, for slow-progress gamblers who are in the initial stages of problem gambling, there is time and opportunity to develop interventions that are more comprehensive and focused on long term outcomes.

The first contributions in this area stressed the role of gender in determining the speed of gambling progression. Among treatment-seeking gamblers and callers of a gambling helpline, female gamblers advanced faster than their male counterparts from gambling onset to the beginning of gambling problems, with time intervals varying from 6 months to 2 years (Potenza et al., 2001; Tavares et al., 2001). Generally, women started gambling later than men, but since they developed gambling problems more rapidly, both sought treatment roughly at the same age, usually in their mid-40s.

Besides gender, types of games have been related to the addictive potential of gambling and to faster progression, with the suggestion that games that sustain continuous arousal are more likely to provoke careless gambling. This can be achieved by associating gambling with an ongoing event in which the outcome is known at the end—the case of horse racing (Coventry & Norman, 1997)—or by shortening the time between placing the bet and observing the outcome, which allows faster repeated betting and continuous play—the case of EGMs (Dickerson & O'Connor, 2006). Indeed, in two studies, preference for an EGM was associated with faster gambling progression (Breen & Zimmerman, 2002; Tavares et al., 2003a). Thus, female gamblers may be at particular risk for rapid progression to gambling problems because of factors related to gender itself and because they have a greater preference for EGMs than men.

The gender factor seems to group several differences both with the demographic profile and with gambling behavior. Compared to male gamblers, female gamblers usually have a slightly lower socioeconomic status and more family responsibilities (Nelson et al., 2006). Despite coming from families that are less tolerant of gambling than those of men, women more frequently report having their initial gambling experiences with a family member, whereas men report having gambled for the first time with friends. They also differ in terms of games preference. For both genders, slot machines and EGMs are the most popular types of games, but women have a clear preference for EGMs and other games that are games of pure chance. They play a small variety of games and seem more loyal to one or two games. Men play table games more frequently and play a wider variety of games (Tavares et al., 2003a).

With so many differences reported, is gender a true factor in determining the course and natural history of PG or is it just a proxy for other potential cause–effect relationships? Studying the course of PG in 2256 pathological gamblers who sought treatment in the Iowa Gambling Treatment Program, Nelson and colleagues (2006) described four distinct gambling trajectories: (1) early start/fast progression; (2) early start/slow progression; (3) a gambling cycle developed during young adulthood (progressing from gambling onset to treatment seeking between the 20s and 30s—the young adult trajectory); and (4) a gambling cycle developed in mid-life (from the 40s to the early 50s—the mid-life trajectory). They also identified two more types of trajectories related to seniors— young-life gambling onset with late-life gambling problems and late-life onset with late-life gambling problems—but both had small numbers that did not yield statistical exploration. More men displayed the trajectory of an early start combined with slow or fast progression. Women prevailed in the mid-life trajectory, which was also related to low family acceptance, less game variety, and less comorbidity with substance-related addictions. The young adult trajectory correlated with fewer family responsibilities, a preference for games other than slots, and reports of a criminal history. Gender predicted

faster progression only when gambling onset was not regarded. When age at initiation of gambling was taken into consideration, the impact of gender on gambling progression variation was reduced to less than 2%. But gender had a significant effect on age of gambling onset, with women starting to gamble significantly later than men. In other words, gender is the best predictor of gambling onset, but everything that happens after this is a function of the time of life when gambling begins. It makes sense that dissocial behavior is more likely for early-onset gamblers, since youth tend to be more impulsive. Whether the progression for those early gamblers will be fast or slow seems to depend on environmental factors such as family acceptance and responsibilities. Gambling onset in mid-life involves individuals with more time and money to spare, and probably with less pressure to succeed professionally, marry, and raise young children. With fewer factors to divert their attention, mid-life gamblers can devote more time to gambling— hence the faster progression.

If variations on gambling behavior (onset, game of preference, etc.), environment (access, social and family tolerance, etc.), and individual factors (gender, impulsivity, etc.) can affect the expression of gambling problems, the motives for seeking and engaging in treatment must also vary. Seeking treatment could be critical in pathological gamblers' lives, even if they are likely to end PG by themselves, since it could shorten the time needed to achieve resolution and reduce the negative consequences. As stated above, only a minority of pathological gamblers ever seek treatment for their problems. Therefore, it is important to study what motivates both treatment seeking and treatment delay.

Suurvali and colleagues (2010) conducted a review on motivations for ending gambling by oneself and with specific help. They surveyed 10 studies addressing reasons for quitting gambling. Help seekers differed from those who solved their own problem in that among help seekers, gambling-related harm (financial and relationship issues and negative emotions including hitting rock bottom) was the most frequent reason for trying to quit. Self-help gamblers more frequently reported that gambling became incompatible with recent changes in the environment or with their lifestyle. They were also more likely to stop by using self-appraisal and decision making. It is worth noting that treatment seekers are very much aware of the negative consequences of gambling, but they are unlikely to acknowledge the slim chances of making money through gambling and reaching a decision based on this evaluation. This fact gives support to cognitive and motivational enhancement techniques applied in different gambling therapies and suggests that treatment is especially indicated for those pathological gamblers who may have biases in assessing the pros and cons of risk-taking behaviors that may compromise the decision-making process. Studies applying open-ended questions also identified incompatibility with self-image as a frequent reason to quit gambling (Hodgins et al., 2002; Toneatto et al., 2008). Indeed, during the first sessions when a patient is not entirely convinced of his or her desire or capacity to stop gambling, it is much more fruitful to focus on regaining self-control and restoring family ties as objectives shared by the therapist and patient, and then proceeding to the analysis of what is needed to achieve such goals. Usually, in time, gambling patients will realize that the image of the willpowered man or the caring, responsible woman they pursue is not compatible with gambling.

Suurvali and colleagues (2009) also conducted a review of barriers between pathological gamblers and treatment. The most frequently identified barriers were the wish to deal with gambling problems by themselves (including attempts to regain money lost to gambling by gambling even more, i.e., chasing), shame and secrecy, unwillingness to admit problems or not feeling ready to change the behavior, and concerns about treatment utility and requirements. Looking for further validation, Tavares and colleagues (2002) correlated self-reported reasons for delaying treatment with the number of years elapsed between the first gambling problem and the first specific attempt to seek help, called the *problem gambling interval*. "Chasing losses and efforts for self-control" and "shame and secrecy" correlated positively with the extent of the problem gambling interval; "lack of readiness to change" approached significance. It was also noted that after information on the potential addictiveness of gambling and where to seek help was made available, admissions for gambling treatment spiked.

PG and Personality

As previously mentioned, problem gambing and PG are both related to impulsive actions and the need for emotional regulation. Since the concept of personality deals with innate predispositions to motivated behavior and the processing and expression of emotions, it is natural to suppose that personality components may play a role in gambling initiation and persistence (Tavares & Gentil, 2007). Indeed, impulsive personality traits identified in

early childhood predicted gambling involvement in late childhood (Pagani et al., 2009) and problem gambling in adolescence (Vitaro et al., 1999) in two cohort studies. Meanwhile, negative emotionality has been deemed to play a role in vulnerability to gambling (Hand, 1998).

Two distinct approaches to personality issues in PG have been used. One is to investigate the frequency of personality disorders within the categories specified in the DSM; the other is to compare traits and personality dimensions in persons with PG with those in normal controls or social gamblers. The results vary greatly in studies that investigate the prevalence of personality disorders in PG, from 25% to 93% (Fernández-Montalvo & Echeburúa, 2004). There are several reasons for this discrepancy: (1) the studies differ in the type of gambling sample—because treatment-seeking gamblers represent a more severe stratum, clinically based samples typically report higher prevalences of personality disorders; (2) the studies that opted for self-report instruments displayed an almost threefold higher prevalence of personality disorder compared to those adopting a semistructured diagnostic interview; (3) comparing pathological gamblers with nongambling controls instead of nonproblem regular gamblers may artificially inflate the association between PG and personality disorder; (4) the studies fail to account for the impact of Axis I comorbidities in personality assessment; and (5) the studies fail to account for several overlaps between personality diagnoses, which inflate estimates and blur the description of associations with specific personality disorders. Bagby and colleagues (2008) compared pathological gamblers to social gamblers from the community in terms of personality disorder while accounting for all the potential biases described above. Twenty-three percent of the pathological gamblers presented at least one personality disorder, but only borderline personality disorder (10%) remained significantly associated with PG after statistical control for Axis I and II comorbidities was applied. Two fundamental features of borderline personality disorder are impulsivity and affect dysregulation, which is in keeping with previous descriptions of PG psychopathology.

Indeed, results from studies using different dimensional models of personality assessment display a fair amount of convergence. Usually problem and pathological gamblers have higher scores on dimensions representing negative emotionality and lower scores on dimensions representing constraint and self-control (or, by contrast, higher scores on impulsivity measures) compared to nongamblers and nonproblem gamblers. Using the Multidimensional Personality Questionnaire, Slutske and colleagues (2005) described this same profile for pathological gamblers and for alcohol-, nicotine-, and cannabis-dependent subjects and concluded that this combination of vulnerability to negative affects and impulsivity constituted the personality underpinning of addictions. A parallel outcome was reported by Nordin and Nylander (2007), who used the Temperament and Character Inventory, with pathological gamblers scoring higher than controls on two temperament factors: novelty seeking and harm avoidance. The former is closely associated with impulsivity and the latter with vulnerability to mood and anxiety disorders. Additionally, Vachon and Bagby (2009) propose that personality dimensions should be taken as a platform to investigate evidence-based subtypes of PG. Using the five-factor model of personality, they conducted a cluster analysis and described three subtypes of PG. Group one had normative personality scores and few or no comorbidities with Axis I and II conditions, and was distinguished from nonpathological gamblers only by the presence of PG, hence named *simple PG*. Groups two and three shared low scores on constraint, which translated into difficulty in controlling their urges and acting without full consideration for consequences. However, they differed regarding some aspects of the impulsive traits' profile. Group two, named *hedonic gamblers*, was high in excitement seeking, described as curious, excitable, and attracted to stimulation and pleasure. Group three, named *demoralized gamblers*, was high in neuroticism and low in extraversion, characterized by extreme negative affects, emotional instability, and social inhibition. Here lies another interesting convergence. Blaszczynski and Nower (2002) previously proposed a three-pathway model of problem and pathological gambling development. The pathways are not mutually exclusive, and the gambler may follow one pathway, any combination of two pathways, or all three at the same time. Pathway one groups the well-known features of operant and classical conditioning, relying mostly on environmental reinforcement contingencies. Pathway two subsumes issues related to emotional vulnerability and psychiatric comorbidity. Pathway three includes impulsive traits, dissocial behavior, and other factors believed to be genetically inherited. The three-pathway model proposes an interaction between environment and individual predispositions in

which different proportions of both function in each individual to lead him or her to PG. Vachon and Bagby's typology seems to contemplate this variation, with simple PG being less dependent upon individual differences, hedonic PG resembling the extraverted, disinhibited gambler and demoralized PG clearly associated with psychiatric instability. This last type includes a constellation of features that resembles the doubly conflicted nature of borderline patients. They are ready to throw their lives into the hands of fate without thinking twice, and at the same time they want to avoid harm and will not tolerate frustration. Furthermore, this personality-based typology provides evidence for the earlier clinically based division of pathological gamblers into action seekers and escape seekers. The assessment of the patient's personality is helpful in understanding the motivations for gambling and shaping psychosocial intervention strategies. Maybe in the near future it will also be useful in choosing the most appropriate pharmacological approach.

PG and Related Medical Conditions

The gambling habit has not been associated with good health by common sense, and recent data from a national survey support this perception. Among young and old respondents, gambling was significantly related to obesity, chronic medical conditions, and poor subjective health (Desai et al., 2007). Epidemiological surveys and clinical reports describe elevated frequencies of lifetime alcohol misuse (over 70%) and tobacco smoking (around 60%) among pathological gamblers (McGrath & Barrett, 2009). In addition, frequent gambling is likely to disrupt behavioral routines like nourishment and sleep, which are important for the maintenance of good health. Data from the NESARC show that pathological gamblers compared to nongamblers and low-risk gamblers had a twofold higher risk of being treated in an emergency room in the previous year, a more than twofold higher risk of being diagnosed with tachycardia and/or angina, an almost fourfold higher risk of developing hepatic cirrhosis, and a threefold higher risk of having any other liver disease (Morasco et al., 2006). Such associations remained significant even after controlling for demographic variations, body mass index, alcohol intake, smoking, and comorbidity with mood and anxiety disorder. The fact that PG remained significantly associated with liver disease even after accounting for alcohol intake calls attention to other factors of liver damage including infectious hepatic diseases. Since many of these diseases are caused by sexually transmitted viruses, it is feasible to speculate that impulsivity pervades other aspects of pathological gamblers' lives, including sexual behavior, which exposes them to further threats to physical health (Martins et al., 2004).

The risk for atherosclerosis, coronary diseases, and other cardiopathologies must be elevated as well, considering all the facts described above. Besides external factors (alcohol use, tobacco use, obesity, etc.), internal distress may also play a role. The importance of psychosocial stress to cardiopathy is a growing concern. An international study covering more than 52 countries concluded that permanent distress at work and/or at home, episodic vital distress, depression, and serious financial difficulties were all related to an increased risk for acute myocardial infarction (Rosengren et al., 2004). All of these conditions are the rule rather than the exception in PG. Nonetheless, the potential association between PG, acute myocardial infarction, and other cardiac events remains inexplicably underinvestigated and underreported. So far, all available evidence is indirect. Casino play was related to elevations in heart rate and salivary cortisol, showing that gambling is a stressful activity (Meyer et al., 2000). One study investigated 398 casino-related deaths in Atlantic City, New Jersey, from 1982 to 1986 and concluded that 83% of them were caused by acute myocardial infarction (Jason et al., 1990). However, the authors call for caution in interpreting this result because methodological constraints did not allow weighing of the specific contribution of gambling to sudden cardiac arrests. Nevertheless, considering that betting can be a stressful endeavor, subjects at risk for a heart condition should be warned that gambling could precipitate a cardiac event. Moreover, all gambling venues should have an on-site automated defibrillator, since the presence of this device was related to increased odds of survival following cardiac arrests in casinos (Valenzuela et al., 2000). Finally, Sharkey and colleagues (2005) examined 22 consecutive patients with reversible cardiomyopathy provoked by stress (all female patients) who were admitted to emergency rooms in the Minneapolis area. This syndrome was first reported in Japanese women, at the beginning also named the *broken heart syndrome*. Its clinical and laboratory findings perfectly mimic those of a myocardial infarction, and it may be triggered by an acutely stressful episode such as the death of a relative, domestic abuse, arguments, appalling medical

circumstances, and overwhelming financial or gambling losses.

A new interface between gambling and medical conditions is the association of Parkinson's disease and PG. The first reports date from 2000 (Molina et al., 2000; Seedat et al., 2000). The emergence of uncontrolled gambling in Parkinson's disease patients has been associated with the use of dopamine agonists for the treatment of the syndrome's movement impairment. Overstimulation of the corticostriatal dopaminergic system by these medications may cause not just hazardous gambling, but a host of impulsive behaviors like shopping, binge eating, and hypersexuality (Voon et al., 2009). Speculations have focused on the greater risk for this so-called dopamine dysregulation syndrome and specific dopamine agonists, particularly L-dopa and selective agonists of D3 receptors (pramipexole and ropinirole) that are primarily located in the limbic system (Dodd et al., 2005). However, since, in the majority of cases reported so far, more than one dopamine agonist was involved, it is not possible to assert that impulsive behaviors induced by dopaminergic agonists are due to specific action on any of the dopamine receptors or to general overstimulation of the dopaminergic system. Nonetheless, clinicians must be aware of this risk in treating Parkinson's disease, other movement disorders (e.g., restless legs syndrome), or any other medical condition that may require the use of dopamine agonists (e.g., prolactinoma).

The facts presented above demonstrate the importance of providing a thorough medical assessment of pathological gamblers who are initiating treatment, including physical and neurological examinations, as well as any additional laboratory exams suggested by the clinical anamnesis.

PG and Psychosocial and Environmental Stressors

Pathological gambling is clearly influenced by contextual factors, however little is known about the impact of psychosocial and environmental stressors in gambling behavior. For instance, attendance, participation, and group and family support are all important factors in abstinence maintenance for members of GA (Oei & Gordon, 2008). Epidemiological surveys clearly correlate PG and indicators of loneliness (divorce, separation, widowhood, etc.), shallow community ties (migration, unemployment, etc.), financial burdens, and problems with the legal system (Blaszczynski & Silove, 1996; Mazzoleni et al., 2009; Petry et al., 2005). But the causality direction in these cases is not yet defined. One study has investigated prior traumatic events and a lifetime history of PG (Scherrer et al., 2007) in a twin cohort. After controlling for psychiatric disorders, genetic factors, and family environmental influences, PG was still significantly associated with child abuse, child neglect, witnessing domestic violence, and physical attack. Moreover, genetic factors and family environment partly mediated the movement from trauma exposure to development of gambling symptoms. In adolescent gambling, the role of psychosocial factors is even more dramatic. In a sample of students from grades 7 to 13, it was found that problem gambling was associated with poor perceived family and peer support, substance misuse, conduct problems, family problems, and parental involvement in gambling and substance use (Hardoon et al., 2004). For adolescents and other patients with limited autonomy, some degree of intervention to lessen environmental adversity is warranted (e.g., family or couples therapy, foster homes, day-hospital care, or other forms of extended therapeutic support).

PG, Associated Risky Behaviors, and Global Functioning

Pathological gamblers are likely to seek treatment during episodes of personal crisis. In such situations they can be overwhelmed by psychiatric symptoms, and a host of medical and psychosocial conditions added to their intrinsically impulsive nature can compromise the capacity to fulfill the requirements of daily living. In such conditions, the gambler may develop moderate to severe impairment in social, occupational, or school functioning owing to suicidal ideation and risky behaviors. It is a common mistake, when initiating treatment for gambling or any other addiction, to focus on the patient's main complaint and forget that addictions and impulse control disorders are usually associated with other risky behaviors (Dell'Osso et al., 2006). For example, Hurt and colleagues (1996) reported that more than half of the deaths among patients previously treated for alcohol dependence were tobacco related. The same could be true for pathological gamblers, considering the high prevalence of nicotine dependence among them.

Pathological gambling is significantly associated with risky behaviors such as suicide attempts, sexually risky behaviors (most of all due to unprotected sex with casual partners or with a partner at risk for sexually transmitted diseases), substance misuse (mainly alcohol and tobacco), and legal problems

that require clinical attention. Impulsivity, age, gender, and emotional distress play different roles for different risk behaviors. In a clinical sample of 78 pathological gamblers, suicide attempts were associated with female gender and depression. Sexually risky behavior was associated with male gender and impulsivity measured by the Barratt Impulsiveness Scale. Alcohol misuse was solely related to male gender. Illegal activities to sustain gambling were equally reported by men and women, which correlated with lower age and impulsivity (Martins et al., 2004).

Suicide attempts are frequent in clinical samples (22% of patients reported having tried suicide at least once before receiving any mental health treatment; Martins et al., 2004) and in community-drawn samples of pathological gamblers. Newman and Thompson (2007) reported a more than three-fold higher risk of suicide attempts for pathological gamblers compared to nongamblers, even after accounting for other comorbid psychiatric disorders. However, the first study on suicidality and PG was published only in 2002 (Petry & Kiluk, 2002). It reported that gamblers with suicidal ideation had more psychiatric symptoms and comorbidities, were unsatisfied with their living conditions, and reported having more arguments and confrontations than usual in the month prior to the start of treatment. Suicidal ideation was also associated with gambling severity, higher cravings, and larger amounts of money spent prior to entering treatment. Acknowledging the factors associated with suicide attempts and other risk-taking behaviors in PG helps to extend treatment coverage and preventive efforts.

Treatment

In general terms, the treatment of any given disorder follows a cause–effect logic, meaning that the withdrawal of causes should annul the consequence. Unfortunately, for PG and almost all psychobiological conditions, we have very little knowledge of the causes. However, we do know a little about risk factors and processes that may lead to disordered gambling, and current treatments are mostly based on the reversal of such processes, elimination of risk factors, or compensatory strategies when such measures are not applicable. Acting according to this reasoning allows the clinician to achieve the general goals of PG treatment, which can be summarized in three guidelines: suppression of problematic gambling behavior, elimination of gambling-related problems, and promotion of general health (mental and physical) and a good quality of life (Walker et al., 2006).

As described above, a thorough assessment of the gambling patient helps to identify matters requiring urgent attention. It should also allow the formulation of hypotheses about factors leading to and maintaining problem gambling, which will determine the therapeutic strategy and the extent of intervention. To optimize the use of resources, brief interventions that try to match gambling severity and amount of intervention have been developed (Hodgins, 2005). Minimal interventions proposed include brief advice; one or a few motivational interviews; and use of a self-help workbook alone, or combined with one motivational interview over the telephone, or in person (Petry et al., 2008). In the following sections, a complete treatment approach to PG will be described. Applying any or all of the techniques described should depend upon the patient's needs and the resources available. Besides strictly gambling-oriented interventions, in particular cases PG treatment may benefit from further support such as family therapy (Ingle et al., 2008), couples therapy (Bertrand et al., 2008), and counseling for financial and forensic issues. Enrollment in GA may be particularly beneficial for gamblers facing difficulties with debt management and legal problems; valuable and practical strategies are passed on in these groups (Gamblers Anonymous, 1984). Besides, GA alone has been regarded as a valid approach to PG and is even better when combined with professional support (Petry et al., 2006). These supplementary aids to PG treatment will not be reviewed here; the interested reader will find further information about them in the references directly above.

No data are available on the efficacy of treatment programs developed for specific populations of gamblers (e.g., women, adolescents, the elderly, or young males) compared to standard programs. Likewise, no direct comparisons of different treatment formats (group versus individual therapy) have been done, except for one study that contrasted individual and group cognitive-behavioral treatment (CBT) for female pathological gamblers (Dowling et al., 2007). In this study, only individual CBT was superior to the control condition. Another study reported that in a community sample of gamblers attending outpatient CBT sessions, men improved more than women on measures of gambling severity and abstinence from betting. Generally, men found treatment components more useful, while women found specific gambling-related interventions

(identification of high-risk situations and distorted beliefs about gambling) less helpful (Toneatto & Wang, 2009).

First Measures: Comorbidity, Impulsivity, and Craving Pharmacology

Once a psychiatric comorbidity is identified in a gambler initiating treatment, it should be promptly treated; failure to do so can compromise treatment compliance and hence the efficacy of therapy. In addition, the institution of proper pharmacotherapy for the comorbid disorder promotes craving control (Dell'Osso et al., 2005). The management of depressive symptoms may be especially helpful, since the intensity of gambling cravings seem proportional to depression severity (Tavares et al., 2005). Though useful, antidepressants have one shortcoming: they require at least 2 weeks to have a significant clinical impact, and craving can be a disturbing experience in the first days of treatment (de Castro et al., 2007). In this case, complementary measures may include contingencies management and physical exercises. Some environmental cues work as nearly universal triggers for gambling craving, including availability of money and proximity to gambling venues and related stimuli. So, some of the first recommendations to patients are to reduce their access to credit (carrying money corresponding to a 1-week allowance only; leaving checkbooks, credit cards, and debt cards at home) and to avoid places and companies related to gambling whenever possible. In a pilot study, physical exercise proved to be beneficial for acute management of gambling cravings. Mean decrease in craving after exercise sessions was significantly correlated with reduction of gambling frequency, time and money spent betting, and distress caused by gambling after a 4-week program of aerobic activity (Angelo et al., 2009). However, both contingencies, control and exercise, are initiatives that require motivation and willingness to change gambling behavior; therefore, in some cases, it is advisable to implement these strategies after readiness to change is assessed and a motivation enhancement intervention is performed.

Currently, there is no medication approved by the U.S. Food and Drug Administration for the treatment of PG. But the psychopharmacology of impulsivity and gambling craving is a promising field. In a recent meta-analysis of pharmacological treatments for PG, Pallesen and colleagues (2007) described an overall effect size of 0.78. Cohen (1977) proposed that, for the behavioral sciences, effect sizes between 0.5 and 0.8 should be considered a medium effect, and values above 0.8 should be considered a large effect. Thus, the impact of pharmacological treatment could be regarded as relevant, but its appraisal must be relativized by some considerations. First, in another meta-analysis conducted by the same group (Pallesen et al., 2005), the estimated effect size for psychological treatments was 2.01. Indeed, research on psychosocial interventions for PG started earlier than research on pharmacological ones and seems to be in a more advanced stage of development. However, comparing the effect sizes of these two different approaches can be challenging because of methodological differences in measuring outcomes (somewhat stricter for pharmacological trials) and the choice of the control condition, generally active for pharmacological trials (placebo medication) and passive for psychological trials (waiting list). Second, estimated effect sizes of studies adopting a placebo-control condition are usually lower than the effect sizes of studies adopting a pre/posttreatment design, and in this meta-analysis only half of the 16 studies included were randomized, placebo-controlled trials. Finally, no differences in outcome were observed for the three main pharmacological classes (antidepressants, opiate antagonists, and mood stabilizers). Therefore, there is no solid empirical basis for preferring one type of medication over another.

The fact that most pharmacological trials include only subjects with low or no psychiatric comorbidity hinders the appreciation of how these medications work in real-life clinical situations. Moreover, it does not allow evaluation of the assumption that the comorbidity profile could determine the most suitable drug for a patient (e.g., preferring mood stabilizers over antidepressants for subjects within the bipolar spectrum of diseases). A better match between the patient's clinical needs and the chosen therapeutic drug could help improve compliance with the medication regimen and reduce treatment attrition, which is usually fairly high in both PG research and clinics. Another interesting finding is that women responded better than men in pharmacological trials for PG. Whether this can be attributed to gender differences in the biochemical underpinnings of PG or to placebo sensibility remains to be determined. The high response to placebo in PG has been a challenge for clinical research (Black et al., 2007a) but good news for clinical practice. It can be attributed to other factors coexisting with a pharmacological intervention, such as several reassessments emulating self-appraisal of the gambler's behavior and a budding bond with the therapist

that evolves during follow-up (Grant et al., 2003). In other words, a therapeutic alliance translated into shared goals and good rapport can work wonders in any chosen treatment route.

With the exception of bupropion, all of the antidepressants tried in PG treatment had serotonergic action because low serotonin activity has been related to impulsivity and to PG itself (Williams & Potenza, 2008). The majority of the antidepressants were selective serotonin reuptake inhibitors (SSRIs): fluvoxamine, paroxetine, citalopram, escitalopram, and sertraline. The results so far are inconsistent for several reasons: methodological inconsistencies (small sample sizes, open- label studies, and lack of control groups), treatment attrition, and a strong response to placebo. To deal with the last problem, researchers have resorted to a procedure called *lead-in*: all patients are assigned to placebo for a variable length of time prior to randomization. Randomized, controlled trials (RCTs) that have not applied the lead-in procedure have reported placebo response rates ranging from 47% (Black et al., 2007a) to as high as 72% (Saiz-Ruiz et al., 2005).

Typically, drugs that showed some promise in pre/posttreatment design investigations were not found to be superior to placebo on controlled tests. Fluvoxamine is one such drug. Hollander and colleagues (1998) reported that 7 out of 10 pathological gamblers completing an 8-week placebo lead-in phase followed by an 8-week single-blind fluvoxamine regimen were much improved or very much improved. Two RCTs that followed did not find a significant difference between fluvoxamine and placebo. Hollander and colleagues (2000) used a crossover design and found that, overall, fluvoxamine was not superior to placebo; however, in the second stage of the study, it caused a significant improvement compared to the control condition, suggesting that the placebo response could wear off with time. However, Blanco and colleagues (2002) undertook a longer fluvoxamine RCT (for 6 months) and found no differences. The placebo response rate was 59%. Nonetheless, fluvoxamine was significantly more effective in males and in younger patients. This finding points to the fact that not having an established typology of PG is a potential hindrance in accounting for treatment responses that could be specific to certain gambling subgroups. Paroxetine was tested in a 1-week lead-in/8-week RCT; the antidepressant was reported to have a significant impact on gambling urges and gambling severity (Kim et al., 2002). However, the results were inconclusive in the following larger multicenter study carried on by the same group (Grant et al., 2003). Sertraline was also tested in an RCT with negative results (Saiz-Ruiz et al., 2005). Citalopram (Zimmerman et al., 2002) and escitalopram (Black et al., 2007b; Grant & Potenza, 2006) were tested only in open-label studies with small samples; despite the preliminary auspicious results, they still await further validation.

Bupropion is an antidepressant with an unusual profile of action, including norepinephrine and dopamine agonism. It has been deemed useful in the treatment of nicotine dependence and ADHD (Wilkes, 2006). Gambling has been associated with changes in dopamine neurotransmission (Williams & Potenza, 2008), tobacco smoking (McGrath & Barrett, 2009), and ADHD (Rodriguez-Jimenez et al., 2006), which has fostered research investigations on the utility of bupropion in PG treatment. Again, promising results from an open-label study (Black, 2004) were not confirmed in a following RCT (Black et al., 2007a). The same problem with comorbidities and the profile of symptoms may have happened in this case. In fact, Zack and Poulos (2009a) reported a bidirectional effect of modafinil, a dopamine agonist psychostimulant, for gambling-related variables in pathological gamblers. The sample was divided into high-impulsivity and low-impulsivity gamblers according to an impulsivity median score. High-impulsivity gamblers showed a decrease in gambling desire, salience of gambling-related stimuli, disinhibition, and risky decision making after administration of a single 200-mg dose of modafinil. The opposite effect was observed for low-impulsivity gamblers. Many of the items from the impulsivity scale used in this study resemble attention deficit symptoms. Modafinil was found to be efficacious in the treatment of ADHD (Kumar, 2008). Finally, metilphenidate, a first-choice psychostimulant for the treatment of ADHD, was found to reduce risky decision making in a gambling task for children with ADHD (DeVito et al., 2008). All of this concurrent evidence suggests that psychiatric comorbidity and subtypes of impulsivity, whether related to affective or cognitive imbalance, may help identify the proper choice of medication for gambling patients. It also opens a new frontier of investigation into the use of psychostimulants in the treatment of PG.

Following this rationale, some authors have proposed that pathological gamblers suffering from affective instability should do better with mood stabilizers than with antidepressants (Hollander et al., 2005). However, the same problems described above

for antidepressants plague investigations of the efficacy of mood stabilizer in PG treatment. The only RCT conducted with a mood stabilizer tested the efficacy of sustained-release lithium carbonate in 40 patients with comorbid PG and disorders included in the bipolar spectrum (bipolar II disorder, bipolar disorder not otherwise specified, or cyclothymia). Patients taking lithium showed significantly decreased gambling urges and gambling-related behaviors; in addition, reduction in gambling severity was significantly correlated with reduction in symptoms of mania. Eighty-three percent of patients taking lithium were rated as responders compared to only 29% of the placebo group. One study compared lithium to valproate in PG treatment (Pallanti et al., 2002). Both were found equally efficacious, with a slight advantage for valproate in controlling anxiety symptoms, but the appreciation of this study is hampered because it lacks a control group. Extended-release carbamazepine for PG was tested in a small open-label study; despite the high rate of response (88%), several dropouts from the study due to adverse effects raise concerns (Black et al., 2008).

Opiate antagonists are probably the best-studied class of drugs in PG treatment. Their use is based on the several similarities between PG, alcohol, and other substance addictions. Naltrexone is a mu-opioid receptor antagonist that modulates the release of dopamine in the ventral tegmental area/nucleus accumbens/medial orbitofrontal cortex circuit, the so-called brain reward system. It has proven efficacious in the treatment of alcohol dependence syndrome, mostly by reducing the reinforcing properties of drinking and the intensity of craving episodes (Volpicelli et al., 1992). Naltrexone was superior to placebo for the treatment of PG in two methodologically sound RCTs. The first study applied a flexible dose design (Kim et al., 2001). Interestingly, the mean dose prescribed was 187.5 mg/day, which is much larger than the dose prescribed for alcohol dependence (50 mg/day). The second study investigated the specific effect of three dose regimens (50, 100, and 150 mg/day) against placebo on gambling cravings (Grant et al., 2008a). All three naltrexone groups were superior to placebo, but no difference in craving reduction was found between them. Naltrexone is well tolerated and safe, provided that the patient takes no acetaminophen, aspirin, or other nonsteroidal anti-inflammatory drugs; otherwise, liver enzyme levels may become elevated (Kim et al., 2006). In the same line, Grant and colleagues (2006) conducted an RCT of nalmefene in PG. Nalmefene is also a mu-opioid receptor antagonist. Compared to naltrexone, its advantages were a longer half-life, superior oral bioavailability, and no dose-dependent association with liver toxicity. It was also more effective than placebo, but although no direct comparison was carried out, in contrast to naltrexone, nalmefene was associated with more adverse effects (nausea, dizziness, and insomnia), and doses above 25 mg/day were hardly tolerated. Examining a combined sample from the three RCTs just described, Grant and colleagues (2008b) found that patients reported intense craving, and taking higher doses of an opioid antagonist was associated with a treatment response but only on a trend level, whereas a family history of alcoholism was a strong predictor of response. Nonetheless, a recent RCT for pathological gamblers demonstrating comorbidity with either alcohol abuse or dependence showed no significant action of naltrexone on gambling or drinking behavior (Toneatto et al., 2009). The fact that both groups received CBT counseling during the trial may have clouded potential differences, but it also underscores the importance of psychological treatments in PG. The majority of patients from both groups retained the treatment effects at the 1-year follow-up, demonstrating the strong impact of the therapeutic program.

Other ways to intervene in the brain reward system have been investigated through the use of modulators of glutamate neurotransmission and dopamine blockers. Grant and colleagues (2007) conducted an 8-week open-label trial with *N*-acetylcysteine, an amino acid that seems to restore the extracellular concentration of glutamate in the nucleus accumbens. Sixteen out of 27 patients were classified as responders (59%). Topiramate is an antagonist of *N*-methyl-D-aspartate (NMDA) receptors and an agonist of gamma-aminobutyric acid A (GABA-A) receptors. In different RCTs, it was superior to placebo in the treatment of alcohol dependence syndrome (Johnson et al., 2003) and binge eating disorder (Claudino et al., 2007). One study compared topiramate and fluvoxamine; both were considered efficacious, but the meaning of the findings is limited by the lack of a control group (Dannon et al., 2005). In one case report, a 57-year-old female pathological gambler with comorbid bipolar disorder receiving lithium therapy stopped gambling only after 200 mg/day of topiramate was added to the prescription (Nicolato et al., 2007). Specific blockade of D1 and D2 receptors has been speculated to occur in the treatment of PG. However, two RCTs with olanzapine, with negative

results, have dampened enthusiasm for the use of dopamine antagonists in the treatment of PG (Fong et al., 2008; McElroy et al., 2008). Indeed, claims have been made that activation instead of blockage of mesolimbic dopaminergic transmission is preferable in the treatment of psychostimulant dependence, and behavioral addictions that mimic psychostimulants' action (e.g., in gambling) and conditions related to deficient activity of the brain reward system in general (Blum et al., 2008; Zack & Poulos, 2009b).

First Measures: Psychoeducation, Motivation Enhancement, and Contingency Control

The initial psychological measures described here have no particular theoretical bounds. They may be used alone, in brief interventions, or as necessary steps anticipating further psychosocial intervention. Providing information to the patient about definitions of gambling, problem gambling, and PG helps establish the basis for therapy. The commonsense approach to uncontrolled gambling is heavily contaminated with moral appraisals, and so are the perceptions of gamblers and their relatives. This perspective focuses on the past (what should or should not have been done; if I were you; if we only knew what would happen; etc.), eliciting guilt that can increase desperation and further gambling. Pathological gamblers usually behave as if loss of control came out of the blue or was just fate. Thus, it is important that, along with definitions of gambling, patients learn the risk factors for gambling overindulgence, which will foster a more proactive attitude toward their problems. Providing statistics on gambling in the general population allows patients to contrast their own behavior with the norm. Also, information about gambling industry profits and government-revenues calls for a more critical view of gambling as a hidden alternative to direct taxing. Petry and colleagues (2008) developed a brief advice intervention and compared it with assessment only (control condition), motivational enhancement therapy (MET), and MET plus three sessions of CBT. The brief advice intervention was a 10-minute interview during which the therapist gave the patient a one-page handout that compared the patient's level of gambling with that in the general population, outlined risk factors related to severe gambling problems, and provided four steps to restrain the development of gambling problems. Interestingly, the brief advice alone was the only condition that showed a significant decrease in gambling in comparison to the control condition between baseline and week 6, relating to a significant decrease in gambling at month 9.

Treatment seeking is often triggered by a personal crisis. The gambler may be desperate, and this desperation increases chasing behavior even more. With the mind full of gambling worries, it is hard for the patient to concentrate on therapy. Therefore, the therapist has to enforce measures that may provide significant gambling-free periods to yield treatment compliance. This is where contingency control strategies such as the ones cited above are useful. But it is hard to impose anything on a gambler, and if the patient is not ready to comply, then dropout is likely. Early dropout is very common in PG treatment and can preclude a positive outcome. Impulsivity and drug or alcohol misuse are predictors of poor treatment compliance, underscoring the need for early and concurrent intervention for comorbid addictions (Melville et al., 2007). Motivational enhancement interventions increase the commitment to gambling treatment and can be particularly useful at this point in treatment (Wulfert et al., 2006). One simple and efficient intervention is to ask the patient to fill out a decisional balance sheet (Figure 22.1). The sheet contains a table divided into four quadrants. The upper ones concern the pros and cons of gambling, and the lower ones deal with the pros and cons of abstinence. The patient is invited to fill in the quadrants with topics listed in opposition (e.g., the benefits of gambling in the upper left quadrant, the benefits of abstinence in the lower left quadrant, and so on). A simple glance at the decisional sheet helps infer the patient's readiness to change. Too many topics in the pro-gambling quadrant and too few topics in others suggest a gambler in precontemplation. Topics concentrated in the cons quadrant suggest contemplation or preparation for action. More topics in the lower half of the sheet imply greater readiness to get into action and start changing one's behavior. The meaning of the stages in the readiness-to-change model can be discussed with the patient, and practical hints can be passed on (e.g., "You need to focus a bit more on the costs of gambling" for a patient in precontemplation or "You should start reflecting on your life without gambling; otherwise, it will be hard to change without knowing what lies ahead of you" for a patient in contemplation or preparation). Underscoring the relevance of advancing through the stages of change is important since it motivates the patient. In fact, readiness to change has been related to a better gambling treatment response (Petry, 2005).

Fill out the table below, indicating whether each pro/con is a short-term consequence (ST) or a long-term consequence (LT).

Pros (+) of Gambling	**Cons (–) of Gambling**
Pros (+) of Not Gambling	**Cons (–) of Not Gambling**

Reread each of the points you have made and correct any that may not be completely true. Add anything you missed. Which square has the most points? Which factors are most important? Overall, are there more positive or more negative long-term consequences of gambling? What about the long-term consequences of not gambling (abstinence)? What conclusions do you draw from this evaluation?

Fig. 22.1 Evaluating the pros and cons of gambling and abstinence.

One further strategy can be applied. After the decisional sheet is filled in and feedback is provided, the therapist invites the patient to regard the topics listed as consequences of gambling or not gambling. Next, attention is drawn to the fact that some consequences of gambling are short-term and others are long-term. Then the therapist asks the patient to cross out the short-term topics and look at the remaining items in the four quadrants. This will introduce, for the first time in treatment, the greatly needed time perspective discussed earlier. As simple as this exercise may be, it is interesting to observe patients' look of awe when they realize that most of the pros of gambling were eliminated from the decisional sheet—in other words, that gambling pleasures are short-lived. For those patients who are still ambivalent, it may be helpful to ask them to produce weekly logs of their gambling activities and to calculate the net financial outcome of gambling at the beginning of each treatment session.

At the end of the motivational exercise, patients should be asked to declare their goals for treatment. Concerning the gambling activity, it is advisable to present a forced choice among three possibilities: reducing gambling, quitting the most disturbing types of gambling, or abstaining from all forms of gambling. Herein lies a great deal of controversy. Due to the inherited rationale from the addiction field, total abstinence has been the most frequently proposed goal, but arguments against abstinence as the only acceptable treatment objective have been raised. The current literature lacks data on whether the chronicity and severity of gambling problems could predict for which clients choosing control over abstinence would be beneficial (Ladouceur et al., 2009). If a client chooses a treatment goal other than abstinence, the best thing to do is to deal with it candidly, admitting the controversy about it. It can also be pointed out that defining controlled gambling and self-assessment of one's control over

gambling can be tricky, and that gambling abstinence is easier to define and monitor. Finally, the therapist may suggest that patients try to attain abstinence for the duration of treatment, because this may improve the efficacy of therapy, and that afterward, they are free to decide the level of gambling involvement they want to keep.

If the therapist has not had the opportunity to discuss contingencies control and support activities (enrollment in GA, physical exercise, etc.), the period after completion of the motivational interview is the proper time.

Intervening in the Underlying Processes: The Behavioral, Cognitive, and Psychodynamic Models

Patients with severe gambling problems may need more than just psychoeducation, motivation enhancement, and contingencies control. For these gamblers, a host of proposals addressing the hypothetical underpinnings of PG have been proposed. In the field of gambling therapy, democracy still reigns. Behavioral, cognitive, and psychodynamic models have been applied to PG. Mixed interventions are popular, especially CBT and multimodal eclectic programs including psychodynamic techniques (Hodgins & Peden, 2008; Rosenthal, 2008). Few direct comparisons between diverse theoretical orientation programs have been made, and no clear outcome predictors have been associated with specific modalities that could ease the process of matching patients to the treatment that would best suit their needs.

Early attempts from the mid-1960s to apply behavioral principals to PG treatment focused mostly on aversive conditioning methods, but case reports and small, uncontrolled studies failed to provide consistent evidence of their usefulness (Tavares et al., 2003b). Later, covert sensitization, imaginal desensitization, systematic desensitization, relaxation training, and alternative sources of satisfaction were described, either alone or in combination in multimodal behavioral treatments (Hodgins & Peden, 2008) in small case series. The best-studied behavioral technique so far is imaginal desensitization. In a quiet room, patients are taught gradual relaxation. Next, the therapist asks them to visualize a circumstance in which gambling is likely to occur, and then the therapist suggests that they avoid this situation and engage in alternative activity. The format of imaginal desensitization therapy has varied across studies, from 14 sessions delivered twice a day for 1 week in an inpatient program (McConaghy et al., 1991) to 2 or 3 exposures to audiotaped imaginal exposure over 5 weeks (Grant et al., 2009). Three controlled trials compared imaginal desensitization to other techniques. The first study compared imaginal desensitization to aversion therapy (delivering an electric shock to the finger tips). Both therapies were considered effective 1 month after treatment, but only imaginal desensitization retained significant reduction of gambling (70%) 12 months after treatment (McConaghy et al., 1983). The second study compared imaginal desensitization to aversion therapy, imaginal relaxation, and in vivo exposure to gambling situations (McConaghy et al., 1991). Imaginal desensitization exceeded all other modalities, with close to an 80% response rate. Finally, in a recent study, Grant and colleagues (2009) compared imaginal desensitization combined with motivational, cognitive, and other behavioral techniques delivered in an 8-week program to GA referral. The combined program was significantly superior to GA referral, with a 64% abstinence rate at treatment completion. However, the multimodal format of the program precludes appreciation of the specific contribution of imaginal desensitization to the treatment outcome. The only other behavioral method tested in an RCT was in vivo exposure. In this technique, the patient is involved in sessions of progressive exposure to gambling-related stimuli, but only after a period of abstinence when stabilization has been obtained. The first sessions may include standing outside a gambling venue with no money while being coached by the therapist to prevent any behavioral response. The patient must feel the rise of anxiety and arousal, and the exposure can be terminated when both have clearly declined. The following sessions provide progressive exposure by manipulating environmental contingencies (outside/inside the gambling venue, back turned to the machine/front turned to the machine, with/without the therapist, and carrying no money/with money). Echeburúa and colleagues (1996) compared in vivo exposure only, cognitive restructuring, and a combination of both techniques to a waiting list control condition. At treatment completion, the exposure-only condition had a 69% abstinence rate compared to 38% for the other treatment groups and 25% for the control group. The problem, as in aversion therapy, was the steep decline in the abstinence rate a few months after treatment completion. In vivo exposure should be particularly considered when the environment precludes avoidance of gambling stimuli or when avoiding them would cost the patient a great deal of social seclusion. For instance, in some areas of Spain,

gambling machines are allowed in bars and people have the habit of gathering with friends and family in such places after work (E. Echeburúa, personal communication).

Cognitive restructuring aims to correct the cognitive distortions previously described. Among the cognitive and behavioral techniques proposed so far, cognitive restructuring has been the most widely studied. Its efficacy has been tested in both individual and group formats (Ladouceur et al., 2003). Early studies applied cognitive restructuring in conjunction with psychoeducation, problem solving, coping skills training, and relapse prevention. Therefore, doubts were raised about whether cognitive restructuring itself contributed to the results reported. To address this question, Ladouceur and colleagues conducted two RCTs comparing individual (2001) and group (2003) cognitive restructuring in which cognitive correction methods only were applied to identify gamblers' erroneous perceptions about randomness and to prevent relapses. In both cases, the experimental conditions were superior to the waiting list control condition on several gambling-related measures: frequency of gambling, perception of control, perceived self-efficacy, and desire to gamble. Cognitive restructuring is best conducted by asking patients to produce weekly records of their gambling urges (Figure 22.2). In these record sheets, patients note any occurrence of a gambling urge, assigning a score to it according to their subjective perception of intensity, and noting the environmental and internal conditions (bodily sensations and emotions) in which the urge emerged. Then they analyze their own thoughts while experiencing the craving for gambling, taking note and challenging them as to whether they represent rational or irrational approaches to gambling (i.e., if these thoughts defy the principles of randomness, such as uncontrollability and independence of events/unpredictability of future events). For each irrational belief recorded, patients should provide a rational alternative conceived by themselves (self-talk). Finally, a record of what happened afterward is required to determine whether the method used to cope with the urge was effective (What did they do? Did they gamble? How did they feel? What did they think of the whole experience?). In order to stimulate compliance with journal writing, the weekly sheets are handed over at the beginning of each session, completion must be praised, and brief feedback is supplied on the spot. A more detailed appraisal of the weekly log can be delivered in the next session, written on the back of the sheet containing observations about response patterns; hints specific to each patient on how to deal with gambling urges; and, always, praise for any progress recorded. Often the patient comes eagerly to the session just to enjoy the pleasure of reading what the therapist wrote. Besides the journal review, it is advisable to supply handouts about how games of chance operate (including information on real probabilities of winning, negative rates of financial return, the house edge, etc.) and the usual cognitive distortions that come to mind when gambling. Time should be taken to discuss which topics in the handouts apply to the patient's actual experience with gambling.

Psychodynamic and psychoanalytically oriented proposals for PG prevailed in the first half of the twentieth century, but investigations beyond cases and case series reports did not happen and psychodynamic treatments lost ground to modalities based on the diverse theories discussed above. Nonetheless, psychodynamic methods and perspectives survive undercover in multimodal eclectic therapeutic programs. In a review of psychodynamically oriented methods for PG treatment, Rosenthal (2008) concluded that there is enough evidence to justify further clinical research into this topic. Psychodynamic psychotherapy deals with specific issues that differentiate it from other forms of therapy: focus on the patient's emotions, emphasis on past relationships and interpersonal experiences, and exploration of secret or unconscious wishes, fantasies, and dreams. The unconscious mechanisms hypothesized to underlie frenzied gambling are (1) tension in the relationship with the father figure, who is perceived as harsh and never satisfied, leading to a secret desire to overcome laws of reality represented by the father (Freud, 1928); (2) an unconscious masochistic desire to lose fueled by guilt for rebelling against parental authority (Bergler, 1958); and (3) the need to restore a self-image damaged by parental neglect, using gambling as both an oracle and a test to ascertain whether one is loved and deserves the power that one secretly wishes (Rosenthal, 1987). In the multimodal eclectic approaches proposed (along with techniques such as psychoeducation, coping skills training, etc.), patients are encouraged to produce either a verbal or written autobiography while linking each passage with its emotional experience and its gambling potential coping function. Whether this kind of intervention contributes to more mature coping/defense mechanisms, and hence long-standing therapeutic gains in PG treatment, remains to be explored.

Urge	Triggers		Thoughts/self-talk			Consequences		
	External	Internal	Thoughts		Coping self-talk	Actions	Feelings	Thoughts
(0–10)	Situation (where, when, with whom?)	Body sensations or feelings	During urge and/or while gambling	Which thoughts are irrational and why? Illusion of control? Chance vs. skill? Independence of events?	Coping self-talk What's a more rational thing to tell yourself? How can you talk yourself out of gambling?	What did you do? (If you gambled record $ spent)	How did you feel after?	What did you tell yourself after?
Day 1								
Day 2								
Day 3								
Day 4								

Fig. 22.2 Weekly record of gambling urges.

Improving Coping Skills: Problem Solving, Social Skills Training, Stress Management, Broadening of the Leisure Repertoire, and Relapse Prevention

As the treatment progresses to its end, it is important to strengthen the gambler's coping abilities in order to prevent a relapse and to consolidate treatment gains. The methods applied at this stage will vary according to the patient's needs—for example, social skills training for patients with moderate to severe social anxiety or stress management and basic relaxation techniques for gamblers suffering from generalized anxiety (Hodgins & Peden, 2008).

At this point, it is expected that a significant period of abstinence has been attained or that gambling has been considerably reduced. Hence, patients are faced with an extra amount of free time. Leisure and related benefits, previously provided by gambling, must be obtained through new activities. Indeed, in a study of factors related to gambling abstinence in gamblers under treatment, quality of leisure and length of treatment were the best predictors of gambling reduction. Gamblers Anonymous enrollment, alone or in combination with formal treatment, is associated with leisure of better quality (de Castro et al., 2005). It is useful to have a handout with a list of low-cost or free leisure options available in the community and discuss them with the patients so that financial constraints do not become an overwhelming barrier.

Considering the pathological gambler's deficits in executive functions (i.e., control and planning) and vulnerability to negative affective states (anxiety and depression), almost all multimodal CBT programs include practice sessions on problem solving and affective coping methods. A detailed description of simple and efficient techniques for this purpose is presented in the cognitive-behavioral coping skills therapy manual by Kadden and colleagues (1995). With the aid of a five-question form, patients are guided through a stepped process in which they are expected to (1) recognize the problem; (2) describe its constituents; (3) formulate different approaches based on the previous appraisal; (4) balance the pros and cons of each option; and (5) evaluate the outcome of the chosen approach. For enhancement of affective coping, the therapist gives the patients a handout listing the different types of cognitive distortions that usually go along with anxiety and depression. These distortions are reviewed, and the patients are asked to provide examples related to gambling or other problems from their lives when appropriate. Then patients are taught to identify and challenge the distortions in their own thinking. Kadden and colleagues have provided a set of 20 questions meant to guide the patients through this process. The questions aim at encouraging an unbiased evidence-based appraisal of the facts, avoiding exaggeration of consequences and "all-or-nothing" thinking (e.g., "What's the evidence? Would this thought hold up in a court of law or am I jumping to conclusions based on circumstantial evidence?").

Two independent studies, one following GA attendees (Stewart & Brown, 1988) and another following pathological gamblers recruited through media advertisements who had recently quit gambling (Hodgins & el-Guebaly, 2004), reported that only 8% of the gamblers were able to achieve complete abstinence over 1 year of follow-up. Whether under naturalistic conditions, during treatment, or afterward, the fact is that gambling recurrence is expected. Blaszczynski and colleagues (1991) had previously argued that complete abstinence as the only acceptable treatment outcome is too stringent and that, in the phases of life after treatment, occasional gambling could happen and would not comprise the patients' general well-being. In fact, in their seminal work, Marlatt and Gordon (1985) make a point of discussing the utility of recognizing two types of recurrence of an addictive behavior: the lapse and the relapse. A *lapse* is defined as a discrete episode in which the target behavior is performed (e.g., gambling, drinking, drug taking, etc.) or in which actions represent a transgression of previously set boundaries (e.g., betting more than an agreed-on amount for gamblers pursuing controlled gambling or overeating while on a diet). A *relapse* involves a longer episode or several recurrences accompanied by a subjective sense of loss of control. It usually means a return to the addictive pattern of behavior prior to treatment or before a personal resolution. Lapses may happen and they may lead to a relapse, but not all lapses will do so. Thus, a lapse should be regarded as a high-risk situation for a relapse. Whether a lapse will lead to a relapse or not depends upon several feelings and actions taken after it has happened. Among several measures that can be used to keep a lapse from evolving, Marlatt and Gordon put special emphasis on preventing the patient from catastrophizing. The abstinence violation raises guilt that may lead to despair, which in turn will paradoxically provide a subjective excuse for further investment in the addictive behavior ("If everything is lost. . .").

In gambling relapse prevention, patients are taught important facts about relapses and trained to

recognize a lapse, its triggers, and other high-risk situations. Building effective coping skills will enable patients to deal properly with such circumstances, decreasing the likelihood of a relapse. This can be easily achieved by examining previous situations in which a relapse occurred. Next, a problem is proposed: what could be done to prevent a relapse should the patient ever have to face this situation again? Patients try to answer the question by using the five-step problem-solving method described above. In reviewing the literature about this theme, Ledgerwood and Petry (2006b) found that besides withdrawal and craving, cognitive, affective, and contextual precipitants are related to gambling relapses. Hodgins and el-Guebaly (2004) followed pathological gamblers who had made a commitment to gambling abstinence for 1 year. Factors contributing to relapses varied strongly, the most frequently reported being positive expectations about winning (23%), a need to make money (17%), lack of structured time or boredom (13%), giving in to cravings (11%), coping with negative emotions (11%), the need to socialize or fit in (8%), and seeking excitement (7%).

At this point, it may be useful to reassess the patient's impulsivity and cognitions about gambling (Oei & Gordon, 2008), since both have been related to a higher risk for relapse in the posttreatment phase. Goudriaan and colleagues (2008) reported that neuropsychological testing addressing inhibitory control and decision making provided strong predictors of relapse in PG. Problem-solving training can be especially well suited to deal with executive dysfunction and impulsive decision making. Indeed, a combination of problem solving and cognitive restructuring was deemed efficacious not only in treating gambling, but also in preventing at-risk gamblers from developing PG (Doiron & Nicky, 2007). If needed, booster sessions focusing on impulsivity and cognitive distortions can be offered before treatment closure.

In finishing this explanation of treatment methods for acute PG, it is important to reemphasize that the intensity of intervention has to match the level of gambling severity (Hodgins, 2005). Treatment must be tailored to the patient's needs. Severe gambling pathology will require a multimodal approach. Several components of the multimodal treatment packages for PG derive from clinical experience with other addictions (Tavares et al., 2003b). The field of gambling treatment is in dire need of dismantling studies, in which each component of multimodal approaches is tested in isolation so that its specific contribution to treatment can be estimated, as well as for whom, when, and under what condition such methods are likely to ensure a positive outcome. Additionally, Pallesen and colleagues (2005) suggest that future clinical research concentrate on the selection of universal outcome measures and instruments to ease the comparison between treatment modalities. Meanwhile, clinicians will have to base treatment options on the patient's assessment, the methods they are acquainted with, and continuous reassessment to redirect treatment whenever needed.

Maintenance: Stopping the Gambling Pendulum by Improving the Quality of Life

Naturalistic studies and community surveys suggest that the PG diagnosis waxes and wanes over time. Even for treated individuals, a review of various studies suggests that regardless of the treatment modality, it is difficult to ascertain the effects of treatment after 1 year of follow-up (Pallesen et al., 2005). This could be due to several intervening factors (e.g., marriage, childbirth, divorce, unemployment, aging) that accumulate as the time since the last therapeutic intervention increases, or it could be due to the possibility that current treatment modalities cannot provide long-term therapeutic effects. Indeed, the treatments proposed so far concentrate on intra- and interpersonal factors and immediate relationships with the environment at best. No treatment of gambling considers the gambler's life from a wider perspective, with the exception of one couples therapy model for PG. Starting from a systemic point of view, Lee and Rovers (2008) propose to approach four dimensions of human experience: intrapersonal, interpersonal, intergenerational, and universal-spiritual totality. The outcome reported for 24 couples was positive, with accounts of reduction in gambling urges and improvement in marital relationships. However, future controlled reports on short- and long-term outcomes are warranted to validate wider holistic models such as this one.

Nonetheless, the need to stop gambling oscillation remains, and despite the lack of solid evidences, suggestions can be drawn from indirect data. For instance, Sander and Peters (2009) stated that a good quality of life serves as a buffer, making it less likely for a gambler to relapse if he or she has a distressing experience. Frequent attendance at meetings and social support were the best predictors of abstinence for GA members (Oei & Gordon, 2008). But posttreatment initiatives must go beyond gambling-related issues, and a shift of focus from gambling to

enjoyment of life during this maintenance phase is advisable. In that sense, investing in methods to improve the quality of life could be helpful. Magalhães and colleagues (2009) report a group activity comprising nine open sessions offered to gamblers who completed the regular gambling treatment program. The topics debated during the sessions were mental health, physical health, social life, close relationships, work (ergonomics), family, financial health, spirituality, and leisure. Preliminary results show that out of 24 initial patients, 17 (71%) were frequent attendees after 2 years of follow-up. Of those regular participants, five (29%) maintained absolute abstinence, and the remainder, though having gambled occasionally, never fulfilled criteria for PG again. During this posttreatment phase, if patients have not engaged in regular physical exercising, this could be a good moment to try to motivate them. In the case of resolutely sedentary people, it is important to find alternative hobbies or challenges that can make them feel connected and involved. The goals are to create a coherent, healthy, and enjoyable lifestyle in which compulsive behaviors do not fit and to maintain an optimum level of stimulation that makes gambling relapses less likely. In this specific case, pathological gamblers may diverge from alcohol-dependent patients, for whom Alcoholics Anonymous advocates a low-stress, low-stimulation lifestyle (Brown, 1987).

Assessing Treatment Efficacy

The assessment of any therapeutic effort to treat PG is important for research and clinical purposes. Unfortunately, the field still lacks a consistent set of assessment tools that can reliably and comprehensively evaluate the effects of PG treatment. In this regard, pharmacological trials show greater uniformity. Most of them have applied the Yale Brown Obsessive Compulsive Scale adapted for PG (PG-YBOCS), the Clinical Global Impression (CGI), and direct measures of gambling behavior such as gambling frequency and amount of money gambled in a given period (Pallensen et al., 2007). This approach eases comparisons, but it is limited to craving, distress caused by gambling, and a few aspects of gambling behavior. Conversely, psychological trials have employed a greater variety of gambling variables to assess treatment, but variations on the assessment methods and variables adopted preclude comparisons. A group of experts, named the *Banff consensus*, proposed that the minimal required information to assess therapeutic results in PG should cover three domains: (1) measures of gambling behavior including the monthly net expenditure, the frequency (in days per month), and the time spent thinking about or engaged in gambling per month; (2) measures of gambling-related problems (relationships, financial and legal problems, etc.); and (3) measures of the process of change related to treatment modality (e.g., measures of cognitive distortions for gambling cognitive restructuring programs, defense mechanisms for psychodynamic treatments). The authors also state that the assessment of gambling problems could be complemented by measures of the quality of life (Walker et al., 2006). However, since both constructs seem to tap into different realms that play different roles (gambling problems having a determinant role in distress and motivation for treatment, quality of life being important to relapse prevention and consolidation of therapeutic gains), it would be advisable to approach both as separate domains. In particular, quality of life is a multidimensional concept whose definition has been a bit elusive. Maybe the best approach to it would be to unpack the model and choose specific domains that should be important for recovering gamblers, like a sense of belonging to a community, satisfaction with one's attainments in relation to one's culture, and shared value systems, social adjustment, and mental and physical health (Magalhães et al., 2009).

But the Banff consensus is about minimal requirements, and a good deal of valuable information would be lost if treatment assessment were restricted to the three domains mentioned above. As treatment success may depend upon the identification and treatment of Axis I and II psychiatric comorbidities, it is important to provide pre- and posttreatment assessment for both. A dimensional evaluation of specific personality domains, such as neuroticism and constraint/impulsiveness, could be even more specific and helpful. Core psychopathological features like cravings, inhibitory control, and decision-making biases should also be evaluated. Cognitive distortions, especially the subset that relates to overestimation of the odds of winning, should not be restricted to assessment of cognitive restructuring therapy, since they relate to positive expectancies about gambling that have an important role in facilitating relapses (Oei & Gordon, 2008). Besides, cognitive distortions can be reduced by other therapeutic approaches that do not include specific cognitive restructuring methods (Toneatto & Gunaratne, 2009). To date there is no comprehensive tool that satisfactorily taps into all of these domains. With the addition of a gambling session

to the Addiction Severity Index, this instrument has become the most comprehensive questionnaire currently available (Petry, 2007), but it still presents too narrow a range of gambling behavioral variables and its semistructured interview format requires time and training to use, making it far from ideal. There is still an unmet need for a brief and reliable instrument that can assess the main domains of gambling therapy while being complementary to more specific tools that should be selected according to the treatment goals and patients' specific features (de Castro et al., 2005).

Treating pathological gamblers can be challenging and sometimes tiresome, but the bulk of data on PG assessment and treatment reveal a condition that, though potentially devastating, is treatable, with fairly good response rates. Clinicians inclined to philosophical reflections will find in gambling problems a fruitful field for considerations about fate, acceptance, insurgence, risk, and choice—in other words, a lot to think about in life.

References

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.; DSM-III). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., Rev. Ed.; DSM-III-R). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision; DSM-IV-TR). Washington, DC: Author.

Angelo, D. L., Tavares, H., Bottura, H. M., & Zilberman, M. L. (2009). Physical exercise for pathological gamblers. *Revista Brasileira de Psiquiatria, 31*(1), 76.

Bagby, R. M., Vachon, D. D., Bulmash, E., & Quilty, L. C. (2008). Personality disorders and pathological gambling: A review and re-examination of prevalence rates. *Journal of Personality Disorders, 22*(2), 191–207.

Bechara, A. (2003). Risky business: Emotion, decision-making, and addiction. *Journal of Gambling Studies, 19*(1), 23–51.

Bechara, A. (2005). Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience, 8*(11), 1458–1463.

Bergler, E. (1958). *The psychology of gambling.* New York: International Universities Press.

Bertrand, K., Dufour, M., Wright, J., & Lasnier, B. (2008). Adapted Couple Therapy (ACT) for pathological gamblers: A promising avenue. *Journal of Gambling Studies 24*(3), 393–409.

Black, D. W. (2004). An open-label trial of bupropion in the treatment of pathologic gambling. *Journal of Clinical Psychopharmacology, 24*(1), 108–110.

Black, D. W., Arndt, S., Coryell, W. H., Argo, T., Forbush, K. T., Shaw, M. C., et al. (2007a). Bupropion in the treatment of pathological gambling: A randomized, double-blind, placebo-controlled, flexible-dose study. *Journal of Clinical Psychopharmacology, 27*(2), 143–150.

Black, D. W., Shaw, M. C., & Allen, J. (2008). Extended release carbamazepine in the treatment of pathological gambling: An open-label study. *Progress in Neuropsychopharmacology, Biology and Psychiatry*, 32(5), 1191–1194.

Black, D. W., Shaw, M., Forbush, K. T., & Allen, J. (2007b). An open-label trial of escitalopram in the treatment of pathological gambling. *Clinical Neuropharmacology, 30*(4), 206–212.

Blanco, C., Petkova, E., Ibanez, A., & Saiz-Ruiz, J. (2002). A pilot placebo-controlled study of fluvoxamine for pathological gambling. *Annals of Clinical Psychiatry, 14*(1), 9–15.

Blaszczynski, A., McConaghy, N., & Frankova, A. (1991). A comparison of relapsed and non-relapsed abstinent pathological gamblers following behavioural treatment. *British Journal of Addiction, 86*(11), 1485–1489.

Blaszczynski, A., & Nower, L. (2002). A pathways model of problem and pathological gambling. *Addiction, 97*(5), 487–499.

Blaszczynski, A., & Silove, D. (1996). Pathological gambling: Forensic issues. *Australia and New Zealand Journal of Psychiatry, 30*(3), 358–369.

Blum, K., Chen, A. L., Chen, T. J., Braverman, E. R., Reinking, J., Blum, S. H., et al. (2008). Activation instead of blocking mesolimbic dopaminergic reward circuitry is a preferred modality in the long term treatment of reward deficiency syndrome (RDS): A commentary. *Theoretical Biological Medicine Model, 5*, 24.

Blume, S. B., & Tavares, H. (2004). Pathological gambling. In J. R. Lowinson, R. B. Millman, & J. G. Langrod, J. G. (Eds.), *Substance abuse: A comprehensive textbook* (4th ed., pp. 488–498). Philadelphia: Lippincott Williams & Wilkins.

Breen, R. B., & Zimmerman, M. (2002). Rapid onset of pathological gambling in machine gamblers. *Journal of Gambling Studies, 18*(1), 31–43.

Brown, R. I. (1987). Gambling addictions, arousal, and an affective/decision-making explanation of behavioral reversions or relapses. *International Journal of Addictions, 22*(11), 1053–1067.

Claudino, A. M., de Oliveira, I. R., Appolinario, J. C., Cordas, T. A., Duchesne, M., Sichieri, R., et al. (2007). Double-blind, randomized, placebo-controlled trial of topiramate plus cognitive-behavior therapy in binge-eating disorder. *Journal of Clinical Psychiatry, 68*(9), 1324–1332.

Cohen, J. (1977). *Statistical Power Analysis for the Behavioral Sciences* (Revised edn). Hillsdale, NJ: Lawrence Erlbaum Associates, Inc.

Coventry, K. R., & Norman, A. C. (1997). Arousal, sensation seeking and frequency of gambling in off-course horse racing bettors. *British Journal of Psychology*, *88*(Pt 4), 671–681.

Cunningham-Williams, R. M., Gattis, M. N., Dore, P. M., Shi, P., & Spitznagel, E. L., Jr. (2009). Towards DSM-V: Considering other withdrawal-like symptoms of pathological gambling disorder. *International Journal of Methods in Psychiatric Research, 18*(1), 13–22.

Cunningham-Williams, R. M., Ostmann, E. L., Spitznagel, E. L., & Books, S. J. (2007). Racial/ethnic variation in the reliability of DSM-IV pathological gambling disorder. *Journal of Nervous and Mental Disease, 195*(7), 551–559.

Cusack, J. R., Malaney, K. R., & DePry, D. L. (1993). Insights about pathological gamblers. "Chasing losses" in spite of the consequences. *Postgraduate Medicine, 93*(5), 169–176, 179.

Custer, R., & Milt, H. (1985). *When luck runs out: Help for compulsive gamblers and their families.* New York: Facts On File Publications.

Dannon, P. N., Lowengrub, K., Gonopolski, Y., Musin, E., & Kotler, M. (2005). Topiramate versus fluvoxamine in the treatment of pathological gambling: A randomized, blind-rater comparison study. *Clinical Neuropharmacology, 28*(1), 6–10.

de Castro, V., Fong, T., Rosenthal, R. J., & Tavares, H. (2007). A comparison of craving and emotional states between pathological gamblers and alcoholics. *Addiction Behavior, 32*(8), 1555–1564.

de Castro, V., Fuentes, D., & Tavares, H. (2005). The gambling follow-up scale: Development and reliability testing of a scale for pathological gamblers under treatment. *Canadian Journal of Psychiatry, 50*(2), 81–86.

de Oliveira, M. P., da Silveira, D. X., de Carvalho, S. V., Collakis, S. T., Bizeto, J., & Silva, M. T. (2009). Reliability, validity and classification accuracy of the South Oaks Gambling Screen in a Brazilian sample. *Journal of Gambling Studies*, *25*(4), 557–568.

Dell'Osso, B., Altamura, A. C., Allen, A., Marazziti, D., & Hollander, E. (2006). Epidemiologic and clinical updates on impulse control disorders: A critical review. *European Archives of Psychiatry and Clinical Neuroscience, 256*(8), 464–475.

Dell'Osso, B., & Hollander, E. (2005). The impact of comorbidity on the management of pathological gambling. *CNS Spectrum, 10*(8), 619–621.

Derevensky, J. L., & Gupta, R. (2000). Prevalence estimates of adolescent gambling: A comparison of the SOGS-RA, DSM-IV-J, and the GA 20 questions. *Journal of Gambling Studies, 16*(2–3), 227–251.

Desai, R. A., Desai, M. M., & Potenza, M. N. (2007). Gambling, health and age: Data from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychology of Addiction Behavior, 21*(4), 431–440.

Desai, R. A., & Potenza, M. N. (2008). Gender differences in the associations between past-year gambling problems and psychiatric disorders. *Social Psychiatry and Psychiatric Epidemiology, 43*(3), 173–183.

DeVito, E. E., Blackwell, A. D., Kent, L., Ersche, K. D., Clark, L., Salmond, C. H., et al. (2008). The effects of methylphenidate on decision making in attention-deficit/hyperactivity disorder. *Biological Psychiatry, 64*(7), 636–639.

Dickerson, M., & O'Connor, J. (2006). *Gambling as an addictive behaviour: Impaired control, harm minimisation, treatment and prevention*. Cambridge: Cambridge University Press.

Dodd, M. L., Klos, K. J., Bower, J. H., Geda, Y. E., Josephs, K. A., & Ahlskog, J. E. (2005). Pathological gambling caused by drugs used to treat Parkinson disease. *Archives of Neurology, 62*(9), 1377–1381.

Doiron, J. P., & Nicki, R. M. (2007). Prevention of pathological gambling: A randomized controlled trial. *Cognitive Behavior Therapy, 36*(2), 74–84.

Dowling, N., Smith, D., & Thomas, T. (2007). A comparison of individual and group cognitive-behavioural treatment for female pathological gambling. *Behaviour Research and Therapy, 45*(9), 2192–2202.

Drummond, D. C. (2001). Theories of drug craving, ancient and modern. *Addiction, 96*(1), 33–46.

Echeburúa, E., Baez, C., & Fernandez-Montalvo, J. (1996). Comparative effectiveness of three therapeutic modalities in the psychological treatment of pathological gambling: Long-term outcome. *Behavioural and Cognitive Psychotherapy, 24*(1), 51–72.

Edwards, G. (1986). The alcohol dependence syndrome: A concept as stimulus to enquiry. *British Journal of Addiction, 81*(2), 171–183.

Fernandez-Montalvo, J., & Echeburúa, E. (2004). Pathological gambling and personality disorders: An exploratory study with the IPDE. *Journal of Personality Disorders, 18*(5), 500–505.

Fisher, S. (2000). Developing the DSM-IV criteria to identify adolescent problem gambling in non-clinical populations. *Journal of Gambling Studies, 16*(2–3), 253–273.

Fong, T., Kalechstein, A., Bernhard, B., Rosenthal, R., & Rugle, L. (2008). A double-blind, placebo-controlled trial of olanzapine for the treatment of video poker pathological gamblers. *Pharmacology and Biochemical Behavior, 89*(3), 298–303.

Freud, S. (1928). *Dostoyevsky and parricide* (Vol. III). London: Hogarth Press.

Gamblers Anonymous. (1984). *Big book: Sharing recovery Through Gamblers Anonymous*. Los Angeles: Author.

Gotestam, K. G., Johansson, A., Wenzel, H. G., & Simonsen, I. E. (2004). Validation of the Lie/Bet Screen for pathological gambling on two normal population data sets. *Psychology Reports, 95*(3 Pt 1), 1009–1013.

Goudriaan, A. E., Oosterlaan, J., de Beurs, E., & Van Den Brink, W. (2004). Pathological gambling: A comprehensive review of biobehavioral findings. *Neuroscience Biobehavioral Review, 28*(2), 123–141.

Goudriaan, A. E., Oosterlaan, J., de Beurs, E., & Van Den Brink, W. (2006). Neurocognitive functions in pathological gambling: A comparison with alcohol dependence, Tourette syndrome and normal controls. *Addiction, 101*(4), 534–547.

Goudriaan, A. E., Oosterlaan, J., De Beurs, E., & Van Den Brink, W. (2008). The role of self-reported impulsivity and reward sensitivity versus neurocognitive measures of disinhibition and decision-making in the prediction of relapse in pathological gamblers. *Psychological Medicine, 38*(1), 41–50.

Grant, J. E., Donahue, C. B., Odlaug, B. L., Kim, S. W., Miller, M. J., & Petry, N. M. (2009). Imaginal desensitisation plus motivational interviewing for pathological gambling: Randomised controlled trial. *British Journal of Psychiatry, 195*(3), 266–267.

Grant, J. E., Kim, S. W., & Hartman, B. K. (2008a). A double-blind, placebo-controlled study of the opiate antagonist naltrexone in the treatment of pathological gambling urges. *Journal of Clinical Psychiatry, 69*(5), 783–789.

Grant, J. E., Kim, S. W., Hollander, E., & Potenza, M. N. (2008b). Predicting response to opiate antagonist and placebo in the treatment of pathological gambling. *Psychopharmacology, 200*(4), 521–527.

Grant, J. E., Kim, S. W., & Odlaug, B. L. (2007). *N*-acetyl cysteine, a glutamate-modulating agent, in the treatment of pathological gambling: a pilot study. *Biological Psychiatry, 62*(6), 652–657.

Grant, J. E., Kim, S. W., Potenza, M. N., Blanco, C., Ibanez, A., Stevens, L., et al. (2003). Paroxetine treatment of pathological gambling: A multi-centre randomized controlled trial. *International Clinical Psychopharmacology, 18*(4), 243–249.

Grant, J. E., & Potenza, M. N. (2006). Escitalopram treatment of pathological gambling with co-occurring anxiety: An open-label pilot study with double-blind discontinuation. *International Clinical Psychopharmacology, 21*(4), 203–209.

Grant, J. E., Potenza, M. N., Hollander, E., Cunningham-Williams, R., Nurminen, T., Smits, G., et al. (2006).

Multicenter investigation of the opioid antagonist nalmefene in the treatment of pathological gambling. *American Journal of Psychiatry, 163*(2), 303–312.

Hand, I. (1998). Pathological gambling: A negative state model and its implications for behavioral treatments. *CNS Spectrums, 3*(6), 58–71.

Hardoon, K. K., Gupta, R., & Derevensky, J. L. (2004). Psychosocial variables associated with adolescent gambling. *Psychology of Addiction Behavior, 18*(2), 170–179.

Hodgins, D. C. (2005). Implications of a brief intervention trial for problem gambling for future outcome research. *Journal of Gambling Studies, 21*(1), 13–19.

Hodgins, D. C., & el-Guebaly, N. (2004). Retrospective and prospective reports of precipitants to relapse in pathological gambling. *Journal of Consulting Clinical Psychology, 72*(1), 72–80.

Hodgins, D. C., Makarchuk, K., el-Guebaly, N., & Peden, N. (2002). Why problem gamblers quit gambling: A comparison of methods and samples. *Addiction Research & Theory, 10*(2), 203–218.

Hodgins, D. C., & Peden, N. (2008). [Cognitive-behavioral treatment for impulse control disorders]. *Revista Brasileira de Psiquiatria, 30*(Suppl 1), S31–S40.

Hollander, E., DeCaria, C. M., Finkell, J. N., Begaz, T., Wong, C. M., & Cartwright, C. (2000). A randomized double-blind fluvoxamine/placebo crossover trial in pathologic gambling. *Biological Psychiatry*, *47*(9), 813–817.

Hollander, E., DeCaria, C. M., Mari, E., Wong, C. M., Mosovich, S., Grossman, R., et al. (1998). Short-term single-blind fluvoxamine treatment of pathological gambling. *American Journal of Psychiatry, 155*(12), 1781–1783.

Hollander, E., Pallanti, S., Allen, A., Sood, E., & Baldini Rossi, N. (2005). Does sustained-release lithium reduce impulsive gambling and affective instability versus placebo in pathological gamblers with bipolar spectrum disorders? *American Journal of Psychiatry, 162*(1), 137–145.

Hurt, R. D., Offord, K. P., Croghan, I. T., Gomez-Dahl, L., Kottke, T. E., Morse, R. M., et al. (1996). Mortality following inpatient addictions treatment: Role of tobacco use in a community-based cohort. *Journal of the American Medical Association, 275*(14), 1097–1103.

Ingle, P. J., Marotta, J., McMillan, G., & Wisdom, J. P. (2008). Significant others and gambling treatment outcomes. *Journal of Gambling Studies, 24*(3), 381–392.

Jacobs, D. (1986). A general theory of addictions: A new theoretical model. *Journal of Gambling Studies, 2*(1), 15–31.

Jason, D. R., Taff, M. L., & Boglioli, L. R. (1990). Casino-related deaths in Atlantic City, New Jersey 1982–1986. *American Journal of Forensic Medicine Pathology, 11*(2), 112–123.

Johnson, B. A., Ait-Daoud, N., Bowden, C. L., DiClemente, C. C., Roache, J. D., Lawson, K., et al. (2003). Oral topiramate for treatment of alcohol dependence: A randomised controlled trial. *Lancet, 361*(9370), 1677–1685.

Johnson, E. E., Hamer, R., Nora, R. M., Tan, B., Eisenstein, N., & Engelhart, C. (1997). The Lie/Bet Questionnaire for screening pathological gamblers. *Psychology Reports, 80*(1), 83–88.

Joukhador, J., Maccallum, F., & Blaszczynski, A. (2003). Differences in cognitive distortions between problem and social gamblers. *Psychology Reports, 92*(3 Pt 2), 1203–1214.

Kadden, R., Carroll, K., Donovan, D., Cooney, N., Monti, P., Abrams, D., Litt, M., & Hester, R. (Eds.). (1995). *Cognitive-behavioral coping skills therapy manual: A clinical research guide for therapists treating individuals with alcohol abuse and dependence* (Vol. 3). Rockville, MD: National Institute on Alcohol Abuse and Alcoholism.

Kallmen, H., Andersson, P., & Andren, A. (2008). Are irrational beliefs and depressive mood more common among problem gamblers than non-gamblers? A survey study of Swedish problem gamblers and controls. *Journal of Gambling Studies, 24*(4), 441–450.

Kessler, R. C., Hwang, I., LaBrie, R., Petukhova, M., Sampson, N. A., Winters, K. C., et al. (2008). DSM-IV pathological gambling in the National Comorbidity Survey Replication. *Psychology and Medicine, 38*(9), 1351–1360.

Kim, S. W., Grant, J. E., Adson, D. E., & Shin, Y. C. (2001). Double-blind naltrexone and placebo comparison study in the treatment of pathological gambling. *Biological Psychiatry, 49*(11), 914–921.

Kim, S. W., Grant, J. E., Adson, D. E., Shin, Y. C., & Zaninelli, R. (2002). A double-blind placebo-controlled study of the efficacy and safety of paroxetine in the treatment of pathological gambling. *Journal of Clinical Psychiatry, 63*(6), 501–507.

Kim, S. W., Grant, J. E., Potenza, M. N., Blanco, C., & Hollander, E. (2009). The Gambling Symptom Assessment Scale (G-SAS): A reliability and validity study. *Psychiatry Research, 166*(1), 76–84.

Kim, S. W., Grant, J. E., Yoon, G., Williams, K. A., & Remmel, R. P. (2006). Safety of high-dose naltrexone treatment: Hepatic transaminase profiles among outpatients. *Clinical Neuropharmacology*, *29*(2), 77–79.

Koob, G. F. (2000). Neurobiology of addiction: Toward the development of new therapies. *Annals of the New York Academy of Sciences, 909*, 170–185.

Kumar, R. (2008). Approved and investigational uses of modafinil: An evidence-based review. *Drugs, 68*(13), 1803–1839.

Ladd, G. T., Molina, C. A., Kerins, G. J., & Petry, N. M. (2003). Gambling participation and problems among older adults. *Journal of Geriatric Psychiatry and Neurology, 16*(3), 172–177.

Ladouceur, R., Lachance, S., & Fournier, P. M. (2009). Is control a viable goal in the treatment of pathological gambling? *Behaviour Research and Therapy, 47*(3), 189–197.

Ladouceur, R., Sylvain, C., Boutin, C., Lachance, S., Doucet, C., & Leblond, J. (2003). Group therapy for pathological gamblers: A cognitive approach. *Behaviour Research and Therapy, 41*(5), 587–596.

Ladouceur, R., Sylvain, C., Boutin, C., Lachance, S., Doucet, C., Leblond, J., et al. (2001). Cognitive treatment of pathological gambling. *Journal of Nervous and Mental Disease, 189*(11), 774–780.

Langer, E. (1975). The illusion of control. *Journal of Personality and Social Psychology, 32*, 311–328.

Ledgerwood, D. M., & Petry, N. M. (2006a). Psychological experience of gambling and subtypes of pathological gamblers. *Psychiatry Research, 144*(1), 17–27.

Ledgerwood, D. M., & Petry, N. M. (2006b). What do we know about relapse in pathological gambling? *Clinical Psychology Review, 26*(2), 216–228.

Lee, B., & Rovers, M. (2008). "Bringing torn lives together again": Effects of the first congruence couple therapy training application to clients in pathological gambling. *International Gambling Studies, 8*(1), 113–129.

Lesieur, H. R. (1984). *The chase: Career of the compulsive gambler* (2nd ed.). Rochester, VT: Schenkman Books.

Lesieur, H. R. (1988). Altering the DSM-III criteria for pathological gambling. *Journal of Gambling Studies, 4*(1), 38–47.

Lesieur, H. R., & Blume, S. B. (1987). The South Oaks Gambling Screen (SOGS): A new instrument for the identification of pathological gamblers. *American Journal of Psychiatry, 144*(9), 1184–1188.

Lesieur, H. R., & Blume, S. B. (1992). Revising the South Oaks Gambling Screen in different settings. *Journal of Gambling Behavior, 9*(3), 213–219.

Lesieur, H. R., & Blume, S. B. (1993). Pathological gambling, eating disorders, and the psychoactive substance use disorders. *Journal of Addictive Disease, 12*(3), 89–102.

Lesieur, H. R., & Rosenthal, R. J. (1991). Pathological gambling: A review of the literature (prepared for the American Psychiatric Association Task Force on DSM-IV Committee on Disorders of Impulse Control Not Elsewhere Classified). *Journal of Gambling Studies, 7*(1), 5–39.

Lobo, D. S., & Kennedy, J. L. (2009). Genetic aspects of pathological gambling: A complex disorder with shared genetic vulnerabilities. *Addiction, 104*(9), 1454–1465.

Magalhaes, A. C., Jungerman, F. S., Silva, M. C., Moraes, M. M., & Tavares, H. (2009). Post-therapy group for pathological gamblers: Improvement beyond symptoms. *Revista Brasileira de Psiquiatria, 31*(2), 181–182.

Marlatt, G., & Gordon, J. R. (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: Guilford Press.

Martins, S. S., Lobo, D. S., Tavares, H., & Gentil, V. (2002). Pathological gambling in women: A review. *Revista do Hospital das Clinicas da Facudade de Medicina de São Paulo, 57*(5), 235–242.

Martins, S. S., Tavares, H., da Silva Lobo, D. S., Galetti, A. M., & Gentil, V. (2004). Pathological gambling, gender, and risk-taking behaviors. *Addiction Behavior, 29*(6), 1231–1235.

Mazzoleni, M. H., Gorenstein, C., Fuentes, D., & Tavares, H. (2009). Wives of pathological gamblers: Personality traits, depressive symptoms and social adjustment. *Revista Brasileira de Psiquiatria, 31*(4), 332–337.

McConaghy, N., Armstrong, M. S., Blaszczynski, A., & Allcock, C. (1983). Controlled comparison of aversive therapy and imaginal desensitization in compulsive gambling. *British Journal of Psychiatry, 142*, 366–372.

McConaghy, N., Blaszczynski, A., & Frankova, A. (1991). Comparison of imaginal desensitisation with other behavioural treatments of pathological gambling. A two- to nine-year follow-up. *British Journal of Psychiatry, 159*, 390–393.

McElroy, S. L., Nelson, E. B., Welge, J. A., Kaehler, L., & Keck, P. E., Jr. (2008). Olanzapine in the treatment of pathological gambling: A negative randomized placebo-controlled trial. *Journal of Clinical Psychiatry, 69*(3), 433–440.

McGrath, D. S., & Barrett, S. P. (2009). The comorbidity of tobacco smoking and gambling: A review of the literature. *Drug and Alcohol Review, 28*(6), 676–681.

Melville, K. M., Casey, L. M., & Kavanagh, D. J. (2007). Psychological treatment dropout among pathological gamblers. *Clinical Psychology Review, 27*(8), 944–958.

Meyer, G., Hauffa, B. P., Schedlowski, M., Pawlak, C., Stadler, M. A., & Exton, M. S. (2000). Casino gambling increases heart rate and salivary cortisol in regular gamblers. *Biological Psychiatry, 48*(9), 948–953.

Molina, J. A., Sainz-Artiga, M. J., Fraile, A., Jimenez-Jimenez, F. J., Villanueva, C., Orti-Pareja, M., et al. (2000). Pathologic gambling in Parkinson's disease: A behavioral manifestation of pharmacologic treatment? *Movement Disorders, 15*(5), 869–872.

Monaghan, S., Blaszczynski, A., & Nower, L. (2009). Consequences of winning: The role of gambling outcomes in the development of irrational beliefs. *Behavior and Cognitive Psychotherapy, 37*(1), 49–59.

Morasco, B. J., Pietrzak, R. H., Blanco, C., Grant, B. F., Hasin, D., & Petry, N. M. (2006). Health problems and medical utilization associated with gambling disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Psychosomatic Medicine, 68*(6), 976–984.

Nelson, S. E., Gebauer, L., Labrie, R. A., & Shaffer, H. J. (2009). Gambling problem symptom patterns and stability across individual and timeframe. *Psychology of Addiction Behavior, 23*(3), 523–533.

Nelson, S. E., Laplante, D. A., Labrie, R. A., & Shaffer, H. J. (2006). The proxy effect: Gender and gambling problem trajectories of Iowa gambling treatment program participants. *Journal of Gambling* Studies, *22*(2), 221–240.

Newman, S. C., & Thompson, A. H. (2007). The association between pathological gambling and attempted suicide: Findings from a national survey in Canada. *Canadian Journal of Psychiatry, 52*(9), 605–612.

Nicolato, R., Romano-Silva, M. A., Correa, H., Salgado, J. V., & Teixeira, A. L. (2007). Lithium and topiramate association in the treatment of comorbid pathological gambling and bipolar disorder. *Australia and New Zealand Journal of Psychiatry, 41*(7), 628.

Nordin, C., & Nylander, P. O. (2007). Temperament and character in pathological gambling. *Journal of Gambling Studies, 23*(2), 113–120.

Oei, T. P., & Gordon, L. M. (2008). Psychosocial factors related to gambling abstinence and relapse in members of Gamblers Anonymous. *Journal of Gambling Studies, 24*(1), 91–105.

Pagani, L. S., Derevensky, J. L., & Japel, C. (2009). Predicting gambling behavior in sixth grade from kindergarten impulsivity: A tale of developmental continuity. *Archives of Pediatric and Adolescent Medicine, 163*(3), 238–243.

Pallanti, S., Quercioli, L., Sood, E., & Hollander, E. (2002). Lithium and valproate treatment of pathological gambling: A randomized single-blind study. *Journal of Clinical Psychiatry, 63*(7), 559–564.

Pallesen, S., Mitsem, M., Kvale, G., Johnsen, B. H., & Molde, H. (2005). Outcome of psychological treatments of pathological gambling: A review and meta-analysis. *Addiction, 100*(10), 1412–1422.

Pallesen, S., Molde, H., Arnestad, H. M., Laberg, J. C., Skutle, A., Iversen, E., et al. (2007). Outcome of pharmacological treatments of pathological gambling: A review and meta-analysis. *Journal of Clinical Psychopharmacology, 27*(4), 357–364.

Park, S., Cho, M. J., Jeon, H. J., Lee, H. W., Bae, J. N., Park, J. I., et al. (2010). Prevalence, clinical correlations, comorbidities, and suicidal tendencies in pathological Korean gamblers: Results from the Korean Epidemiologic Catchment Area Study. *Social Psychiatry and Psychiatric Epidemiology, 45*(6), 621–629.

Pasternak, A. V. T., & Fleming, M. F. (1999). Prevalence of gambling disorders in a primary care setting. *Archives of Family Medicine, 8*(6), 515–520.

Petry, N. M. (2001). Pathological gamblers, with and without substance use disorders, discount delayed rewards at high rates. *Journal of Abnormal Psychology, 110*(3), 482–487.

Petry, N. M. (2005). Stages of change in treatment-seeking pathological gamblers. *Journal of Consulting Clinical Psychology, 73*(2), 312–322.

Petry, N. M. (2006). Should the scope of addictive behaviors be broadened to include pathological gambling? *Addiction, 101*(Suppl 1), 152–160.

Petry, N. M. (2007). Concurrent and predictive validity of the Addiction Severity Index in pathological gamblers. *American Journal of Addiction, 16*(4), 272–282.

Petry, N. M., Ammerman, Y., Bohl, J., Doersch, A., Gay, H., Kadden, R., et al. (2006). Cognitive-behavioral therapy for pathological gamblers. *Journal of Consulting Clinical Psychology, 74*(3), 555–567.

Petry, N. M., & Kiluk, B. D. (2002). Suicidal ideation and suicide attempts in treatment-seeking pathological gamblers. *Journal of Nervous and Mental Disease, 190*(7), 462–469.

Petry, N. M., Litt, M. D., Kadden, R., & Ledgerwood, D. M. (2007). Do coping skills mediate the relationship between cognitive-behavioral therapy and reductions in gambling in pathological gamblers? *Addiction, 102*(8), 1280–1291.

Petry, N. M., Stinson, F. S., & Grant, B. F. (2005). Comorbidity of *DSM-IV* pathological gambling and other psychiatric disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 66*(5), 564–574.

Petry, N. M., Weinstock, J., Ledgerwood, D. M., & Morasco, B. (2008). A randomized trial of brief interventions for problem and pathological gamblers. *Journal of Consulting Clinical Psychology, 76*(2), 318–328.

Pietrzak, R. H., & Petry, N. M. (2005). Antisocial personality disorder is associated with increased severity of gambling, medical, drug and psychiatric problems among treatment-seeking pathological gamblers. *Addiction, 100*(8), 1183–1193.

Potenza, M. N. (2009). Non-substance and substance addictions. *Addiction, 104*(6), 1016–1017.

Potenza, M. N., Fiellin, D. A., Heninger, G. R., Rounsaville, B. J., & Mazure, C. M. (2002). Gambling: An addictive behavior with health and primary care implications. *Journal of General Internal Medicine, 17*(9), 721–732.

Potenza, M. N., Steinberg, M. A., McLaughlin, S. D., Wu, R., Rounsaville, B. J., & O'Malley, S. S. (2001). Gender-related differences in the characteristics of problem gamblers using a gambling helpline. *American Journal of Psychiatry, 158*(9), 1500–1505.

Rodriguez-Jimenez, R., Avila, C., Jimenez-Arriero, M. A., Ponce, G., Monasor, R., Jimenez, M., et al. (2006). Impulsivity and sustained attention in pathological gamblers: Influence of childhood ADHD history. *Journal of Gambling Studies*, *22*(4), 451–461.

Rosecrance, J. (1986). Attributions and the origins of problem gambling. *Sociological Quarterly, 27*(4), 463–477.

Rosengren, A., Hawken, S., Ounpuu, S., Sliwa, K., Zubaid, M., Almahmeed, W. A., et al. (2004). Association of psychosocial risk factors with risk of acute myocardial infarction in 11,119 cases and 13,648 controls from 52 countries (the INTERHEART study): Case-control study. *Lancet, 364*(9438), 953–962.

Rosenthal, R. J. (1987). The psychodynamics of pathological gambling: A review of the literature. In T. Galski (Ed.), *The handbook of pathological gambling* (pp. 41–70). Springfield, IL: Charles C Thomas.

Rosenthal, R. J. (2008). [Psychodynamic psychotherapy and the treatment of pathological gambling]. *Revista Brasileira de Psiquiatria, 30*(Suppl 1), S41–50.

Rosenthal, R. J., & Lesieur, H. R. (1992). Self-reported withdrawal symptoms and pathological gambling. *American Journal on Addictions, 1*, 150–154.

Saiz-Ruiz, J., Blanco, C., Ibanez, A., Masramon, X., Gomez, M. M., Madrigal, M., et al. (2005). Sertraline treatment of pathological gambling: A pilot study. *Journal of Clinical Psychiatry, 66*(1), 28–33.

Sander, W., & Peters, A. (2009). Pathological gambling: Influence of quality of life and psychological distress on abstinence after cognitive-behavioral inpatient treatment. *Journal of Gambling Studies, 25*(2), 253–262.

Sartor, C. E., Scherrer, J. F., Shah, K. R., Xian, H., Volberg, R., & Eisen, S. A. (2007). Course of pathological gambling symptoms and reliability of the Lifetime Gambling History measure. *Psychiatry Research, 152*(1), 55–61.

Scherrer, J. F., Xian, H., Kapp, J. M., Waterman, B., Shah, K. R., Volberg, R., et al. (2007). Association between exposure to childhood and lifetime traumatic events and lifetime pathological gambling in a twin cohort. *Journal of Nervous and Mental Disease, 195*(1), 72–78.

Seedat, S., Kesler, S., Niehaus, D. J., & Stein, D. J. (2000). Pathological gambling behaviour: Emergence secondary to treatment of Parkinson's disease with dopaminergic agents. *Depression and Anxiety, 11*(4), 185–186.

Shaffer, H. J., Freed, C. R., & Healea, D. (2002). Gambling disorders among homeless persons with substance use disorders seeking treatment at a community center. *Psychiatric Service, 53*(9), 1112–1117.

Shaffer, H. J., & Kidman, R. (2003). Shifting perspectives on gambling and addiction. *Journal of Gambling Studies, 19*(1), 1–6.

Sharkey, S. W., Lesser, J. R., Zenovich, A. G., Maron, M. S., Lindberg, J., Longe, T. F., et al. (2005). Acute and reversible cardiomyopathy provoked by stress in women from the United States. *Circulation, 111*(4), 472–479.

Sharpe, L., & Tarrier, N. (1993). Towards a cognitive-behavioural theory of problem gambling. *British Journal of Psychiatry, 162*, 407–412.

Slutske, W. S. (2006). Natural recovery and treatment-seeking in pathological gambling: Results of two U.S. national surveys. *American Journal of Psychiatry, 163*(2), 297–302.

Slutske, W. S., Blaszczynski, A., & Martin, N. G. (2009). Sex differences in the rates of recovery, treatment-seeking, and natural recovery in pathological gambling: Results from an Australian community-based twin survey. *Twin Research and Human Genetics, 12*(5), 425–432.

Slutske, W. S., Caspi, A., Moffitt, T. E., & Poulton, R. (2005). Personality and problem gambling: A prospective study of a birth cohort of young adults. *Archives of General Psychiatry, 62*(7), 769–775.

Slutske, W. S., Jackson, K. M., & Sher, K. J. (2003). The natural history of problem gambling from age 18 to 29. *Journal of Abnormal Psychology, 112*(2), 263–274.

Specker, S. M., Carlson, G. A., Christenson, G. A., & Marcotte, M. (1995). Impulse control disorders and attention deficit disorder in pathological gamblers. *Annals of Clinical Psychiatry, 7*(4), 175–179.

Spunt, B. (2002). Pathological gambling and substance misuse. *Substance Use and Misuse, 37*(8–10), 1299–1304.

Steenbergh, T. A., Meyers, A. W., May, R. K., & Whelan, J. P. (2002). Development and validation of the Gamblers' Beliefs Questionnaire. *Psychology of Addiction Behavior*, *16*(2), 143–149.

Stewart, R. M., & Brown, R. I. (1988). An outcome study of Gamblers Anonymous. *British Journal of Psychiatry, 152*, 284–288.

Stinchfield, R. (2002). Reliability, validity, and classification accuracy of the South Oaks Gambling Screen (SOGS). *Addiction Behavior, 27*(1), 1–19.

Sullivan, S., Arroll, B., Coster, G., Abbott, M., & Adams, P. (2000). Problem gamblers: Do GPs want to intervene? *New Zealand Medical Journal, 113*(1111), 204–207.

Suurvali, H., Cordingley, J., Hodgins, D. C., & Cunningham, J. (2009). Barriers to seeking help for gambling problems: A review of the empirical literature. *Journal of Gambling Studies, 25*(3), 407–424.

Suurvali, H., Hodgins, D. C., & Cunningham, J. A. (2010). Motivators for resolving or seeking help for gambling problems: A review of the empirical literature. *Journal of Gambling Studies*, *26*(1), 1–33.

Tavares, H., Carneiro, E., Sanches, M., Pinsky, I., Caetano, R., Zaleski, M., Laranjeira, R. (2010). Gambling in Brazil: Lifetime prevalences and socio-demographic correlates. *Psychiatry Research, 180*(1), 35–41.

Tavares, H., & Gentil, V. (2007). Pathological gambling and obsessive-compulsive disorder: Towards a spectrum of disorders of volition. *Revista Brasileira de Psiquiatria, 29*(2), 107–117.

Tavares, H., Martins, S. S., Lobo, D. S., Silveira, C. M., Gentil, V., & Hodgins, D. C. (2003a). Factors at play in faster progression for female pathological gamblers: An exploratory analysis. *Journal of Clinical Psychiatry, 64*(4), 433–438.

Tavares, H., Martins, S. S., Zilberman, M. L., & el-Guebaly, N. (2002). Gamblers and treatment-seeking: Why haven't they come earlier? *Addiction Disorders and Their Treatment, 1*(2), 65–70.

Tavares, H., Zilberman, M. L., Beites, F. J., & Gentil, V. (2001). Gender differences in gambling progression. *Journal of Gambling Studies, 17*(2), 151–159.

Tavares, H., Zilberman, M. L., & el-Guebaly, N. (2003b). Are there cognitive and behavioural approaches specific to the treatment of pathological gambling? *Canadian Journal of Psychiatry, 48*(1), 22–27.

Tavares, H., Zilberman, M. L., Hodgins, D. C., & el-Guebaly, N. (2005). Comparison of craving between pathological gamblers and alcoholics. *Alcohol Clinics in Experimental Research, 29*(8), 1427–1431.

Toce-Gerstein, M., Gerstein, D. R., & Volberg, R. A. (2003). A hierarchy of gambling disorders in the community. *Addiction, 98*(12), 1661–1672.

Tolchard, B., Thomas, L., & Battersby, M. (2007). GPs and problem gambling: Can they help with identification and early intervention? *Journal of Gambling Studies, 23*(4), 499–506.

Toneatto, T., Blitz-Miller, T., Calderwood, K., Dragonetti, R., & Tsanos, A. (1997). Cognitive distortions in heavy gambling. *Journal of Gambling Studies, 13*(3), 253–266.

Toneatto, T., Brands, B., & Selby, P. (2009). A randomized, double-blind, placebo-controlled trial of naltrexone in the treatment of concurrent alcohol use disorder and pathological gambling. *American Journal of Addiction, 18*(3), 219–225.

Toneatto, T., Cunningham, J., Hodgins, D. C., Adams, M., Turner, N., & Koski-Jannes, A. (2008). Recovery from problem gambling without formal treatment. *Addiction Research Theory, 16*(2), 111–120.

Toneatto, T., & Gunaratne, M. (2009). Does the treatment of cognitive distortions improve clinical outcomes for problem gambling?. *Journal of Contemporary Psychotherapy, 39*(4), 221–229.

Toneatto, T., & Wang, J. J. (2009). Community treatment for problem gambling: Sex differences in outcome and process. *Community Mental Health Journal, 45*(6), 468–475.

Vachon, D. D., & Bagby, R. M. (2009). Pathological gambling subtypes. *Psychological Assessment, 21*(4), 608–615.

Valenzuela, T. D., Roe, D. J., Nichol, G., Clark, L. L., Spaite, D. W., & Hardman, R. G. (2000). Outcomes of rapid defibrillation by security officers after cardiac arrest in casinos. *New England Journal of Medicine, 343*(17), 1206–1209.

Vitaro, F., Arseneault, L., & Tremblay, R. E. (1999). Impulsivity predicts problem gambling in low SES adolescent males. *Addiction, 94*(4), 565–575.

Volpicelli, J. R., Alterman, A. I., Hayashida, M., & O'Brien, C. P. (1992). Naltrexone in the treatment of alcohol dependence. *Archives of General Psychiatry, 49*(11), 876–880.

Voon, V., Fernagut, P. O., Wickens, J., Baunez, C., Rodriguez, M., Pavon, N., et al. (2009). Chronic dopaminergic stimulation in Parkinson's disease: From dyskinesias to impulse control disorders. *Lancet Neurology, 8*(12), 1140–1149.

Walker, M., Toneatto, T., Potenza, M. N., Petry, N., Ladouceur, R., Hodgins, D. C., et al. (2006). A framework for reporting outcomes in problem gambling treatment research: The Banff, Alberta, Consensus. *Addiction, 101*(4), 504–511.

Welte, J. W., Barnes, G. M., Wieczorek, W. F., & Tidwell, M. C. (2004). Gambling participation and pathology in the United States—a sociodemographic analysis using classification trees. *Addiction Behavior, 29*(5), 983–989.

Welte, J. W., Barnes, G. M., Wieczorek, W. F., Tidwell, M. C., & Parker, J. (2002). Gambling participation in the U.S.—results from a national survey. *Journal of Gambling Studies, 18*(4), 313–337.

Wilkes, S. (2006). Bupropion. *Drugs Today* (*Barcelona*)*, 42*(10), 671–681.

Williams, W. A., & Potenza, M. N. (2008). [The neurobiology of impulse control disorders]. *Revista Brasileira de Psiquiatria, 30*(Suppl 1), S24–S30.

Winters, K. S., & Fulkerson, J. (1993). Toward the development of an adolescent gambling problem severity scale. *Journal of Gambling Studies, 9*, 63–84.

Winters, K. S. R., & Kim, L. (1995). Monitoring adolescent gambling in Minnesota. *Journal of Gambling Studies, 11*, 165–183.

Wood, W. S., & Clapham, M. M. (2005). Development of the Drake Beliefs about Chance inventory. *Journal of Gambling Studies, 21*(4), 411–430.

Wulfert, E., Blanchard, E. B., Freidenberg, B. M., & Martell, R. S. (2006). Retaining pathological gamblers in cognitive behavior therapy through motivational enhancement: A pilot study. *Behavior Modification, 30*(3), 315–340.

Young, M. M., & Wohl, M. J. (2009). The Gambling Craving Scale: Psychometric validation and behavioral outcomes. *Psychology of Addiction Behavior*, *23*(3), 512–522.

Zack, M., & Poulos, C. X. (2009a). Effects of the atypical stimulant modafinil on a brief gambling episode in pathological gamblers with high vs. low impulsivity. *Journal of Psychopharmacology, 23*(6), 660–671.

Zack, M., & Poulos, C. X. (2009b). Parallel roles for dopamine in pathological gambling and psychostimulant addiction. *Current Drug Abuse Review, 2*(1), 11–25.

Zimmerman, M., Breen, R. B., & Posternak, M. A. (2002). An open-label study of citalopram in the treatment of pathological gambling. *Journal of Clinical Psychiatry, 63*(1), 44–48.

Zimmerman, M., Chelminski, I., & Young, D. (2006). A psychometric evaluation of the *DSM-IV* pathological gambling diagnostic criteria. *Journal of Gambling* Stud*ies, 22*(3), 329–337.

CHAPTER 23

The Assessment and Treatment of Trichotillomania

Gary A.-H. Christenson

Abstract

The successful treatment of trichotillomania necessitates an initial clinical evaluation of the cognitive, emotional, behavioral, and situational characteristics contributing to an individual's hair pulling. Assessment also requires a comprehensive psychological/psychiatric interview to assess for comorbid illness, which may either contribute to hair pulling or require separate attention. Several instruments have been designed to assist in quantifying the core symptoms of trichotillomania and can be useful for monitoring treatment progress over time. Treatment approaches include medication, hypnosis, and behavioral therapies, especially modifications of habit reversal therapy. Controlled studies are few in number and are limited to only a few behavioral treatment approaches and medication classes. Research suggests that variations of habit reversal therapy have the greatest efficacy of the interventions investigated thus far. There is additional support for treatment with clomipramine, *N*-acetylcysteine, and olanzapine, in contrast to multiple other drugs that have been studied or suggested as useful for trichotillomania.

Keywords: assessment, behavioral therapy, clomipramine, cognitive behavioral therapy, habit reversal, hair pulling, hypnosis, olanzapine, naltrexone, N-acetylcysteine, SSRI, SNRI, trichotillomania

Introduction

Although trichotillomania has been clinically recognized for over a century (Hallopeau, 1889), the literature on this disorder remains scant compared to that of other psychological, psychiatric, and behavioral disorders. Increased interest in trichotillomania arose in the late 1980s out of the consideration of its possible connection to obsessive-compulsive disorder (OCD; see the chapter "Phenomenology and Epidemiology of Trichotillomania") and the realization that trichotillomania is more prevalent than was previously recognized (Christenson et al., 1991d). The conceptualization of trichotillomania as a possible variant of OCD influenced both the initial approach to assessment as well as investigations of pharmacological treatment. In contrast, the preferred behavioral treatments for the two disorders diverged, with exposure/response-prevention having been established as the most effective intervention for most manifestations of OCD (Abramowitz, 1997), whereas habit reversal therapy became the standard treatment for trichotillomania (Keuthen et al., 1999). However, further research has suggested that these two disorders may not be as closely related as was initially hypothesized and likely require different pharmacological as well as behavioral approaches. As with any condition, the effectiveness of any treatment intervention depends on a thorough clinical assessment as well as comprehension of the treatment literature.

Assessment

Clinical Assessment

Appropriate clinical assessment of trichotillomania will guide the choice of treatment intervention and includes an assessment of hair pulling itself as well

as coexisting psychopathology. Additional variables such as age, intellectual and emotional maturity, motivation, treatment preference, and likelihood for compliance also factor into the treatment choice and may be predictive of the treatment response.

An adequate assessment of hair-pulling behavior should include a survey of all potential hair-pulling sites including the scalp, brow, lashes, facial hair, pubic hair, and extremity hair. Pulling hair from other persons, pets, and inanimate objects, although rare, should also be considered (Christenson et al., 1991a). More specific inquiries about the distribution of hair pulling within sites, particularly the scalp, may provide useful information to guide individualization of various forms of therapy. For example, pulling from the nondominant side of the scalp may suggest that certain activities involving the dominant hand (e.g., writing) may be high-risk situations for pulling out hair for that individual. Alternatively, symmetrical hair pulling may suggest that certain compulsive rules may be followed (Christenson et al, 1991a). Inquiries about the cognitions, emotions, and sensory experiences associated with an individual's hair pulling may suggest that other conditions, such as anxiety or depression, may be fueling the disorder or that a person's hair pulling may be more phenomenologically similar to OCD. Specific cues such as pruritis or scalp tingling may initiate the behavior (Christenson et al, 1991a). Alternatively, there may be little awareness of hair pulling so that efforts targeting increased awareness may be essential for improvement. Tactile and visual cues such as thick, wiry, or gray hairs should be noted (Christenson et al., 1991a). Although the current criteria for trichotillomania as stated by the American Psychiatric Association (2000) require the presence of mounting tension when pulling out hair or while attempting to resist the behavior, as well as pleasure, gratification, or relief when pulling out hair, the clinician will encounter circumstances in which these reactions are absent. This is particularly true in younger patients (Reeve et al., 1992). Most treatment studies of hair pulling have included individuals who do not meet all of these experiential criteria (Christenson & Crow, 1996).

The clinical assessment of hair pulling should include an inquiry into activities most likely to be associated with this practice. These are often activities that require sustained attention but limited physical activity such as reading, driving, watching television, or speaking on the phone (Christenson et al., 1995). However, hair pulling may also occur in the context of both physical and mental relaxation. These attributes of hair pulling are particularly useful to note prior to embarking on behavioral interventions. As many hair pullers engage in some kind of oral manipulation of hair and some actually ingest their hair (trichophagy), it is important to inquire about this specific behavior. The presence of hair ingestion should prompt a review of gastrointestinal symptoms and appropriate additional evaluation if there is any suggestion of the presence of a trichobezoar (hair ball) in the stomach or intestines (see the chapter "Phenomenology and Epidemiology of Trichotillomania")

Comorbidity with anxiety, depression, OCD, and other psychiatric symptoms is common in trichotillomania (see the chapter "Phenomenology and Epidemiology of Trichotillomania"), so assessment should always include a thorough psychiatric review of symptoms. Comorbid conditions are likely to influence the treatment intervention (e.g., a serotonin reuptake inhibitor in the presence of OCD).

The level of awareness of hair pulling may be a useful distinction during the assessment of trichotillomania. Two phenomenologically distinct patterns of hair pulling have been suggested in both adults and children (Christenson et al, 1995; Flessner et al, 2008b). One is more *focused*, compulsive, and clearly within the hair puller's awareness. The other, usually referred to as *automatic*, is more habitual, less likely to be the center of the hair puller's attention, and generally occurs simultaneously in the presence of another focused activity such as reading, driving, or speaking on the phone. Many hair pullers describe varying degrees of these two qualitatively dissimilar hair-pulling styles. One study of 47 patients with trichotillomania revealed that 32% were primarily automatic pullers and 15% were mostly focused pullers. Various combinations of the two styles accounted for the other 53% of subjects (Du Toit et al., 2001). It has been hypothesized that focused hair pulling may be better targeted by medication interventions, whereas automatic hair pulling may be more responsive to the application of behavioral techniques (Christenson et al., 1995). Others have suggested that the two different styles can both be addressed with behavioral therapy but the specific style may dictate the application of contrasting behavioral techniques (Flessner et al., 2008b).

Assessment of Severity and Change

Several strategies for the monitoring of hair-pulling severity and progress over time have been suggested. These have included measuring hair length, counting

the number of hairs in a randomly sampled area, counting the number of bald spots, and assessment of visual change over time by comparison of photographs at baseline and posttreatment (Rothbaum et al., 1999). The variability of hair-pulling sites over time, as well as challenges in interpreting photographs in the context of more broadly distributed hair thinning in particular, are likely to render these specific approaches unsatisfactory for most clinical situations. Self-monitoring techniques are more likely to prove useful as long as the patient is adequately motivated and compliant. In addition, self-monitoring itself may reduce hair pulling (Stanley et al., 1991). Self-monitoring usually involves some variation on daily entries into a hair-pulling log that may include such variables as time of episode, number of hairs pulled, urges to pull out hair, associated circumstances, and other behavioral, emotional, and cognitive variables (Rothbaum et al., 1999).

Instruments Measuring Trichotillomania Symptom Change

A number of instruments have been developed to assess the baseline severity of trichotillomania and to monitor symptom change over time. Although the initial development and application of these instruments were for research purposes, many instruments are easily adaptable to clinical settings.

The Massachusetts General Hospital Hairpulling Scale

The Massachusetts General Hospital Hairpulling Scale (MGH-HPS; Keuthen et al., 1995) is the most extensively researched instrument to assess the severity of hair pulling. The MGH-HPS was modeled on the Yale Brown Obsessive Compulsive Scale, an instrument that has also been modified by other researchers of trichotillomonia (Stanley et al., 1993). The MGH-HPS has been documented to have good internal consistency, test-retest reliability, covergent validity, and divergent validity (Keuthen et al., 1995; O'Sullivan et al., 1995). This self-administered scale consists of seven questions concerned hair-pulling symptoms experienced within the preceding week. Each question is rated 0–4, with a total score range of 0–28. Individual questions target frequency of hair-pulling urges, intensity of urges, ability to control urges, hair-pulling frequency, attempts to resist hair pulling, control over hair pulling, and associated distress. Factor analysis of the instrument (Keuthen et al, 2007a) revealed two separate factors: (1) severity and (2) resistance and control. Attention to these factors may be clinically useful. For example, less progress over time on the resistance and control factor may reveal motivational issues that can then be more strongly addressed in therapy (Keuthen et al., 2007a).

NIMH scales

The National Institute of Mental Health (NIMH) Trichotillomania scales (Swedo et al., 1989) have been frequently used in trichotillomania research. The Trichotillomania Symptom Severity Scale (NIMH-TSS) consists of six questions addressing various aspects of hair-pulling behavior, including average daily amount of time spent engaged in hair pulling, amount of time consumed by hair pulling, thoughts or feelings preceding hair-pulling episodes, resistance to pulling out hair, degree of distress resulting from hair pulling, and interference of hair pulling with daily functioning. Severity for each question is rated from 0 to 5.

The NIMH Trichotillomania Impairment Scale (NIMH-TIS) is a clinician-rated 10-point scale with descriptors of impairment to guide the clinician in assigning severity in ranges labeled as 0 (none), 1–3 (minimal), 4–6 (mild), and 7–10 (moderate/severe). The NIMH Physician Rating of Clinical Progress is a 20-point Likert scale with three anchors at 0 (Cured), 10 (Baseline), and 20 (Worst ever imaginable).

Together, the NIMH scales have demonstrated good interrater reliability (Swedo et al, 1989). However, the NIMH-TSS has shown poor correspondence with self-reported trichotillomania severity and degree of alopecia (Diefenbach et al., 2005).

The Psychiatric Institute Trichotillomania Scale

The Psychiatric Institute Trichotillomania Scale (PITS; Winchel et al., 1992a) was developed as a structured clinician-rated instrument. It includes an introductory interview that inquires about the age of onset of hair pulling, consistency/variability of hair pulling, duration of the current period of hair pulling, sites of hair pulling, and variation in sites over time, as well as methods used to hide or disguise hair loss. The scale itself includes six questions that inquire about the number of active hair-pulling sites (e.g., scalp and lashes), amount of daily time spent in the preceding week thinking about or pulling out hair, inability to resist hair pulling, avoidance of activities due to hair pulling, resultant distress, and severity of hair loss as assessed by visual inspection. Each content area is rated as 0–7, with total scores ranging from 0 to 42. The PITS has been

criticized as having somewhat arbitrary anchors for some of its ratings (Rothbaum et al., 1999) as well as poor internal consistency (Diefenbach, et al., 2005). Ratings using the PITS have been shown to strongly correlate with the MGH-HPS (O'Sullivan et al., 1995), suggesting that it remains a useful instrument for monitoring change in trichotillomania over time.

TRICHOTILLOMANIA SCALE FOR CHILDREN

The Trichotillomania Scale for Children (Tolin et al., 2008) was developed to provide an instrument for the specific assessment of hair pulling in children. Two versions, one for children (TSC-C) and one for parents (TSC-P), aim to address potential limitations that might be expected from either information source and/or due to the inability of children to participate in the scoring of their hair pulling. The 12 questions on the TSC-C and TSC-P parallel each other in content but are worded differently to reflect the standpoint of the rater. Five severity items target frequency of hair-pulling urges and hair-pulling behavior, duration of hair-pulling episodes, number of hairs pulled, and sense of control over hair pulling. Seven distress/impairment items inquire about the effect of hair pulling on preparation to enter social settings, avoidance of social activities, perceived effect on appearance, guilt feelings, sense of embarrassment, self-frustration, and related affect. The child or parent selects statements that best describe the characteristics of hair pulling during the previous week. Each statement is assigned a value from 0 to 2. In general, most items have three statements to select from. However, three items have 4–13 possible statements that capture a broader range of situations and events that might be experienced, described, or observed; in these cases, the scorer rates the item from 0 to 2 based on the highest score within that question set. The sums of the five severity items and the seven distress impairment items are separately averaged and the averages are added together for the total score, which is expressed in a range from 0 to 4. During its development, the TSC was shown to have good internal consistency, adequate test-retest reliability, and adequate convergence validity. In addition, agreement between the TSC-C and TSC-P was observed. However, the TSC has yet to be evaluated for sensitivity to change over time (Tolin et al., 2008).

As documented above, the clinician or researcher has several scales to choose from. However, the MGH-HPS has become preferred over other instruments due to its systematic development, brevity, and similarity of format to self-administered instruments used for other clinical conditions (Diefenbach et al., 2005).

Behavioral Treatment of Trichotillomania

Habit Reversal Therapy

Numerous behavioral approaches have been employed in the treatment of trichotillomania (Keuthan et al., 1999). However, both clinical practice and research have primarily focused on habit reversal therapy (HRT), a multicomponent treatment originally described by Azrin and Nunn (1973) and later expanded by these authors (Azrin et al., 1980). The 13 therapeutic components of HRT are (1) competing response training, (2) awareness training, (3) identification of response precursors, (4), identification of habit-prone situations, (5) relaxation training, (6) prevention training, (7) habit interruption, (8) positive attention/overcorrection, (9) competing reaction, (10) self-recording, (11) display of improvement, (12) social support, and (13) annoyance review. However, the majority of these components can be categorized within three broader treatment approaches: (1) substitution of and alternative response for hair-pulling, (2) increasing awareness of hair pulling, and (3) relaxation training.

One of the central components of HRT is the substitution of hair pulling for another incompatible behavior. The technique initially advocated by Azrin and Nunn was the formation of a tight fist with the hair-pulling hand, which is then held for 3 minutes (HRT component: competing response training). This competing response is applied whenever a patient is aware of pulling out hair or experiences an urge to pull out hair (HRT component: habit interruption) or in anticipation of hair pulling in the context of a situation frequently associated with hair pulling (HRT component: prevention training). This simple procedure disrupts hair pulling by preventing the fingers from grasping hair and also models the mounting tension and tension release often experienced by hair pullers. Some patients also note that the technique can lead to finger fatigue, which further discourages hair pulling. Once the competing response is mastered, patients are instructed to actively seek out situations in which hair pulling is likely to occur in order to further practice and demonstrate the effectiveness of this technique (HRT component: display of improvement).

The second core group of HRT components promotes increased awareness and more detailed

understanding of the sequential chain of behaviors that culminate in pulling out hair. Patients are instructed to become aware of the sequence of hand, arm, and postural motions that constitute their hair-pulling behavior (HRT component: awareness training). This awareness is further strengthened by the patient observing the hair-pulling behavior in front of a mirror. As the patient becomes more aware of precursor behaviors preceding the actual hair pull, he or she is able to better interrupt the hair-pulling sequence through improved observation of the proximity of the hands to hair, coupled with a decision to distance the hand, or by interjection of the competing reaction described above. Subjects are additionally trained to identify the immediate precursor to the hair pull (HRT component; identifying response precursors), which can be motoric (e.g., touching the hair) and/or tactilely cued (e.g., specifically encountering a coarse, thick, or kinky hair). Awareness is further broadened to identify the environmental and emotional contexts (HRT component: identifying habit-prone situations) in which hair pulling is likely to occur. These high-risk situations often include "sedentary contemplative" settings such as watching TV, driving, speaking on the phone, reading, or lying in bed (Christenson et al., 1991a). Emotional states often include nervousness, sadness, or boredom. To better facilitate the identification of these high-risk situations, patients maintain a daily record (HRT component: self-recording) of the time, situation, and emotional state of each hair-pulling episode. This latter technique also contributes to awareness of hair-pulling change over time due to the quantification of hair-pulling episodes and, if desired, an estimate or actual count of the hairs pulled.

Relaxation training is the third core component of HRT and includes education in deep breathing as well as postural adjustment. Additional aspects of HRT include the training and involvement of significant others to support the patient's efforts as well as to prompt the use of HRT techniques when hair pulling is observed (HRT component: social support). Positive interactions with hair (HRT component: overcorrection) such as hair brushing are also encouraged. Finally, a review of the negative consequences of hair pulling increases the motivation to stop pulling out hair. It is unclear which components of HRT are necessary for a treatment response. Simplified approaches using fewer components (discussed below) have been observed to be beneficial for some individuals.

The first controlled study of HRT in trichotillomania was a randomized comparison of HRT to negative practice (Azrin et al., 1980). The negative practice group was instructed to stand in front of a mirror every hour for 30 seconds while engaging in their usual motions of hair pulling but without actually pulling out hair. Nineteen subjects received HRT instruction during a single 2-hour session, while 15 were instructed in negative practice. Outcomes were reduction in the number of hair-pulling episodes or, in the case of continuous hair pulling, reduction in the amount of time spent pulling out hair. The results of the study favored HRT, with 90% of HRT subjects reporting improvement at 4 months. This compared to reductions of 52% to 68% in the negative practice group at 3-month follow-up. Follow-up by phone 22 months later revealed that 8 of 12 subjects in the HRT group were still not pulling out their hair. Although this was a landmark study in the treatment of trichotillomania, it has been criticized on several methodological grounds, including the lack of a waiting list control group that would assess for the influence of factors such as contact with a therapist and passage of time. Other criticisms include the absence of monitoring for treatment compliance, reliance on outcome measures primarily dependant on subject reports, and long-term follow-up conducted via phone (Rothbaum & Ninan, 1999). Variable reported endpoints also contribute to the challenges in interpreting this study. Finally, the negative practice condition, although demonstrably inferior to HRT, was still relatively effective for a majority of subjects instructed in this approach.

Several studies of behavioral therapy have modified HRT by adding cognitive or other therapeutic components. Ninan et al. (2000) conducted a 9-week placebo-controlled, randomized trial of cognitive behavioral therapy and clomipramine in 23 subjects (16 completers) with trichotillomania. Cognitive therapy included comprehensive HRT, which was augmented with additional stimulus control and stress management techniques. These included deep muscle relaxation, differential relaxation, breathing retraining, thought stopping, cognitive restructuring, guided self-dialogue, role playing, covert modeling (cognitive role playing), and relapse prevention. Subjects assigned to behavior therapy were treated in nine weekly 45-minute sessions. Cognitive therapy proved highly effective and significantly better than clomipramine treatment or placebo on clinician-administered measures of improvement. Additionally, all completers of cognitive

therapy were deemed to be responders (scored as very much improved or improved on the Clinical Global Scale of Improvement [CGI]) (Guy, 1976, as did 71% of intent-to-treat subjects; this compared to 67% and 40% of the clomipramine treatment group). Variations of cognitive therapy have been reported as effective when used as the primary treatment technique or in combination with other behavioral treatments for trichotillomania and have included identification and restructuring of maladaptive thoughts and beliefs, rational-emotive therapy, covert sensitization, cognitive desensitization, positive imagery, and thought stopping (Bernard et al., 1983; Bornstein & Rychtarik 1978; Gluhoski, 1995; Levine, 1976; Taylor, 1963).

Behavioral therapy has also been compared to the selective serotonin reuptake inhibitor (SSRI) fluoxetine for the treatment of trichotillomania utilizing a randomized design with a waiting list control (van Minnen et al., 2003). Forty subjects completed this 12-week study, with 14 completers randomized to behavioral therapy, 11 to medication, and 15 to waiting list control. Behavioral therapy was administered during six 45-minute sessions over the course of the 12 weeks. Behavioral treatment components included many of those characteristic of HRT but significantly modified from the original program of Azrin et al. (1980) in regard to selection of specific interventions (e.g., the competing response of making a fist was absent, and stimulus-response interventions consisted of more complex recreational or task-oriented activities). Behavioral strategies included behavioral chain identification and self-monitoring, both via logging of hair pulls and collecting hair in an envelope. Increased awareness of pulling was targeted via the introduction of sensory indicators, which varied by the individual. Targeted sensations included tactile (e.g., wearing bandages on the fingers), olfactory (e.g., placing heavy makeup on the wrist), and/or auditory (e.g., wearing tinkling bracelets on the hair-pulling arm) measures. Stimulus control was approached by organizing the environment to be less conducive to hair pulling. Incompatible behaviors were also introduced to interrupt the chain of hair-pulling behavior. Examples of substituted behaviors included going for a walk, going for a jog, calling a friend, and cleaning a room. Subjects were permitted to pull out hair if the urge to pull persisted provided that they had completed the substituted behavior and had experienced an associated delay in the hair-pulling behavioral chain. The majority of subjects also engaged in stimulus control by wearing gloves in high-risk situations. Some also administered self-rewards, although specific details about the types of rewards were not mentioned. Results of this study favored behavioral therapy over both medication treatment and waiting list control in regard to decreased symptoms of trichotillomania as well as clinically meaningful improvements. Sixty-four percent of the behaviorally treated group were deemed to have achieved a clinically significant change compared to 20% of the waiting list controls and only 9% of those receiving medication treatment. The authors concluded that behavioral therapy was highly effective in the treatment of trichotillomania, whereas fluoxetine was not.

A follow-up report of the above study examined the long-term benefits of behavioral therapy in 28 participants who were either initially randomized to behavioral treatment or who subsequently received behavioral treatment upon completion of the waiting list arm (Keijsers et al., 2006). Data were available for 24 subjects at 2 years posttreatment. A significant decline in hair-pulling symptoms was noted compared to baseline. However, an increase in hair pulling from treatment completion to 2-year follow-up was also observed. Only three subjects remained symptom free and only six had maintained an improvement rate of 50% or more. Two of the subjects reported more than a 30% increase in hair pulling at 2-year follow-up compared to pretreatment. This loss of benefits over time was also observed in an open trial of cognitive behavioral treatment of trichotillomania in which two-thirds of treatment responders relapsed at long-term follow-up (Lerner et al., 1998).

Habit reversal therapy has been combined with acceptance and commitment therapy (ACT) and compared to a wait-list control in a randomized behavioral treatment study of trichotillomania (Woods et al., 2006b). Acceptance and commitment therapy targets experiential avoidance by changing the cognitive orientation from one that supports emotional control strategies to one that promotes the acceptance of private feelings, thoughts, and urges (Hayes et al., 1999). Acceptance and commitment therapy was considered to be possibly useful for the treatment of trichotillomania based on survey results suggesting that hair-pulling severity increases with experiential avoidance (Begotka et al., 2004). In addition, an initial trial of ACT/HRT, also referred to as *acceptance enhanced behavior therapy* (AEBT), for the treatment of trichotillomania demonstrated effectiveness with this technique in four of six subjects (Twohig & Woods, 2004). Twelve subjects

completed the AEBT trial, as did 13 in the waiting list control. Acceptance enhanced behavior therapy was administered in 10 sessions over the 12 weeks of the study. Clinically significant improvement was observed in 66% of the treatment group compared to only 8% of the waiting list group. Impairment ratings of trichotillomania decreased by 33% in the treatment group but only by 6% in the waiting list group. Finally, the number of hairs pulled was reported to have decreased by 58% with AEBT, whereas this number actually increased by 28% in the waiting list group. Despite the study's support for AEBT, none of the participants experienced complete resolution of hair pulling. Additionally, posttreatment follow-up at 3 months revealed a significant increase in overall symptoms of trichotillomania since the end of treatment but no significant change in hair-pulling severity or number of hairs pulled per day. Further exploration of this technique in a small sample of patients with trichotillomania and chronic skin picking suggests that the clinical efficacy of AEBT is not dependant on the sequential ordering of the ACT and HRT components (Flessner et al., 2008a).

Bloch and colleagues (2007) conducted a meta-analysis of published studies of behavioral therapy that incorporated aspects of HRT versus control conditions. Three of the studies described above were included (Ninan et al., 2000; van Minnen et al., 2003; Woods et al., 2006b), with a fourth, the original study of Azrin et al. (1980), excluded due to lack of blinded clinical ratings of outcomes. The meta-analysis of data from the 59 completers demonstrated significant benefits of HRT-based behavioral treatment when compared to placebo or waiting list control.

The above-mentioned studies primarily included adults with trichotillomania. The literature on the behavioral treatment of children has generally been limited to case reports and case series using a variety of behavioral approaches (reviewed below). An open trial of cognitive behavioral therapy in children suggests that approaches similar to those employed in treating adults may be useful in this younger population. Tolin et al. (2007) enrolled 22 children and adolescents (mean age, 12.6 years) in a two-phase study of manual-based cognitive behavioral therapy for trichotillomania. The first phase consisted of active treatment administered weekly over 2 months. Individual sessions focused on progressive acquisition of knowledge and skills and included psychoeducation, competing response training, stimulus control, progressive muscle relaxation, cognitive restructuring, guided self-dialogue and relapse prevention strategies including covert modeling. The second phase concentrated on relapse prevention and consisted of four biweekly sessions and brief intersession phone contacts with the therapist. During this phase, children were reminded of the various strategies acquired during the active treatment phase. The strategies employed by the child or adolescent were also reviewed, with guidance for troubleshooting when needed. Fourteen children completed the study. Based on changes in NIMH trichotillomania scales and Clinical Global Improvement Scales, 77% of subjects were classified as responders. However, at 6-month follow-up, only 63% continued to be responders, representing a loss of one-quarter of the responders. Of interest, the researches classified 32% of subjects as "excellent responders," and none of this group lost their response at follow-up.

HRT Group Therapy

Diefenbach and colleagues (2006) compared HRT-based behavioral group therapy to supportive group therapy for the treatment of trichotillomania. Twelve subjects were randomly assigned to each group and compared to a waiting period consisting of the time necessary for groups to form. The subjects in the behavioral group experienced significantly greater reductions in hair pulling and observable hair loss compared to the support therapy group based on both a self-assessment instrument and clinical ratings. Both treatments demonstrated improvements compared to the waiting period. However, only 17% of subjects in the behavioral group treatment and 25% of those in the supportive group treatment were reported to have met predetermined criteria for clinically meaningful improvement. Substantial relapse was noted during the successive 6-month posttreatment follow-up. The authors concluded that the study only provided partial support of treatment with HRT-based behavioral therapy in the group format.

Other Behavioral Techniques

Habit reversal therapy with or without modification may not be appropriate for everyone seeking treatment for trichotillomania. Motivation, practice, and comprehension of the techniques are important to achieve benefits. For this reason, children, patients with developmental and cognitive delays, and some adults may require different approaches. Case reports and case series suggest that the use of a few isolated behavioral techniques may be sufficient

to achieve clinical benefit in some individuals with trichotillomania.

For hair pulling that occurs automatically and with limited awareness, techniques to increase awareness would be expected to assist in breaking the chain of hair-pulling behaviors. Himle et al. (2008) developed an awareness-enhancing monitoring device composed of a watch, bracelet, magnetic necklace, and pager that was hard-wired for remote activation of a vibrating alert by an observer when hair pulling occurred. The authors reported marked reductions in hair pulls, near hair pulls, and touching in the vicinity of hair when the device was used.

Self-monitoring used as a principal intervention has been reported to be an effective treatment in some cases of trichotillomania. For example, Anthony (1978) reported the positive response of a 9-year-old child who was instructed to record both hair pulling and actual hair pulls on a wrist counter. However, other components of treatment were also likely to have played a role, such as behavioral sequence interruption due to the introduction of a barrier (the patient was instructed to wear a cap) and social reinforcement (Rothbaum et al., 1999).

Several authors report successful interventions when the patient is required not only to monitor but also to collect and bring the pulled hairs to the session (Bayer, 1973; Stabler & Warren, 1974; Wulfsohn & Barling, 1978). The latter requirement not only holds the patient accountable, but can also be conceptualized as a mild socially aversive consequence due to the associated embarrassment that patients often report in association with their hair pulling.

Response prevention alone may be useful in some childhood cases. Blum and colleagues, 1993) reported on two cases of children who avoided hair pulling by wearing hand socks, sitting on their hands, or grasping a pencil.

Aversive consequences of hair pulling have also been utilized, such as self-administered rubber band snaps to the wrist (Mastellone, 1974) and rigorous exercise (Stevens, 1984). Therapist-administered aversive consequences have included faradic shock, hand slaps, and aromatic ammonia inhalation (Altman et al., 1978; Gray, 1979; Horne, 1977). For young children who pull out hair while sucking their thumbs, the application of an aversive-tasting substance to the thumb has been reported to reduce both behaviors (Altman et al., 1982; Friman & Hove, 1987; Knell & Moore, 1988). Response cost, the denial of privileges, and removal of a valued possession or experience as a consequence of hair pulling have been also been employed but usually in the context of additional interventions (Cordle & Long, 1980; Epstein & Peterson, 1973).

Overcorrection is a technique in which the patient is taught to substitute appropriate hair-focused behaviors such as combing or brushing for hair pulling. Overcorrection has been reported to be effective in case reports of trichotillomania in developmentally disabled children and adults (Barrett & Shapiro, 1980; Matson et al., 1978)

Facial screening has also been reported to be an effective treatment component when treating trichotillomania in developmentally disabled children (Barmann & Vitali 1982; Gross et al., 1982). This technique involves covering the child's face with a soft cloth as a consequence of hair pulling. It interferes with access to the hair-pulling region, and both interrupts and times out the chain of behavior associated with hair pulling.

In contrast to negative consequences for hair-pulling behavior, other reports document the usefulness of positive reinforcement in the treatment of children. These have included therapist and parental praise, food rewards, and token economies (Altman et al., 1982; Evans, 1976; Gray, 1979; Sanchez, 1979; Sapir, 1971; Wulfsohn & Barling, 1978).

A Flexible Multicomponant Behavioral Approach

Mansueto and coauthors (1999) have proposed a comprehensive model for the behavioral treatment of trichotillomania. The model emphasizes broad flexibility in the selection of available treatment techniques based on an initial and ongoing evaluation of the cognitive, sensory, emotional, behavioral, environmental, consequential, and motivational variables that initiate and support hair pulling as well as the patient's preference for, and acceptance of, treatment interventions. Each individual is expected to have a unique set of variables that characterizes his or her hair pulling, and these variables will differ in the relative degree to which they contribute to hair-pulling initiation and continuation. Potential interventions include nearly all of the behavioral approaches reported in the literature, including those embodied in HRT. Treatment techniques are matched to variables that are particularly important to the specific patient's hair pulling, whether they be cognitive (e.g., thought stopping, cognitive challenging, positive coping statements), affective

(e.g., relaxation and breathing techniques, stress management skill training, imaginal exposure, referral for medication), motoric (e.g., self-monitoring, increased awareness strategies, response prevention, competing response training), sensory (e.g., distraction, substitution, extinction), or environmental (e.g., stimulus control, contingency management, punishment strategies). Each selected technique is practiced and assessed in regard to its acceptability, practicality, and effectiveness. Continuation, modification, addition, and/or substitution of strategies are allowed throughout treatment and obstacles to treatment are assessed, including noncompliance. Although Mansueto et al. provide examples of long-term sustained benefits in two patients treated with their comprehensive approach, it is yet to be supported by any empirical studies. This may, in fact, be a difficult methodological undertaking considering the flexibility, multiple techniques, and individualized treatment plans inherent in this approach.

Medication Treatment of Trichotillomania

Pharmacological treatment of trichotillomania is common but has limited support in the literature. Placebo-controlled studies are limited and have often negated the initial enthusiasm for medications that appeared promising in open-label trials. Currently, the literature supports a role for clomipramine, olanzapine and *N*-acetylcysteine for trichotillomania itself, although other interventions may be reasonable in the context of comorbid conditions that may be contributing to the severity of hair pulling.

Clomipramine

Although a limited number of reports of apparently effective pharmacological treatment of trichotillomania already existed in the literature, it wasn't until 1989 that the first comparison study of medication treatment of trichotillomania was published. Based on observations that trichotillomania had phenomenological similarities to obsessive-compulsive disorder (OCD), Swedo et al. (1989) compared the strongly serotonergic tricyclic antidepressant clomipramine to desipramine, a tricyclic primarily characterized by noradrenergic reuptake inhibition. Clomipramine had been demonstrated to be superior to less serotonergic antidepressants in studies of OCD (Leonard et al., 1989; Volavka et al., 1985). Thirteen subjects with severe hair pulling completed this double-blind, randomized study, which consisted of 5 weeks treatment with either medication prior to immediate crossover to the alternative medication. Clomipramine (mean dose, 181 mg/day) proved statistically superior to desipramine, as measured by two of the three NIMH trichotillomania scales. These positive findings are impressive considering the short duration of treatment, initial medication titrations, and lack of a washout period in the study design.

In a second abstracted but unpublished study (Pigott et al., 1992), 12 subjects with trichotillomania participated in a comparison study with 10 weeks of clomipramine treatment of the same duration with the more selective serotonin reuptake inhibitor fluoxetine in a randomized, double-blind crossover design with a 4-week intervening placebo substitution phase. Both clomipramine and fluoxetine demonstrated statistically significant reductions in trichotillomania, with loss of effectiveness observed between treatment phases.

Ninan et al. (2000) compared 9 weeks of clomipramine (mean dose, 117 mg) treatment to placebo in a randomized parallel design study, which also included a behavioral treatment arm (discussed above). Sixteen subjects completed the study. Clomipramine treatment was associated with greater symptom reduction than placebo, but this result was not statistically significant.

A meta-analysis (Bloch et al., 2007) of the treatment responses of the 24 completers in the abovementioned studies of Swedo et al. (1989) and Ninan et al. (2000) revealed a significant treatment effect of clomipramine compared to the control conditions. This observation remained significant with a subsequent last observation carried forward sensitivity analysis.

As evidenced above, there is good support for the use of clomipramine in the short-term treatment of trichotillomania. However, trichotillomania's characteristic chronic course requires evidence for prolonged, sustained improvements over time. Swedo's group reported continued benefit at 6 months in the subjects in their initial study of clomipramine, as well as when assessed by phone at a mean of 4.3 years later. A 40% reduction in trichotillomania symptoms was retained. However, many subjects had changed or added medications and/or had participated in psychotherapy or behavioral therapy (Swedo et al., 1993). Pollard and colleagues (1991) also reported on the relapse of three of four patients initially treated with clomipramine when followed for 7 to 12 weeks. Taken together, these observations suggest caution in interpreting the long-term

treatment potential of medications for trichotillomania based on short-term treatment studies.

Selective Serotonin Reuptake Inhibitors

Several studies have looked at the effectiveness of selective serotonin reuptake inhibitors (SSRIs) in the treatment of trichtotillomania. Presumably SSRIs would offer treatment advantages similar to those of clomipramine if trichotillomania and OCD were etiologically similar, since SSRIs have been repeatedly demonstrated to have anti–obsessive-compulsive properties (Abramowitz, 1997). Also, the SSRIs offer additional treatment advantages, as they are associated with fewer side effects compared to the undesired anticholinergic, antihistaminergic and alpha-adrenergic effects of a tricyclic such as clomipramine.

In the first placebo-controlled study of an SSRI for trichotillomania, Christenson et al. (1991b) studied 16 hair pullers in a 19-week double-blind, randomized crossover study of fluoxetine titrated up to 80 mg/day (mean dose, 77.5 mg/day sustained over the final 2 weeks). Treatment with either agent was for 6 weeks, with a 5-week washout period between the two study conditions. Outcome measures included subject-rated severity of hair pulling and urges to pull, as well as estimated hair loss and number of hair-pulling episodes per week. Contrary to the suggested benefit of fluoxetine in several early open-label series (Benarroche, 1990; Koran et al., 1992; Stanley et al., 1991; Winchel et al., 1992b), no statistically significant advantage was observed for fluoxetine over placebo on any of the outcome measures. The short duration of the active treatment and the titration schedule were potential shortcomings of this study, although a similar duration of treatment was effective in the above-mentioned clomipramine study of Swedo et al. (1989). Strichenwein and Thomby (1995) addressed these methodological limitations by replicating the Christenson et al. study but extending the treatment phase to 12 weeks for both agents in a study of 16 hair pullers. Despite the extended length of treatment, fluoxetine again proved to be ineffective compared to placebo. Finally, van Minnen et al. (2003) evaluated the responses of 40 completers in a randomized parallel-group trial of fluoxetine compared to HRT and a waiting list control group. Fluoxetine recipients were given a dose titrated up to 60 mg/day over 2 weeks and then sustained at this dose for 12 weeks. Habit reversal treatment was determined to be statistically superior to both fluoxetine and delayed (waiting list) treatment; fluoxetine was deemed ineffective in the short-term treatment of trichotillomania.

The utility of the SSRI sertraline in the treatment of trichotillomania was investigated in a 12- week double-blind, placebo-controlled, randomized study that also assigned additional HRT (described above) administration to nonresponders (Dougherty et al., 2006). Of the 42 subjects initially enrolled in the study, 26 completed the 22-week study, of whom only 2 received placebo and were thus excluded from further statistical analysis. Therefore, the benefit of placebo control was lost. By the end of the study, both the sertraline and the combination treatment groups demonstrated improvement, although the sertraline group was limited to only four subjects. The combined treatment group demonstrated significantly better improvement compared to the sertraline group, although the design of the study and the limited number of subjects made the individual contributions to the treatment response in the combined treatment difficult to ascertain.

Two open studies have investigated the potential efficacy of the SSRI fluvoxamine for the treatment of trichotillomania. In the first, Stanley and colleagues (1997) administered fluvoxamine to 21 subjects in a 12-week open trial. Thirteen subjects with trichotillomania completed the trial. Significant improvement in hair pulling was noted on only a few measures, and the authors concluded that fluvoxamine was not effective for trichotillomania. However, further analysis, including the additional eight subjects who had initiated but not completed treatment with fluvoxamine, demonstrated improvement on measures of associated hair-pulling distress, episode duration, sense of control, and ability to resist hair pulling, as well as degree of coexisting anxiety and depression. The high dropout rate was primarily attributed to medication side effects, which may have been related to a forced dose titration from 50 mg to 300 mg over the first 3 weeks of treatment. In the second study, Christenson and colleagues (1998) conducted an 8-week study of fluvoxamine in 19 patients with trichotillomania. Statistically significant improvements were noted on four of five outcome measures. However, only four subjects were deemed responders based on criteria of greater than 50% improvement in two of the three primary outcome measures. These responders chose to continue on medication; all lost the majority of their benefits at 4-month follow-up.

Citalopram (mean dose, 36 mg/day) was administered to 14 patients (13 completers) in a 12-week naturalistic study of hair pullers (Stein et al., 1997).

Of the completers, 39% were deemed responders at 12 weeks. Escitalopram, a racemic isomer of citalopram, was investigated in a 12-week open-label study involving 20 women with trichotillomania (Gadde et al., 2007). Of the 16 subjects who had at least one postbaseline assessment, 8 experienced significant reduction in hair pulling on doses of escitalopram ranging from 10 to 30 mg/day. However, these results must be considered cautiously in view of the tendency for initially promising results associated with open-label treatment of trichollomania to be negated later on by studies incorporating randomized, double-blind comparison methodology.

The previously mentioned meta-analysis of treatment studies by Block et al reviewed all published controlled studies for SSRIs. Four of the studies described above were identified with acceptable methodology, representing a total of 72 completers who had received either fluoxetine of sertraline. No difference was noted between control conditions and treatment with SSRIs. The overall estimated effect size was negligible and favored placebo. A last observation carried forward sensitivity analysis also revealed a lack of statistically significant difference in SSRI treatment compared to control conditions.

Taken together, there is little evidence to support the use of SSRIs for trichotillomania alone. However, SSRIs may still play a role in treating comorbid conditions such as depression, generalized anxiety, and OCD, all of which could play contributing roles in the severity of trichotillomania. Additionally, it has yet to be determined whether hair pulling that is more focused and compulsive in nature might represent a subset of trichotillomania with a greater likelihood of responding to this pharmacological class.

Selective Serotonin and Norepinephrine Reuptake Inhibitors

Serotonin and norepinephrine reuptake inhibitors (SNRIs) retain the strong serotonergic properties initially suspected to be of benefit for the treatment of trichotillomania while offering additional noradrenergic activity similar to, albeit stronger than, that of clomipramine. Considering the initial expectation that serotonergic agents would be of benefit for the treatment of trichotillomania and the data that continue to lend stronger support for the efficacy of clomipramine, it is not surprising that the SNRI venlafaxine would have been investigated as a possible treatment for trichotillomania. O'Sullivan et al (1998) reported on 10 patients who had been treated with venlafaxine (mean dose = 274 mg/day) for 8-28 weeks. Significant improvement was observed in an intent-to-treat analysis as measured by the MGH-HPS and the PITS. Ninan et al. (1998) treated 20 patients with venlafaxine (mean dose, 322.5 mg/ day) for up to 12 weeks. Responders were defined as demonstrating a minimal 50% reduction in hair-pulling symptoms as well as being clinician rated as "much" to "very much" improved. Based on these criteria, 55% of subjects were deemed treatment responders. Eight responders were then entered into a double-blind discontinuation trial in which half were randomly assigned to continue on medication and half were switched to placebo. With the exception of one patient who received placebo, all subjects relapsed within the next 24 weeks.

Opiate Antagonists

Naltrexone has been hypothesized to be a potential treatment for trichotillomania due to its ability to blunt endogenous opioid activity that might be rewarding hair-pulling behavior. Additionally, naltrexone might increase pain perception during hair pulls, with resultant increased awareness of and mild aversive consequence for the behavior (Christenson et al., 1994a). The benefits of naltrexone in other repetitive, impulsive, or self-abusive behaviors have been documented in both humans and animals (Dodman et al., 1998; Sonne et al., 1996; Taylor et al.,1991; White, 1990). Christenson et al. (1994a) studied the opiate antagonist naltrexone (50 mg/ day) for the treatment of trichotillomania in a randomized, placebo-controlled, double-blinded study. Seventeen subjects completed the study, but only seven completers received the active agent. Three of the seven demonstrated more than a 50% reduction in hair-pulling symptoms compared to none in the placebo group. The active-treatment group demonstrated a statistically significant improvement in symptoms, as measured by the NIMH trichotillomania severity scale, but not on two other measures. There was no statistically significant difference in the reported number of hair-pulling episodes, although improvement was in the expected direction with naltrexone.

An open-label exploration of the treatment of trichotillmania with naltrexone (De Souza, 2008) in 14 children (mean age, 9 years) resulted in a positive outcome for 11 subjects. Treatment doses ranged from 25 to 100 mg/day, with a mean dose of 66 mg/day.

Dopamine Antagonists

Olanzapine is an atypical neuroleptic with potent selective serotonergic (5-HT2) and dopaminergic

(D2) receptor antagonism. Several case reports and series have suggested the utility of both traditional (pimozide and haloperidol) and atypical (respiridone, olanzapine, quitiapine) neuroleptics when added to serotonin reuptake inhibitors in treatment-resistant trichotillomania (Crescente et al., 2003; Epperson et al., 1999; Gabriel, 2001; Pathak et al., 2004; Potenza et al., 1998; Stien & Hollander, 1992; van Ameringen et al., 1999) or when used as monotherapy (Khouzam et al., 2002; Ravindran et al., 2004; Şentürk & Tanrıverdi, 2002). The dopamine modulator, aripiprazole, has also been reported to have produced prolonged resolution of trichotillomania when used alone in a treatment-resistant case (Jeffreys & Burrows, 2008). Stewart and Nejtek (2003) reported the results of an open-label, flexible dose trial of olanzapine in 17 patients with trichotillomania who had completed at least 1 week of treatment. Doses were titrated up to a maximum of 10 mg/day by 3 weeks (final dose, 7.5-10 mg/day), and the total duration of treatment was 3 months. Hair-pulling symptoms decreased by 66%. Four patients experienced complete resolution of symptoms by the end of the study period. Of 12 patients evaluated 1 month after olanzapine discontinuation, 8 had maintained their improvement while 4 were noted to have relapsed. Only one was without any urges to pull hair.

Van Ameringen and colleagues (2010) randomly assigned 25 patients with trichotillomania to olanzapine or placebo in a 12-week study. Olanzapine was administered in a flexible manner, ranging from an initial dose of 2.5 mg/day to a maximum dose of 20 mg/day during the eighth and twelfth week. The average olanzapine dose at endpoint was 11 mg/day. Eleven of 13 (85%) subjects assigned to olanzapine were considered responders according to the CGI-Impovement subscale compared to only 2 of 12 (17%) in the placebo group. Significant improvement in hair-pulling symptoms was noted, as measured by the TTM-YBOCS and the CGI- Severity of Illness subscale.

In a retrospective study of nine children and adolescents (age range, 9–15 years) treated with atypical antipsychotics (resperidone, quetiapine; Mancini et al., 2009), either alone or as adjuncts to SSRIs, eight were considered to be responders. However, only two patients achieved remission.

Although there is evidence supporting the effectiveness of olanzapine and other atypical drugs for the treatment of trichotillomania, the known potential side effects of these agents, including weight gain, hyperglycemia, hyperlipidemia, cardiac arrhythmia, and tardive dyskinesia (Üçok & Gaebel, 2008), suggest judicial consideration of the risks and benefits, as well as consideration of whether the severity of trichotillomania in individual cases justifies treatment with this pharmacological class.

Mood Stabilizers

Single case reports and case series have suggested that mood stabilizers such as lithium (Christenson et al., 1991c), valproate (Adewuya et al., 2008), and lamotragine (Moretti, 2008) may benefit some patients with trichotillomania. The utility of the antiepileptic and proposed mood stabilizer topiramate for the treatment of trichotillomania was investigated in an open-label study of 14 patients with this disorder (Lochner et al., 2006). Nine subjects completed 16 weeks of treatment with topirimate administered in a dose ranging from 50 to 250 mg/day. The severity of hair pulling decreased significantly by the end of the study. Six of nine completers were classified as responders. However, topiramate was associated with a high dropout rate due to its side effects.

Topical Agents

Since trichotillomania may exist with reduced awareness of the pain expected with hair pulling (Christenson et al., 1994b) or in response to physical sensations such as pruritis (Christenson et al., 1991a), one avenue for treatment that has been only minimally explored is the use of topical agents to either restore the expected sensory consequences of hair pulling or to reduce sensory cues that prompt the pulling. For example, the antipruritic and analgesic topical agent benzocaine cream was reported to decrease hair pulling when combined with behavioral therapy in an adolescent with a strong pruritic sensory component cueing her hair pulling (Dia, 2008). Topical fluocinolone has been used as a beneficial adjunct to clomipramine treatment (Black & Blum, 1992, Gupta & Freimer, 1993). Additionally, the topical antibiotic tobramycin has been reported to be effective in a case of lash and brow pulling in which blepharitis appeared to play a role in cueing the hair pulling (O'Sullivan et al., 1999).

In contrast to its usually more frequent dosing schedule, low-dose topical capsaicin, when applied once daily, increases pain sensitivity rather than producing analgesia via continuous localized endogenous neuropeptide release. Low-dose topical capsaicin was noted to reduce hair pulling in a single case of trichotillomania when used adjunctively with behavioral therapy (Ristved & Christenson, 1996).

Glutamate Modulators

Glutamate is concentrated in the extracellular regions of the nucleus accumbens, and glutamatergic dysfunction in this region has been proposed to be associated with repetitive behaviors including those in OCD (Chakrabarty et al., 2005). In a follow-up to some initially promising responses of trichotillomania to the glutamate modulator *N*-acetylcysteine (Odlaug & Grant, 2007), Grant et al. (2009) conducted a 12-week randomized, double-blind, placebo-controlled trial of *N*-acetylcysteine in 50 subjects with trichotillomania. *N*-acetylcysteine was given in a dose of 1200 mg/day for the first 6 weeks and increased to 2400 mg/day if subjects had not experienced complete cessation of hair pulling during the preceding 3 weeks. The results favored *N*-acetylcysteine over placebo. Fifty-six percent of the active treatment group were rated as "much" or very "much improved" compared to only 16% of those on placebo. The *N*-acetylcysteine group also demonstrated a statistically significant 41% symptom reduction on the primary symptom outcome measure, the MGH-HPS. Significant improvement was noted after 9 weeks of treatment, which could reflect either a delayed response or the dose escalation in the majority of subjects (18 subjects randomized to the active agent required dose titration). Subjects in this study were allowed to continue the treatments present at study entrance. Eight percent of subjects participated in ongoing psychotherapy and 56% took psychotropic medications including SSRIs, SNRIs, and stimulants. Rates of psychotropic medication use did not differ between the two treatment groups, nor was any particular medication associated with a treatment response, suggesting that the observed benefits did not reflect a primary augmentation role for *N*-acetylcysteine. The availability of *N*-acetylcysteine in health food stores without a prescription, and its relatively low cost, are advantages for this treatment approach. However, it should also be recognized that 44% of subjects receiving *N*-acelylcysteine failed to respond.

A case report of the response of trichotillomania to the antiglutamatergic agent riluzole (Coric et al., 2007) supports the need for additional exploration of treatment approaches targeting glutamate regulation in trichotillomania.

Anxiolytics

Since SSRIs and SNRIs have anxiolytic properties and several have specific indications for generalized anxiety, traditional anxiolytics have also been suggested as potentially effective interventions for hair pulling. Özcan (2003) treated 12 adolescents with trichotillomania with the anxiolytic/antihistamine hydroxyzine in an open-label design. All subjects received 30 mg/day for 4 weeks. Reduction of hair pulling was determined by pre- and posttreatment examination by a dermatologist who assessed for areas of alopecia, "remnants of hair particles," and new hair growth. No information on how these observations were quantified was provided. Following treatment, the subjects were noted to have decreased alopecia, decreased hair remnants, and increased hair growth. Scores on a standardized scale for the measurement of depression in children and adolescents were significantly reduced as well. The authors hypothesized that the anxiolytic effects of hydroxyzine may have had a secondary benefit for hair pulling. A case of hair pulling responding to the anxiolytic buspirone has been reported (Reid, 1992), and the benzodiazepine clonazepam has been noted to be of benefit for some patients with trichotillomania (Christenson & Crow, 1996).

Other Agents

The above review documents the exploration of multiple pharmaceutical classes in the treatment of trichotillomania. Nearly every psychotropic class has been reported to have been of benefit in single case reports or small series of cases. These cases are often characterized by additional treatment approaches as well as comorbid illness. In addition to those already mentioned, case reports and series include positive responses to the monoamine oxidase inhibitor isocarboxazid (Krishnan et al., 1984), the tetracyclic antidepressant mianserin (Hussain, 1992), the serotonin antagonist and reuptake inhibitors trazodone (Sunkureddi, & Markovitz, 1993) and nefazodone (O'Sullivan et al., 1999), bupropion (Bhanji & Margolese, 2004), the stimulant methyphenidate (O'Sullivan et al., 1999), the progestin levonorgestrel (Perciaccante & Perciaccante, 1993), the mixed indirect/direct agonist fenfluramine (Mahr, 1993), and the glucose isomer inositol (Seedat et al., 2001).

Combined Behavioral and Medication Treatment

In the only controlled study to explore combined HRT and medication treatment, Dougherty et al. (2006) compared the cognitive behavioral therapy combined with the SSRI sertraline to treatment with either agent alone. Forty-two subjects were initially randomized to treatment with sertraline (maximum dose, 200 mg/day) or placebo in the

initial 12 weeks of this double-blind study. For those subjects who did not respond significantly to sertraline, HRT was added for an additional 10 weeks. Thirteen subjects who received only one of the active treatment modalities (4 on medication and 9 on HRT) completed the 22 weeks of the study, and 11 received both treatments. An additional two completers were revealed to have been on placebo and were excluded from further analysis. Compared to the single-modality group, the dual-modality group showed significantly greater improvement on most within-group change scores. Between-group change scores supported dual-modality treatment as superior to single-modality treatment on both primary outcome measures. Six (54.5%) subjects in the dual-treatment groups were considered responders compared to two (15.4%) in the single-treatment group.

Hypnosis

Hypnosis has been reported to be of benefit for the treatment of trichotillomania in case reports and series of both adults and children; however, no controlled studies of hypnosis have been published. In a review of the literature, Robiner et al. (1999). identified 15 reports that accounted for a total of 32 patients with trichotillomania treated with hypnosis. Their ages ranged from 14 to 35, and the duration of hair pulling ranged from 2 to 28 years. Techniques varied greatly and were frequently employed in the context of additional treatment interventions identified as individual psychotherapies (Adlerian, psychoanalytic, and others), group therapy, family therapy, cognitive behavioral therapy, relaxation and breathing training, and behavioral techniques such as self-monitoring, thought stopping, and aversive techniques. The reported number of hypnosis sessions, when noted, varied from 2 to 10. Depending on the case, the aim of the hypnosis was to increase awareness and control of hair pulling, decrease anxiety and substitute other behaviors, increase pain sensitivity, and/or emphasize respect for one's body. Hypnosis was used in one patient for age regression to identify events and conditions that may have contributed to the onset of hair pulling. Twenty-two (69%) patients were reported to have decreased their hair pulling from 90% to 100%. However, the reviewers cautioned against making generalizations from these data, as ineffective treatments are unlikely to be published as case reports.

Robiner et al. (1999) also reported on an additional 10 patients with trichotillomania who participated in a retrospective survey 2 to 28 weeks after treatment completion. Treatment with hypnosis employed multiple posthypnotic suggestions directed at increasing motivation, enhancing control, decreasing hair-pulling urges, substituting relaxation for urges, increasing awareness, and distancing the hands from the hair-pulling site. Specific posthypnotic suggestions were tailored to address individual patients' high-risk situations for hair pulling. The authors reported that 40% of patients reported complete cessation of hair pulling. Thirty percent reported moderate benefit, and 20% reported mild benefit. Only one reported no benefit at all. The mean overall improvement estimated for the participants was 57%. Ninety percent of patients reported less intense urges to pull out hair, and 70% reported greater control over their behavior. Although the duration of response varied, 80% of patients reported benefits lasting for at least 3 months.

As noted in the review by Robiner et al. (1999), hypnosis appears to be useful for children as well as adults, thus providing an approach for younger hair pullers who may not be able to participate in more complex behavioral treatments or for whom there may be complex family dynamics that foster the continuation of hair pulling. Iglesias (2003) reported on three children with trichotillomania whose behavior was hypothesized to generate secondary gains via opposition to their overbearing and overinvolved parents. Hypnosis centered on suggestions that alerted the children to an impending hair pull but emphasized that they had sole authority over their hair and could choose to pull or resist the behavior. Their parents were instructed to relinquish their authority over their child's hair. All three children were free of hair pulling after seven or fewer sessions, and all were in remission at the 6-month follow-up. Zalsman and colleagues (2001) took a somewhat different approach in emphasizing the patient's authority over his or her hair. They used hypnotic suggestions to portray hair as weak and in need of the patient's protection while treating three adolescents with trichotillomania. Hair pulling was reduced significantly in all three patients, with improvement maintained at the 6-month follow-up.

Naturalistic Treatment Studies

As illustrated above, initial optimistic reports of the efficacy of a specific treatment approach in trichotillomania in case reports and open-label studies are often followed by disappointing outcomes in controlled conditions, and many treatments have yet to be studied with this methodology. Studies have also

tended to focus on a "one treatment fits all" approach despite the heterogeneity of hair-pulling behaviors and comorbidity. In addition, the potential cumulative benefits of additional treatment approaches over time, and the ability to alter strategies in response to differing emotional, environmental, and behavioral circumstances characteristic of clinical practice, are rarely studied due to the complex methodological and design issues involved. Keuthen et al. (1998b) addressed this issue to some degree in a retrospective review of 63 patients with trichotillomania seen at a clinic specializing in this disorder. The use of a standard assessment of hair-pulling severity, depression, and anxiety provided the framework to assess change over time via a survey completed by 67 of 80 patients who had been treated over a period of more than 6 years. Sixty-five percent of those surveyed had received a combination of behavioral therapy and medication. Ninety percent had been treated with behavioral therapy, either alone or in combination with medication. Seventy-three percent had been treated with medication. Medications included clomipramine, four different SSRIs, venlafaxine, and lithium. Additional treatments included hypnosis (37%), psychotherapy (50%), and participation in a support group (33%). Just over half of the patients were still in treatment at the time they were surveyed. Sixty-two percent of these patients were being treated with medication, and 62% were being treated with behavioral therapy. One-quarter of respondents were being treated with both. Compared to the initial assessment, there was significant improvement in hair-pulling symptoms. Fifty-two percent of the patients rated themselves as treatment responders.

Boughn and Holdom (2002) recorded semi-structured phone interviews of 44 self-identified hair pullers to assess their perceptions of their response to treatment. Treatment approaches were divided into five broad categories: medications, behavior modification, individual therapy, group therapy, and alternative therapy. Seventy-seven percent of subjects had received medication for trichotillomania or coexisting disorders such as anxiety and/or depression. Medications included four different SSRIs, clomipramine, venlafaxine, and other antidepressants, as well as lithium, olanzapine, buspirone, and several benzodiazepines. Only two subjects (6%) reported long-term efficacy with medication. Both subjects had four ineffective pharmacological interventions before they responded to either St. John's wort or a combination of olanzapine and paroxetine. Seventy-three percent of subjects had been treated with behavioral modification; 56% found this intervention to be effective. However, participants noted that maintaining the acquired techniques was challenging. Individual psychotherapy was reported to be utilized by 71% of subjects but was less effective than behavioral modification; only 32% of subjects treated with individual psychotherapy responded to this intervention. Group therapy was utilized by 68% of subjects. Five of eight (63%) women treated with group therapy designed to treat trichotillomania found it to be beneficial; in contrast, none of seven women treated with group therapy that did not specifically target hair pulling found this approach to be helpful. The authors used the term *nontraditional group therapy* to describe two other types of group involvement. One of them referred to Internet-based trichotillomania support groups. The other referred to involvement with various aspects of the Trichotillomania Learning Center (TLC), a national organization that provides support, information, referrals, and resources for individuals with trichotillomania. Eighteen (55%) subjects interacted with at least one of these two resources, and seventeen (94%) found them to be effective. Web-based support groups were noted to have the advantage of anonymity, and appeared to be less threatening than face-to-face participation. The authors noted that the existence of both the Web-based support groups and the TLC resources helped to emphasize that subjects were not alone, provided support from others dealing with trichotillomania, and provided resources that otherwise may not have been available in the subject's community.

The Trichotillomania Impact Project (Woods et al., 2006a) employed an Internet-based survey to further clarify phenomenological properties, functional impairment, and treatment response in a large (n = 1697) cohort of subjects who pulled out their hair. Forty-two percent of respondents reported having been treated with medications including SSRIs and SNRIs, strong and weakly serotonergic tricylics, serotonergic antagonist/reuptake inhibitors, traditional and atypical neuroleptics, benzodiazepine and nonbenzodiazepine anxiolytics, mood stabilizers, naltrexone, and clonidine. Thirty-one percent of subjects had been treated with behavioral approaches including self-monitoring, relaxation training, stress management training, HRT, stimulus control techniques, and relapse prevention training. Thirteen percent had participated in a support group and 12% had received hypnosis. When asked about their treatment response, 15% rated their condition as "much improved" to "very much improved."

Twenty-one percent reported minimal improvement, and 38% reported that their hair pulling was unchanged. Sixteen percent reported that their hair pulling had worsened with treatment. There was no attempt to determine whether any treatment type appeared to be superior to the others. This Internet-based survey had several significant methodological limitations, including subjective reports of response and lack of information regarding treatment duration, dosing, and treatment compliance.

Similar to the above study, Franklin et al. (2008) reported the results of the Trichotillomania Impact Survey for Children, an Internet-based survey completed by 135 children and adolescents ages 10 to 17 who pulled out their hair. The survey gathered information from the afflicted youth as well as the impressions of a parent. According to parental reports, 65% of children had received some form of treatment. Treatment included pharmacotherapy (49%), behavior therapy (45%), general psychotherapy (20%), support group (12%), and hypnosis (10%). Forty-seven percent of responders had been treated with pharmacotherapy as well as behavioral therapy, but the survey did not indicate whether treatment was sequential or simultaneously applied. Medication trials ran the gamut of treatment classes, including the serotonin antagonist and reuptake inhibitor trazodone, multiple serotonin reuptake inhibitors, the SNRI venlafaxine, the tricylic antidepressant clomipramine, multiple atypical antipsychotics, benzodiazepine and nonbenzodiazepine anxiolytics, and the opiate antagonist naltrexone, as well as lithium and clonidine. Behavioral treatment included HRT, relaxation training, and stress management training. Eighty-one parents rated their child's response to treatment. Nineteen percent reported that their child's hair pulling was "much" or "very much improved." Twenty-two percent rated hair pulling as minimally improved; 48% reported hair pulling as unchanged, and 11% said that their child's hair pulling was "minimally" improved to "very much worse" following treatment.

Considered together, the above naturalistic studies raise concerns about the long-term prognosis for trichotillomania even when treated with multiple strategies over time. The most encouraging study, that of Keuthen et al. (1988), noted that 52% of subjects were treatment responders, but these were subjects who had all been cared for in a clinic highly specialized in the treatment of trichotillomania, and subjects still required multiple interventions with varying treatment modalities. The other studies, which could be argued to better reflect treatment as administered in the community, suggest that despite multiple treatment strategies, perhaps only 15%–20% of cases of trichotillomania will achieve acceptable sustained improvement.

Conclusion

The treatment of trichotillomania is based on a thorough assessment of all aspects of hair-pulling behavior including hair-pulling sites, hair-pulling behavioral sequences, cues that initiate hair pulling, associated emotions, high-risk situations, awareness and cognitive style, and comorbid conditions. Assessment of improvement can be done via hair-pulling logs, collection of hair, or visual observation of change, or by employing one of several assessment instruments that have been specifically developed to assess change in hair-pulling urges, behaviors, and thoughts. Of the available instruments, the MGH-HPS has become the most commonly employed due to its ease of use and established psychometric properties.

Multiple treatment interventions have been investigated as potentially useful for trichotillomania. These have included medications, behavioral therapy, and hypnosis. However, controlled studies have narrowed the apparently effective treatments to a limited number of approaches. Habit reversal therapy, including variations and enhancements of this behavioral technique, appears be the most promising intervention. Unfortunately, few therapists are aware of, or trained in, this approach. This deficiency is easily remedied, as several behavioral treatment guides based on HRT have been published for therapists and patients (Franklin & Tolin, 2007; Hoogduin et al., 2004; Keuthen et al., 2001; Rothbaum & Ninan, 1999; Stanley & Mouton, 1996).

Medications, particularly those with strongly serotonergic properties, initially appeared to be good treatment candidates for trichotillomania based on phenomenological similarities of the disorder to OCD as well as early open-label treatment successes. However, more methodologically sound investigations of treatment with serotonergic agents have proved unsupportive; clomipramine appears to be a possible exception. These observations challenge the validity of the initial conceptualization of trichotillomania as a compulsive variant. Exploration of other novel pharmacological agents that have demonstrated initial promise in controlled studies, such as *N*-acetylcysteine and olanzapine, is therefore warranted.

Hypnosis continues to be a reasonable treatment intervention based on the current literature support

for this approach. However, the state of research on hypnosis treatment for trichotillomania is in many ways comparable to that of the SSRIs previously and is characterized by reports of positive treatment responses lacking controlled conditions.

Future Directions

Future research on the assessment and treatment of trichotillomania must build upon the clinical and academic foundation reviewed in this chapter while maintaining a broad perspective on potential treatment approaches in recognition of the variable phenomenology of this disorder. Future research should:

1. Clarify the heterogeneity of hair pulling across individuals and further explore whether specific variables are predictive of treatment success.
2. Investigate whether specific neurotransmitter systems other than the serotonergic system may play a role in the development and maintenance of hair-pulling behavior and whether pharmacological manipulation of these systems is effective.
3. Explore the utility of flexible multimodal approaches such as that advocated by Mansueto et al. (1999) by employing long-term prospective naturalistic study designs.
4. Research the effects of treatment with hypnosis in controlled conditions, with further evaluation of the individual variables predictive of treatment response as well as the most effective hypnotic suggestions.
5. Delineate whether combined pharmacological, behavioral, and/or hypnosis treatment is superior to a single-treatment modality once optimal approaches in each of these categories are more firmly established.

Trichotillomania continues to be a challenging disorder to treat. However, a good understanding of the intricacies of the disorder, combined with a broad perspective on potential treatment approaches, will aid the clinician in selecting an intervention most likely to benefit any individual patient.

References

Abramowitz, J. S. (1997). Effectiveness of psychological and pharmacological treatments for obsessive-compulsive disorder: A quantitative review. *Journal of Consulting and Clinical Psychology*, *65*(1), 44–52.

Adewuya, E. C., Zinser, W., & Thomas, C. (2008). Trichotillomania: A case of response to valproic acid. *Journal of Child and Adolescent Psychopharmacology*, *18*(5), 533–536.

Altman, K, Grahs, C., & Friman, P. (1982). Treatment of unobserved trichotillomania by attention-reflection and punishment of an apparent covariant. *Journal of Behavior Therapy and Experimental Psychiatry*, *13*(4), 337–340.

Altman, K., Haavik, S., & Cook, J.W. (1978). Punishment of self-injurious behavior in natural settings using contingent aromatic ammonia. *Behaviour Research and Therapy*, *16*(2), 85–96.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision). Washington, DC: Author.

Anthony, W. Z. (1978). Brief intervention in a case of childhood trichotillomania by self-monitoring. *Journal of Behavior Therapy and Experimental Psychiatry*, *9*(2), 173–178.

Azrin, N. H., & Nunn, R. G. (1973). Habit reversal: A method of eliminating nervous habits and tics. *Behaviour Research and Therapy*, *11*(4), 619–628.

Azrin, N. H., Nunn, R. G., & Frantz, S. E. (1980). Treatment of hair pulling (trichotillomania): A comparative study of habit reversal and negative practice training. *Journal of Behavior Therapy and Experimental Psychiatry*, *11*(1), 13–20.

Barmann, B. C., & Vitali, D. L. (1982). Facial screening to eliminate trichotillomania in developmentally disabled persons. *Behavior Therapy*, *13*(5), 735–742.

Barrett, R. P., & Shapiro, E. S. (1980). Treatment of stereotyped hair-pulling with overcorrection: A case study with long term follow-up. *Journal of Behavior Therapy and Experimental Psychiatry*, *11*(4), 317–320.

Bayer, C. A. (1973). Self-monitoring and mild aversion treatment of trichotillomania. *Journal of Behavior Therapy and Experimental Psychiatry*, *3*, 139–141.

Begotka, A. M., Woods, D. W., & Wetterneck, C. T. (2004). The relationship between experiential avoidance and the severity of trichotillomania in a nonreferred sample. *Journal of Behavior Therapy and Experimental Psychiatry*, *35*(1), 17–24.

Benarroche, C. L. (1990). Trichotillomania symptoms and fluoxetine response. In *New research program and abstracts of the 143rd annual meeting of the American Psychiatric Association*, Philadelphia, NR327.

Bernard, M. E., Kratochwill, T. R., & Keefauver, L. W. (1983). The effects of rational-emotive therapy and self-instructional training on chronic hair pulling. *Cognitive Therapy and Research, 7*(3), 273–280.

Bhanji, N. H., & Margolese, H. C. (2004). Alternative pharmacotherapy for trichotillomania: A report of successful bupropion use. *Journal of Clinical Psychiatry*, *65*(9), 1283.

Black, D. W., & Blum, N. (1992). Trichotillomania treated with clomipramine and a topical steroid. *American Journal of Psychiatry*, *149*(6), 842–843.

Bloch, M. H., Landeros-Weisenberger, A., Dombrowski, P., Kelmendi, B., Wegner, R., Nudel, J., et al. (2007). Systematic review: Pharmacological and behavioral treatment for trichotillomania. *Biological Psychiatry*, *62*(8), 839–846.

Blum, N. J., Barone, V. J., & Friman, P. C. (1993). A simplified behavioral treatment for trichotillomania: Report of two cases. *Pediatrics*, *91*(5), 993–995.

Bornstein, P. H., & Rychtarik, R. G. (1978). Multi-component behavioral treatment of trichotillomania: A case study. *Behaviour Research and Therapy*, *16*(3), 217–220.

Boughn, S., & Holdom, J. A. J. (2002). Trichotillomania: Women's reports of treatment efficacy. *Research in Nursing and Health*, *25*(2), 135–144.

Chakrabarty, K., Bhattacharyya, S., Christopher, R., & Khanna, S. (2005). Glutamatergic dysfunction in OCD. *Neuropsychopharmacology*, *30*(9), 1735–1740.

Christenson, G. A., & Crow, S. J. (1996). The characterization and treatment of trichotillomania. *Journal of Clinical Psychiatry*, *57*(8, Suppl), 42–49.

Christenson, G. A., Crow, S. J., & MacKenzie, T. B. (1994a). A placebo controlled double blind study of naltrexone for trichotillomania. In *New research program and abstracts of the 150th annual meeting of the American Psychiatric Association*. Philadelphia, NR597.

Christenson, G. A., Crow, S. J., Mitchell, J. E. Mackenzie, T. B., Crosby, R. D., & Falls, J. (1998). Fluvoxamine in the treatment of trichotillomania: An eight-week, open-label study. *CNS Spectrums, 3*(10), 64–71.

Christenson, G. A., Mackenzie, T. B., & Mitchell, J. E. (1991a). Characteristics of 60 adult hair pullers. *American Journal of Psychiatry*, *148*(3), 365–370.

Christenson, G. A., Mackenzie, T. B., Mitchell, J. E., & Callies. A. (1991b). A placebo-controlled double-blind crossover study of fluoxetine in trichotillomania. *American Journal of Psychiatry, 148*(11), 1566–1571.

Christenson, G. A., Popkin, M. K., Mackenzie, T. B., & Realmuto, G.M. (1991c). Lithium treatment of chronic hair pulling. *Journal of Clinical Psychiatry*, *52*(3), 116–120.

Christenson, G. A., Pyle, R. L., & Mitchell, J. E. (1991d). Estimated lifetime prevalence of trichotillomania in college students. *Journal of Clinical Psychiatry*, *52*(10), 415–417.

Christenson, G. A., Raymond, N. C., Faris, P. L., McAllister, R. D., Crow, S. J., Howard, L. A., et al. (1994b). Pain thresholds are not elevated in trichotillomania. *Biological Psychiatry*, *36*(5), 347–349.

Christenson, G. A., Risvedt, S. L., & MacKenzie, T. B. (1995). Identification of trichotillomania cue profiles. *Behaviour Research and Therapy*, *31*(3), 315–320.

Cordle, C. J., & Long, C. C. (1980). The use of operant self-control procedures in the treatment of compulsive hair-pulling. *Journal of Behavior Therapy and Experimental Psychiatry*, *11*(2), 127–130.

Coric, V., Kelmendi, B., Pittenger, C., Wasylink, S., Bloch, M. H., & Green, J. (2007). Beneficial effects of the anti-glutamatergic agent riluzole in a patient diagnosed with trichotillomania. *Journal of Clinical Psychiatry*, *68*(1), 170–171.

Crescente, J. A. B, Guzman, C. A., & Tavares, H. (2003). Quetiapine for the treatment of trichotillomania. *Revista Brasileira de Psiquiatria*, *30*(4), 402.

De Souza, A. (2008). An open-label pilot study of naltrexone in childhood-onset trichotillomania. *Journal of Child and Adolescent Psychopharmacology*, *18*(1), 30–33.

Dia, D. A. (2008). "I can't stop pulling my hair!" Using numbing cream as an adjunct treatment for trichotillomania. *Health and Social Work*, *33*(2), 155–158.

Diefenbach, G. J., Tolin, D. F., Crocetto, J., Maltby, N., & Hannan, S. (2005). Assessment of trichotillomania: A psychometric evaluation of hair-pulling scales. *Journal Psychopathology and Behavioral Assessment*, *27*(3), 169–178.

Diefenbach, G. J., Tolin, D. F., Hannan, S., Maltby, N., & Crocetto, J. (2006). Group treatment for trichotillomania: Behavior therapy versus supportive therapy. *Behavior Therapy*, *37*(4), 353–363.

Dodman, N. H., Shuster, L.,White, S., Court, M. H., Parke, D., & Dixon, R. (1988). Use of narcotic antagonists to modify stereotypic self-licking, self-chewing, and scratching behavior in dogs. *Journal of the American Veterinary Medical Association*, *193*(7), 815–819.

Dougherty, D. D., Loh, R., Jenike, M. A., & Keuthen, N. J. (2006). Single modality versus dual modality treatment for trichotillomania: Sertraline, behavioral therapy, or both. *Journal of Clinical Psychiatry*, *67*(7), 1086–1092.

Du Toit, P. L., van Kradenburg, J., Niehaus, D. J. H., & Stein, D. J. (2001). Characteristics and phenomenology of hair-pulling: An exploration of subtypes. *Comprehensive Psychiatry*, *42*(3), 247–256.

Epperson, C. N., Fasula, D., Wasylink, S., Price, L. H., & McDougle, C. J. (1999). Risperidone addition in serotonin reuptake inhibitor–resistant trichotillomania: Three cases. *Journal of Child and Adolescent Psychopharmacology*, *9*(1), 43–49.

Epstein, L. H., & Peterson, G. L. (1973). The control of undesired behavior by self-imposed contingencies. *Behavior Therapy*, *4*(1), 91–95.

Evans, B. (1976). A case of trichotillomania in a child treated in a home token program. *Journal of Behavior Therapy and Experimental Psychiatry*, *7*(2), 197–198.

Flessner, C. A., Busch, A. M., Heideman, P. W., & Woods, D. W. (2008a). Acceptance-Enhanced Behavior Therapy (AEBT) for trichotillomania and skin picking: Exploring the effects of component sequencing. *Behavior Modification*, *32*(5), 579–594.

Flessner, C. A., Conelea, C. A., Woods, D. W., Franklin, M. E., Keuthen, N. J., & Cashin, S. E. (2008b). Styles of pulling in trichotillomania: Exploring differences in symptom severity, phenomenology, and functional impact. *Behaviour Research and Therapy*, *46*(3), 345–357.

Flessner, C. A., Woods, D. W., Franklin, M. E., Keuthen, N. J., & Piacentini, J. (2009a). Cross-sectional study of women with trichotillomania: A preliminary examination of pulling styles, severity, phenomenology, and functional impact. *Child Psychiatry and Human Development*, *40*(1), 153–167.

Flessner, C. A., Woods, D. W., Franklin, M. E., Keuthen, N. J., & Piacentini, J. (2009b). The Milwaukee Inventory of Styles of Trichotillomania-Child Version (MIST-C). Initial development and psychometric styles. *Behavior Modification*, *31*(6), 896–918.

Franklin, M. E., Flessner, M. A., Woods, D. W., Keuthen, N. J., Piacentini, J. C., Moore, P., et al. (2008). The Child and Adolescent Trichotillomania Impact Project: Descriptive psychopathology, comorbidity, functional impairment, and treatment utilization. *Journal of Developmental and Behavioral Pediatrics*, *29*(6), 493–500.

Franklin, M. E., & Tolin, D. F. (2007). *Treating trichotillomania: Cognitive-behavioral therapy for hairpulling and related problems.* New York: Springer.

Friman, P. C., & Hove, G. (1987). Apparent covariation between child habit disorders: Effects of successful treatment for thumb sucking on untargeted chronic hair pulling. *Journal of Applied Behavioral Analysis*, *20*(4), 421–425.

Gabriel, A. (2001). A case of resistant trichotillomania treated with risperidone-augmented fluvoxamine [Letter to the Editor]. *Canadian Journal of Psychiatry*, *46*(3), 285–286.

Gadde, K. M., Wagner, H. R., Conner, K. M, & Foust, M. S. (2007). Escitalopram treatment of trichotillomania. *International Clinical Psychopharmacology*, *22*(1), 39–41.

Gluhoski, L. (1995). A cognitive approach for treating trichotillomania. *Journal of Psychotherapy Practice & Research, 4*(4), 277–285.

Grant, J. E., Odlaug, B. L., & Kim, S. W. (2009). *N*-acetylcysteine, a glutamate modulator, in the treatment of trichotillomania: A double-blind, placebo-controlled study. *Archives of General Psychiatry*, *66*(7), 756–763.

Gray, J. J. (1979). Positive reinforcement and punishment in the treatment of childhood trichotillomania. *Journal of Behavior Therapy and Experimental Psychiatry*, *10*(2), 125–129.

Gross, A. M., Farrar, M. J., & Liner, D. (1982). Reduction of trichotillomania in a retarded cerebral palsied child using over-correction, facial screening and differential reinforcement of other behavior. *Education and Treatment of Children*, *5*, 133–140.

Gupta, S., & Freimer, M. (1993). Trichotillomania, clomipramine, topical steroids [Letter]. *American Journal of Psychiatry*, *150*(3), 524.

Guy, W. (1976). *ECDEU assessment manual for psychopharmacology* (DHHS Publication No. ADM 76-338). Washington, DC: U.S. Government Printing Office.

Hallopeau, H. (1889). Alopecie par grattage (trichomanie ou trichotillomanie). *Annales de Dermatologie et de Syphiligraphie*, *10*, 440–441.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change.* New York: Guilford Press.

Himle, J. A., Perlman, D. M., & Lokers, L. M. (2008). Prototype awareness enhancing and monitoring device for trichotillomania. *Behaviour Research and Therapy*, *46*(10), 1187–1191.

Hoogduin C. A. L., Hagenaars, M. A., Minnen, A. van, & Keijsers, G. P. J. (2004). Protocollaire behandeling van patiënten met ongewenste gewooten: Zelfcontroleprocedures [Manualized treatment of nervous habits: Self-control procedures]. In G. P. J. Keijsers, A van Minnen, & C. A. L. Hoogduin (Eds.), *Protcallaire behandelingen in de ambulante geestlijke gezondheidszorg,* Deel 1 (2nd ed., pp. 261–278). Houten, Netherlands: Bohn Stafleu Van Loghum.

Horne, D. J. (1977). Behaviour therapy for trichotillomania. *Behaviour Research and Therapy*, *15* (2), 192–196.

Hussain, S. H. (1992). Trichotillomania. *Journal of the Pakistan Medical Association, 42*(1), 19–20.

Inglesias, A. (2003). Hypnosis as a vehicle for choice and self-agency in the treatment of children with trichotillomania. *American Journal of Clinical Hypnosis*, *46*(2), 129–137.

Jeffreys, D., & Burrows, G. (2008). Reversal of trichotillomania with aripiprazole. *Depression and Anxiety*, *25*(6), 37–40.

Keijsers, G. P. J., Van Minnen, A., Hoogduin, C. A. L., Klassen, B. N. W., Hendriks, M. J., & Tanis-Jacobs, J. (2006). Behavioural treatment of trichotillomania: Two-year follow-up results. *Behaviour Research and Therapy*, *44*(3), 359–370.

Keuthen, N. J., Aronowitz, B., Badenoch, B. A., & Wilhelm, S. (1999). Behavioral treatment for trichotillomania. In D. J. Stein, G. A. Christenson, & E. Hollander, (Eds.), *Trichotillomania*, (pp. 147–166). Washington, DC: American Psychiatric Press.

Keuthen, N. J., Flessner, C. A., Woods, D. W., Franklin, M. E., Stein, D. J., Cashin, S. E., et al. (2007a). Factor analysis of the Massachusetts General Hospital Hairpulling Scale. *Journal of Psychosomatic Research*, *62*, 707–709.

Keuthen, N. J., Jameson, M., Loh, R., Deckersbach, T., Wilhelm, S., & Dougherty D. D. (2007b). Open-label escitalopram treatment for pathological skin picking. *International Clinical Psychopharmacology*, *22*(5), 268–274.

Keuthen, N. J., O'Sullivan, R. L., Goodchild, P., Rodriquez, B. A., Jenike, M. A., & Baer, L. (1998a). Behavior therapy and pharmacotherapy for trichotillomania: Choice of treatment, patient acceptance, and long-term outcome. *CNS Spectrums, 3*(10), 72–78.

Keuthen, N. J., O'Sullivan, R. L., Goodchild, P., Rodriquez, B. A., Jenike, M. A., & Baer, L. (1998b). Retrospective review of treatment outcome for 63 patients with trichotillomania. *American Journal of Psychiatry*, *155*(4), 560–561.

Keuthen, N. J., O'Sullivan, R. L., Ricciardi, J. N., Shera, D., Savage, C. R., Borgmann, A. S., et al. (1995). The Massachusetts General Hospital (MGH) Hairpulling Scale, 1: Development and factor analysis. *Psychotherapy and Psychosomatics*, *64*(3–4), 141–145.

Keuthen, N. J., Stein, D. J., & Christenson, G. A. (2001) *Help for hair pullers: Understanding and coping with trichotillomania*. Oakland, CA: New Harbinger Press.

Khouzam, H. R., Battista, M. A., & Byers, P. E. (2002). An overview of trichotillomania and its response to treatment with quetiapine. *Psychiatry*, *65*(3), 261–270.

Knell, S. M., & Moore, D. J. (1988). Childhood trichotillomania treated indirectly by punishing thumb sucking. *Journal of Behavior Therapy and Experimental Psychiatry*, *19*(4), 303–310.

Koran, L. M., Ringold, A., & Hewlett, W. (1992). Fluoxetine for trichotillomania: An open clinical trial. *Psychopharmacology Bulletin*, *28*(2), 145–149.

Krishnan, R. R., Davidson, J., & Miller, R. (1984). MAO inhibitor therapy in trichotillomania associated with depression: Case report. *Journal of Clinical Psychiatry*, *45*(6), 267–268.

Leonard, H. L., Swedo, S. E., Rapoport, J. L., Koby, E. V., Lenane, M. C., Cheslow, D. L., et al. (1989). Treatment of obsessive-compulsive disorder with clomipramine and desipramine in children and adolescents: A double-blind crossover comparison. *Archives of General Psychiatry*, *46*(12), 1088–1092.

Lerner, J., Franklin, M. E., Meadows, E. A., Hembree, E., & Foa, E. B. (1998). Effectiveness of a cognitive-behavioral treatment program for trichotillomania: An uncontrolled evaluation. *Behavior Therapy*, *29*(1), 157–171.

Levine, B. A. (1976). Treatment of trichotillomania by covert sensitization. *Journal of Behavior Therapy and Experimental Psychiatry*, *7*(1), 75–76.

Lochner, C., Seedat, S., Niehaus, D. J. H, & Stein, D. J. (2006). Topiramate in the treatment of trichotillomania: An open-label pilot study. *International Clinical Psychopharmacology*, *21*(5), 255–259.

Mahr, G. (1993). Fenfluramine and trichotillomania. *Psychosomatics*, 34(3), 284.

Mancini, C., Van Amerigan, M., Patterson, B., Simpson, W., & Truong, C. (2009). Trichotillomania in youth: A retrospective case series. *Depression and Anxiety*, *26*(7), 661–665.

Mansueto, C. S., Golomb, R. G., Thomas, A. M., & Stemberger, R. M. T. (1999). A comprehensive model for behavioral treatment of trichotillomania. *Cognitive and Behavioral Practice*, *6*, 23–43.

Mastellone, M. (1974). Aversion therapy: A new use for the old rubber band. *Journal of Behavior Therapy and Experimental Psychiatry*, *5*(3), 311–312.

Matson, J. L., Stephens, R. M., & Smith, C. (1978). Treatment of self-injurious behavior with overcorrection. *Journal of Mental Deficiency Research*, *22*(3), 175–178.

Moretti, M. (2008). Trichotillomania and comorbidity—lamotragine in a new perspective. *Neuropsychopharmacologia Hungarica*, *10*(4), 201–212.

Ninan, P. T. (2000). Conceptual issues in trichotillomania, a prototypical impulse control disorder. *Current Psychiatry Reports*, *2*(1), 72–75.

Ninan, P. T., Knight, B., Kirk, L., Rothbaum, B. O., Kelsey, J., & Nemeroff, C. B. (1998). A controlled trial of venlafaxine in trichotillomania: Interim phase I results. *Psychopharmacology Bulletin, 34*(2), 221–224.

Ninan, P. T., Rothbaum, B. O., Marsteller, F. A., Knight, B. T., & Eccard, M. B. (2000). A placebo-controlled trial of cognitive-behavioral therapy and clomipramine in trichotillomania. *Journal of Clinical Psychiatry*, *61*(1), 47–50.

Odlaug, B. L., & Grant, J. E. (2007). *N*-acetyl cysteine in the treatment of grooming disorders. *Journal of Clinical Psychopharmacology*, *27*(2), 227–229.

O'Sullivan, R. L., Christenson, G. A., & Stein, D. J. (1999). Pharmacotherapy of trichotillomania. In D. J. Stein, G. A. Christenson, & E. Hollander (Eds.), *Trichotillomania* (pp. 93–123). Washington, DC: American Psychiatric Press.

O'Sullivan, R. L., Keuthen, N. J., Hayday, C. F., Ricciardi, J. N, Buttolph, M. L., Jenike, M. A., et al. (1995). The Massachusetts General Hospital (MGH) Hairpulling Scale: 2. Reliability and validity. *Psychotherapy and Psychosomatics*, *64*(3–4), 146–148.

O'Sullivan, R. L., Keuthen, N. J., Rodriguez, D., Goodchild, P., Christenson, G. A., & Rauch, S. L. (1998). Venlafaxine treatment of trichotillomania: An open series of ten cases. *CNS Spectrums*, *3*(10), 56–63.

Özcan, Y. D., Özcan, H., & Ensari, H. (2003). Efficacy of hydroxyzine in the treatment of trichotillomania: An open-label study. *Klinic Psikofarmakoloji Bülteni*, *13*(1), 19–22.

Pathak, S., Danielyan A., & Kowatch, R. A. (2004). Successful treatment of trichotillomania with olanzapine augmentation in an adolescent. *Journal of Child and Adolescent Psychopharmacology*, *14*(1), 153–154.

Perciaccannte, M., & Perciaccannte, R. G. (1993). Progestin treatment of obsessive-compulsive disorder. *Psychosomatics*, *34*(3), 284–285.

Pigott, T., L'Heueux, F., Grady, T., Bernstein, S., Dubbert, B., Rubenstein, C., et al. (1992). Controlled comparison of clomipramine and fluoxetine in trichotillomania. In *Abstracts of panels and posters of the 31st annual meeting of the American College of Neuropsychopharmacology*, San Juan, Puerto Rico, p. 157.

Pollard, C. A., Ibe, I. O., Kronjanker, D. N., Kitchen, A. D., Bronson, S. S., & Flynn, T. M. (1991). Clomipramine treatment of trichotillomania: A follow-up report on four cases. *Journal of Clinical Psychiatry*, *52*(3), 128–130.

Potenza, M. N., Wasylink, S., Epperson, C. N., & McDougle, C. J. (1998). Olanzapine augmentation of fluoxetine in the treatment of trichotillomania. *American Journal of Psychiatry*, *155*(9), 1299–1300.

Ravindran, A. V., Tran, V., & Van Ameringen, M. (2004). Trichotillomania: Effective treatment with quetiapine: A case series [Abstract P02.240]. 24th International College of Neuropsychopharmacology (CINP) Congress (Paris), 47.

Reeve, E. A., Bernstein, G. A., & Christenson, G. A. (1992). Clinical characteristics and psychiatric comorbidity in children with trichotillomania. *Journal of the American Academy of Child & Adolescent Psychiatry*, *31*(1), 132–138.

Reid, T. L. (1992). Treatment of generalized anxiety disorder and trichotillomania with buspirone. *American Journal of Psychiatry*, *149*(4), 573–574.

Ristvedt, S. L., & Christenson, G. A. (1996). The use of pharmacological pain sensitization in the treatment of repetitive hairpulling. *Behaviour Research and Therapy*, *34*(8), 647–648.

Robiner, W. N., Edwards, P. E., & Christenson, G. A. (1999). Hypnosis in the treatment of trichotillomania. In D. J. Stein, G. A. Christenson, & E. Hollander (Eds.), *Trichotillomania* (pp. 167–199). Washington, DC: American Psychiatric Press.

Rothbaum, R. O, & Ninan, P. T. (1999). Manual for the cognitive-behavioral treatment of ttrichotillomania. In D. J. Stein, G. A. Christenson, & E. Hollander (Eds.), *Trichotillomania* (pp. 263–284). Washington, DC: American Psychiatric Press.

Rothbaum, R. O., Opdyke, D. C., & Ninan, P. T. (1999). Assessment of trichotillomania. In D. J Stein, G. A. Christenson, & E. Hollander (Eds.), *Trichotillomania* (pp. 285–298). Washington, DC: American Psychiatric Press.

Sanchez, V. (1979). Behavioral treatment of chronic hair pulling in a two-year-old. *Journal of Behavior Therapy and Experimental Psychiatry*, *10*(3), 241–245.

Saper, B. (1971). A report on behavior therapy with outpatient clinic patients. *Psychiatric Quarterly*, *45*(2), 209–215.

Seedat, S., Stein, D. J., & Harvey, B. H. (2001). Inositol in the treatment of trichotillomania and compulsive skin picking. *Journal of Clinical Psychiatry*, *62*(1), 60–61.

Şentürk, V., & Tanrıverdi, N. (2002). Resistant trichotillomania and resperidone. *Psychosomatics*, *43*(5), 429–430.

Sonne, S., Rubey, R., Brady, K., Malcolm, R., & Morris, T. (1996). Naltrexone treatment of self-injurious thoughts and behaviors. *Journal of Nervous and Mental Disease, 184*(3), 192–195.

Stabler, B., & Warren, A. B. (1974). Behavioral contracting in treating trichotillomania: A case note. *Psychololological Reports*, *34*(2), 401–402.

Stanley, M. A., Bowers, T. C., Swann, A. C., & Taylor, D. J. (1991). Treatment of trichotillomania with fluoxetine. *Journal of Clinical Psychiatry*, *52*(6), 282.

Stanley, M. A., Breckenridge, J. K., Swann, A. C., Freeman, B. E, & Reich, L. (1997). Fluvoxamine treatment of trichotillomania. *Journal of Clinical Psychopharmacology*, *17*(4), 278–283.

Stanley, M.A., & Mouton, S. G. (1996). Trichotillomania treatment manual. In V. B. Van Hasselt & M. Hersen (Eds.), *Sourcebook of psychiatric treatment manuals for adult disorders* (pp. 657–687). New York: Plenum Press.

Stanley, M. A., Prather R. C., Wagner, A. L., Davis, M. L., & Swann, A. C. (1993). Can the Yale-Brown Obsessive Compulsive Scale be used to assess trichotillomania? A preliminary report. *Behaviour Research and Therapy*, *31*(2), 171–178.

Stein, D. J., Bouwer, C., & Maud, C. M. (1997). Use of the selective serotonin reuptake inhibitor citalopram in treatment of trichotillomania. *European Archives of Psychiatry and Clinical Neuroscience*, *247*(4), 234–236.

Stein, D. J., & Hollander, E. (1992). Low-dose pimozide augmentation of serotonin reuptake blockers in the treatment of trichotillomania. *Journal of Clinical Psychiatry*, *53*(4), 123–126.

Stevens, M. J. (1984). Behavioral treatment of trichotillomania. *Psychological Reports*, *55*(3), 987–990.

Stewart, R. S., & Nejtek, V. A. (2003). An open-label, flexible-dose study of olanzapine in the treatment of trichotillomania. *Journal of Clinical Psychiatry*, *64*(1), 49–52.

Streichenwein, S. M., & Thornby, J. I. (1995). A long-term, double-blind, placebo-controlled crossover trial of the

efficacy of fluoxetine for trichotillomania. *American Journal of Psychiatry*, *152*(8), 1192–1196.

Sunkureddi, K., & Markovitz, P. (1993). Trazodone treatment of obsessive-compulsive disorder and trichotillomania. *American Journal of Psychiatry*, *150*(3), 523–524.

Swedo, S. E., Lenane, M. C., & Leonard, H. L. (1993). Long-term treatment of trichotillomania (hair pulling). *New England Journal of Medicine*, *329*(2) 141–142.

Swedo, S. E., Leonard, H. L., Rapoport, J. L., Lenane, M. C., Goldberger, E. L., & Cheslow D. L. (1989). A double-blind comparison of clomipramine and desipramine in the treatment of trichotillomania (hair pulling). *New England Journal of Medicine*, *321*(8), 497–501.

Tayler, J. G. (1963). A behavioural interpretation of obsessive-compulsive neurosis. *Behaviour Research and Therapy*, *1*(2–4), 237–244.

Taylor, D. V., Hetrick, W. P., Neri, C. L. Touchette, P., Barron, J. L., & Snadman, C. A. (1991). Effect of naltrexone upon self-injurious behavior, learning and activity: A case study. *Pharmacology Biochemistry and Behavior*, *40*(1), 79–82.

Tolin, D. F., Diefenbach, G. J., Flessner, C. A., Franklin, M. E., Keuthen, N. J., Moore, P., et al. (2008). The Trichotillomania Scale for Children: Development and validation. *Child Psychiatry and Human Development*, *39*(3), 331–349.

Tolin, D. F., Franklin, M. E., Diefenbach, G. J., Anderson, E., & Meunier, S. A. (2007). Pediatric trichotillomania: Descriptive psychopathology and an open trial of cognitive behavioral therapy. *Cognitive Behaviour Therapy*, *36*(3), 129–144.

Twohig, M. P., & Woods, D. W. (2004). A preliminary investigation of acceptance and commitment therapy and habit reversal as a treatment for trichotillomania. *Behavior Therapy*, *35*(4), 803–820.

Üçok, A., & Gaebel, W. (2008). Side effects of atypical antipsychotics: A brief overview. *World Psychiatry, 7*(1), 58–62.

Van Ameringen, M., Mancini C., Oakman, J. M., & Farvolden, P. (1999). The potential role of haloperidol in the treatment of trichotillomania. *Journal of Affective Disorders*, *56*(2–3), 219–226.

Van Ameringen, M., Mancini, C., Patterson, B., Bennett, M., & Oakman, J. (2010). A randomized double-blind, placebo-controlled trial of olanzapine in the treatment of trichotillomania. *Journal of Clinical Psychiatry, 71*(10), 1336–1343.

van Minnen, A., Hoogduin, K. A. L., Keijsers, G. P. J., Hellenbrand, I., & Hendriks, G. J. (2003). Treatment of trichotillomania with behavioral therapy or fluoxetine: A randomized, waiting-list controlled study. *Archives of General Psychiatry*, *60*(5), 517–522.

Volavka, J., Neziroglu, F., & Yaryura-Tobias, J. A. (1985). Clomipramine and imipramine in obsessive-compulsive disorder. *Psychiatry Research*, *14*(1), 83–91.

White, S. (1990). Naltrexone for treatment of acral lick dermatitis in dogs. *Journal of the American Veterinary Medical Association, 196*(7), 1073–1076.

Winchel, R. M., Jones, J. S., Molcho, A., Parsons, B., Stanley, B., & Stanley, M. (1992a). The Psychiatric Institute Trichotillomania Scale (PITS). *Psychopharmacology Bulletin*, *28*(4), 463–476.

Winchel, R. M., Jones, J. S., Stanley, B., Molcho, B., & Stanley, M. (1992b). Clinical characteristics of trichotillomania and its response to fluoxetine. *Journal of Clinical Psychiatry*, *53*(9), 304–308.

Woods, D. W., Flessner, C. A., Franklin, M. E., Keuthen, N. J., Goodwin, R. D., Stein, D. J., et al. (2006a). The Trichotillomania Impact Project (TIP): Exploring phenomenology, functional impairment and treatment utilization. *Journal of Clinical Psychiatry*, *67*(12), 1877–1888.

Woods, D. W., Wetterneck, C. T., & Flessner, C. A. (2006b). A controlled evaluation of acceptance and commitment therapy plus habit reversal for trichotillomania. *Behaviour Research and Therapy*, *44*(5), 639–656.

Wulfsohn, D., & Barling, J. (1978). From external to self-control: Behavioral treatment of trichotillomania in an eleven-year-old girl. *Psychological Reports*, *42*(3 Pt. 2), 1171–1174.

Zalsman, G., Hersmesh, H., & Sever, J. (2001). Hypnotherapy in adolescents with trichotillomania: Three cases. *American Journal of Clinical Hypnosis, 44*(1), 63–68.

CHAPTER

24 Assessment and Treatment of Kleptomania

Jon E. Grant, Brian L. Odlaug, *and* Suck Won Kim

Abstract

Kleptomania, a disabling impulse control disorder, is characterized by repetitive and uncontrollable theft of items that are of little if any use. Kleptomania often goes undiagnosed or is misdiagnosed as a mood disorder, obsessive-compulsive disorder, or a substance use disorder. Unlike typical shoplifters, individuals with kleptomania steal for symptomatic relief rather than personal gain. Although the etiology of kleptomania is unknown, various biological and psychosocial theories may explain why some individuals develop kleptomania. Although cognitive behavioral therapy has shown early promise in treating kleptomania in case reports, the only controlled data for treatment involve the use of the opioid antagonist naltrexone.

Keywords: comorbidity, impulse control disorders, kleptomania, shoplifting, stealing, theft, treatment

Introduction

First coined in 1816 by the Swiss physician Andre Matthey, the term *klopemanie* was derived from the Greek words *kleptein* ("to steal") and *mania* (insanity) to describe persons who could not control their stealing behavior. In 1838, Jean-Etienne Esquirol wrote the first detailed description of this seemingly nonvolitional and irresistible behavior (Abelson, 1989; Esquirol, 1838). This treatise on kleptomania was important, as it distinguished a person with this disorder from those who steal due to a lack of moral character.

The first *Diagnostic and Statistical Manual of Mental Disorders* (DSM-I) did not include kleptomania as a formal diagnostic illness but rather as a supplementary term (APA, 1952). Kleptomania was left out of the DSM-II altogether (APA, 1968). In 1980, the DSM-III categorized kleptomania as an impulse control disorder not elsewhere classified (APA, 1980), the same clinical diagnostic category it currently occupies in the DSM-IV-TR. As an impulse control disorder, kleptomania is currently classified in DSM-IV-TR with pathological gambling, pyromania, intermittent explosive disorder, and trichotillomania (APA, 2000). Although included in DSM-IV, kleptomania is still a poorly understood disorder and has received very little empirical study.

Diagnosis

The DSM-IV-TR defines kleptomania as the following: "A) Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value; B) Increasing sense of tension immediately before committing the theft; C) Pleasure, gratification, or release at the time of committing the theft; D) The stealing is not committed to express anger or vengeance and is not in response to a delusion or a hallucination; and E) The stealing is not better accounted for by Conduct Disorder, a Manic Episode, or Antisocial Personality Disorder" (APA, 2000, p. 669).

Only one instrument has been used to standardize the diagnosis of kleptomania: the Structured

Clinical Interview for Kleptomania (SCI-K; Grant et al., 2006c). The SCI-K has demonstrated excellent test-retest [phi coefficient = 0.956 (95% CI = .937, .970)] and interrater reliability [phi coefficient = 0.718 (95% CI = .506, .848)] in the diagnosis of kleptomania. Excellent concurrent validity was observed with a self-report measure using DSM-IV kleptomania criteria [phi coefficient = 0.769 (95% CI = .653, .850)]. Discriminant validity was observed with a measure of depression [point biserial coefficient = –.020 (95% CI = –.205, .166)]. The SCI-K also demonstrated both high sensitivity and specificity based on longitudinal assessment.

Failure to Diagnose

Although kleptomania may be fairly common among certain populations (e.g., psychiatric patients), it usually goes undiagnosed. There are many reasons why this severely distressing behavior is not diagnosed.

Shame and Secrecy

Shame and secrecy are fundamental to kleptomania. This embarrassment and shame appear to explain, in part, why so few patients will volunteer information regarding this behavior unless specifically asked (Grant et al., 2005). Often related to the shame and secrecy is the patient's misunderstanding of what a mental health clinician is required by law to report. Patients suffering from kleptomania may believe that the clinician is required to report their illegal behaviors. Clinicians therefore may want to inform patients at the outset of the evaluation about what they do and do not have to report.

Patient Lack of Knowledge

Another possible reason for the failure to diagnose kleptomania is that patients often do not know that their behavior is a recognizable disorder with treatment options. Using the kleptomania criteria set forth in DSM-IV (APA, 2000), clinicians can inform patients that if stealing or the urge to steal preoccupies them and if the urge or behavior causes distress or dysfunction, then a more complete discussion of the stealing behavior should occur.

Clinician Lack of Knowledge

Few health care professionals have education or training in kleptomania. In addition, clinicians may have many of the same biases about stealing as do patients. For example, clinicians may see themselves as potential victims of the illegal behaviors associated with impulse control disorders—that is, clinicians pay more for items due to shoplifting—and therefore may not feel that the behavior is an illness and deserving of treatment.

Misdiagnosis

Kleptomania has many phenomenological similarities to other disorders (Grant & Potenza, 2004). This overlap in clinical presentation between kleptomania and other psychiatric disorders makes diagnosis difficult. When this overlap is combined with lack of knowledge regarding the clinical characteristics of kleptomania, the chance for misdiagnosis is considerable. Some of the more common misdiagnoses will now be discussed.

Mood Disorder

Bipolar manic episodes are characterized by impulsive behaviors (APA, 2000). These manic behaviors may include shoplifting, as seen in kleptomania. In addition, individuals with kleptomania have high rates of co-occurring bipolar disorder (10% to 60% in kleptomania; Grant & Kim, 2002a; McElroy et al., 1991a). During a manic episode, however, people often exhibit multiple symptoms of mood dysregulation—excess energy, distractibility, elevation of mood or irritability (APA, 2000)—whereas the behavior of kleptomania usually has none of those associated symptoms. In addition, the behaviors during a manic episode may last for only a few days or a couple of weeks, whereas shoplifting in kleptomania tends to be more consistent. The DSM-IV excludes a diagnosis of kleptomania when the behavior occurs exclusively during a manic episode (APA, 2000). Individuals with kleptomania may also suffer from bipolar disorder. It is then important for the clinician to determine if the shoplifting occurs only during mania or possibly simply worsens during a manic episode.

Many patients may report that they shoplift only when feeling depressed. Rates of depression are elevated in individuals with kleptomania (McElroy et al., 1991a). These patients, therefore, may also be given a diagnosis of bipolar disorder, mixed state, or unipolar depression. In fact, there may be a subtype of kleptomania patients who find mood elevation in their behavior and therefore "self-medicate" with the impulsive behavior (Fishbain, 1987). In cases where the behavior is secondary to mood, the underlying mood should be treated, but the kleptomania may need additional treatment as well. Although the behavior may have been started to self-medicate the mood, the behavior may be maintained by a different neurobiological mechanism.

Obsessive-Compulsive Disorder

There is often significant clinical overlap between obsessive-compulsive disorder and kleptomania (Grant & Potenza, 2006). Patients may even refer to themselves as "obsessive" or engaged in shoplifting "compulsively." Although patients with kleptomania, like those with obsessive-compulsive disorder, may shoplift repetitively and be preoccupied with thoughts of shoplifting, the key difference is that people with kleptomania gain pleasure from the behavior (Grant & Potenza, 2006). Obsessive-compulsive behaviors are generally done to alleviate anxiety.

Substance Use Disorders

Substance use disorders frequently co-occur in individuals with kleptomania (20%–45%; Baylé et al., 2003; McElroy et al., 1991a). It is important for clinicians to determine (1) whether the substance use problem results in shoplifting, perhaps through disinhibition (e.g., shoplifting only when intoxicated) or modulation of particular brain pathways (e.g., methamphetamine may result in shoplifting); (2) whether the shoplifting, and the shame and the desire to escape, cause the substance use (e.g., shame over shoplifting leads to frequent drinking); or (3) whether they are two related but independent problems.

Personality Disorders

Although rates of personality disorders in individuals with kleptomania have not been rigorously examined, one study suggests that personality disorders may be present at higher rates than seen in the community—most commonly paranoid and borderline personality disorders (Grant, 2004). The clinician must therefore determine if the shoplifting merits an independent diagnosis of kleptomania or is secondary to a personality disorder. This may be particularly difficult in the case of borderline personality and antisocial personality disorders that are characterized by impulsivity (APA, 2000). Clinicians should be aware that kleptomania often co-occurs with personality disorders, and clear separation of the behaviors may not be possible at the first assessment. Continued assessments may be necessary to determine to what extent these various disorders interact in a patient.

Distinguishing Kleptomania from Typical Shoplifting

Individuals with kleptomania differ from ordinary shoplifters in that they do not steal for personal gain, but rather for symptomatic relief. It is estimated that more than $13 billion worth of goods are stolen from retailers each year, which translates into more than $35 million per day (National Association for Shoplifting Prevention, 2009). The vast majority of shoplifters are described as amateurs with sporadic activity, with no known history of criminal activity, and who steal for their own consumption rather than for resale.

Rates of kleptomania among people who are arrested for shoplifting have ranged from 0% to 8% (McElroy et al., 1991b). A study that compared kleptomaniacs to shoplifters interviewed directly after apprehension found that 58% of the shoplifters were male compared to only 32.4% of kleptomania patients (Sarasalo et al., 1987). The mean age of shoplifters was 27 years and that of kleptomaniacs was 41 years. Although none of the shoplifters met DSM criteria for kleptomania, approximately one-fifth had not stolen for personal use and had eventually discarded the object. The study also found that both groups reported the same degree of impulsivity and "a feeling of not being oneself." On the other hand, kleptomaniacs reported a relatively greater number of previous thefts compared to shoplifters, which supports the compulsive aspect of kleptomania.

Assessment of Stealing in Children and Adolescents

Both typical shoplifting and kleptomania may start at a relatively early age. A young child generally has little, if any, concept of stealing; for him or her, desiring or wanting means possession of the object. By the age of 6 or 7, children begin to realize that they are doing something wrong when they take something that doesn't belong to them. Children may steal because they are unhappy, lonely, jealous, fearful, or craving attention. For older children and adolescents, stealing can be used to gain acceptance from a group, but it is also a strong predictor of future delinquency and a marker for families lacking in warmth and personal stimulation. Strong attachment to parents decreases involvement in shoplifting.

Overall, studies have shown that roughly 40% of apprehended shoplifters are adolescents. A study involving almost 1700 adolescents found that 37% reported shoplifting at least once in the prior 12 months (Cox et al., 1990). The percentage of respondents peaked at around 10th grade and then declined, which is consistent with official crime statistics. One hypothesis concerning adolescent theft is that it is a function of immaturity during a stressful transition

to adulthood, an inability to purchase certain items, and increased opportunity (the greatest gain of independence occurs at around age 16, when most adolescents are allowed to drive and work; Cox et al., 1990). On the other hand, adolescents report that they shoplift because of the novelty and risk involved, for social reasons, and out of desire for the product. Additionally, no relationship has been found between family occupational status and adolescent shoplifting. How many of these adolescent shoplifters currently suffer from, or will develop, kleptomania is not clear.

Although the age of onset of kleptomania appears to be most often late adolescence (Goldman, 1991; Grant & Kim, 2002b; McElroy et al, 1991b; Presta et al., 2002), there is little information on how kleptomania presents in adolescents. Based on case reports of adolescent kleptomania (Feeney & Klykylo, 1997; Grant & Kim, 2002c), there is some evidence that the current DSM-IV criteria apply to adolescents who report being unable to control their shoplifting. More research, however, is needed to understand the presentation of kleptomania among adolescents.

Treatment

Biological Theories

A growing body of literature implicates multiple neurotransmitter systems (e.g., serotonergic, dopaminergic, opioidergic), as well as familial and inherited factors, in the pathophysiology of the motivated behaviors associated with impulse control disorders such as kleptomania (Grant et al., 2006a). One central aspect of motivated behaviors involves the ventral striatum, a brain region that includes the nucleus accumbens. The ventral striatum is important for controlling motivated behavior that is largely determined through a series of cortical-striatal-thalamic-cortical loops. Although a wide array of neurotransmitters serves to coordinate information processing within this network, arguably the best-characterized neurotransmitters that influence motivated behavior are serotonin, dopamine, and the opioid system.

SEROTONIN AND INHIBITION

Evidence for serotonergic involvement in impulse control disorders comes in part from studies of platelet monoamine oxidase B activity, which correlates with cerebrospinal fluid levels of 5-hydroxyindoleacetic acid and is considered a peripheral marker of serotonin (5-HT) function (Coccaro et al., 1990; Linnoila et al., 1983). Patients with kleptomania report significant increases in impulsivity and risk taking compared to normal controls (Baylé et al., 2003; Grant & Kim 2002d), and diminished inhibitory mechanisms may underlie the risk-taking behavior of kleptomania. Decreased levels of serotonin have long been associated with a variety of adult risk-taking behaviors (Moreno et al., 1991; Virkunnen et al., 1994). One study examined the platelet serotonin transporter in 20 patients with kleptomania. The level of the platelet 5-HT transporter, evaluated by means of binding of 3H-paroxetine, was lower in patients with kleptomania than in healthy control subjects (Marazziti et al., 2000), suggesting serotonergic dysfunction.

Neurocognitive assessment of women with kleptomania revealed, as a group, no significant deficits in tests of frontal lobe functioning when compared to normative values (Grant et al., 2007). Individuals with more severe symptoms of kleptomania, however, had significantly below-average scores on at least one measure of executive functioning. Significantly higher rates of cognitive impulsivity were found in kleptomania subjects compared to a control group of psychiatric patients without kleptomania. This greater impulsivity on the part of kleptomaniacs may in part be mediated through serotonergic function.

Damage to the orbitofrontal-subcortical circuits of the brain has been reported to result in kleptomania. Neuroimaging studies have demonstrated decreased white matter microstructural integrity in the ventral-medial frontal brain regions of individuals with kleptomania compared to controls (Grant et al., 2006b). These images are consistent with findings of increased impulsivity in kleptomaniacs. These studies also support the hypothesis that at least some individuals with kleptomania may not be able to control their impulse to steal.

DOPAMINE AND REWARD DEFICIENCY

Dopaminergic systems influencing rewarding and reinforcing behaviors have also been implicated in impulse control disorders and may play a role in the pathogenesis of kleptomania. A hypothesized hypodopaminergic state involving multiple genes and environmental stimuli that puts an individual at high risk for multiple addictive, impulsive, and compulsive behaviors is one proposed mechanism (Blum et al., 2000). Alterations in dopaminergic pathways have been proposed as underlying the seeking of rewards (e.g., shoplifting) that triggers the release of dopamine and produces a feeling of pleasure (Blum et al., 2000). Furthermore, dopamine

release into the nucleus accumbens has been implicated in the translation of motivated drive into action (Chambers et al., 2003).

OPIOID SYSTEM, CRAVINGS, AND PLEASURE

The underlying biological mechanism of urge-based disorders may also involve the processing of incoming reward inputs by the ventral tegmental area-nucleus accumbens-orbital frontal cortex (VTA-NA-OFC) circuit (Hyman, 1993; Koob & Bloom, 1988; Mogenson et al., 1980). This circuit influences behavior by modulating animal and human motivation (e.g., urges, cravings). Dopamine may play a major role in the regulation of this region's functioning (Koob, 1992; Kuhar et al., 1991).

Urges linked to the experience of reward and pleasure represent an important clinical target in kleptomania. Patients with kleptomania report urges to steal (Grant & Kim, 2002b). Most report fairly frequent urges that result in theft—perhaps two times per week on average (Grant & Kim, 2002b). Many indicate that the act of stealing reduces the urges or the tension these urges produce (McElroy et al., 1991b). Although many report the urges as intrusive, the act of stealing is often a thrill for some, producing a pleasurable feeling (Goldman, 1991; Grant & Kim, 2002b). The mu-opioid system is believed to underlie urge regulation through the processing of reward, pleasure, and pain, at least in part via modulation of dopamine neurons in the mesolimbic pathway through gamma-aminobutyric acid interneurons (Potenza & Hollander, 2002).

One line of evidence supporting the role of the opioid system in the pathogenesis of kleptomania is found in the treatment literature. Studies of the opioid antagonist, naltrexone, in the treatment of kleptomania and other impulse control disorders have demonstrated its efficacy in reducing urges (Dannon et al., 1999; Grant & Kim, 2002a; Kim & Grant, 2001).

Psychological Theories

The etiology of kleptomania is most likely multifactorial (biological, psychological, sociocultural). Why do kleptomania patients continue to engage in a behavior that results in unneeded items when the possible repercussions are so devastating? Attempting to address this complex question, many psychological theories of kleptomania have been postulated: for example, gratification of unconscious sexual impulses (Goldman, 1991; McElroy et al., 1991a), castration anxiety (Levy, 1934), low self-esteem (Goldman, 1991), unresolved dependency (Allen, 1965), and masochism (Rado, 1933). There are no data, however, to confirm or refute these theories (Goldman, 1991). Focusing on the pleasure many patients derive from shoplifting, some have theorized that kleptomania results from an attempt to relieve feelings of depression through stimulation (Goldman, 1991; Gudjonsson, 1987; McElroy et al., 1991a). Risk-taking behavior may therefore produce an antidepressant effect for some patients (Fishbain, 1987; Goldman, 1991). It is possible that depressed individuals may engage in shoplifting to distract themselves from life stressors and unpleasant cognitions. Unlike drugs or alcohol, shoplifting leads to neither intoxication nor a directly impaired ability to function at work and, as such, may be an especially attractive means of escape. Persons who are depressed may also view the objects they steal as a means of achieving significant symptom relief and view the possibility of being apprehended as a relatively minor and theoretical setback. Ironically, problems resulting directly from shoplifting (e.g., embarrassment and shame from getting caught) may, in turn, lead to even more shoplifting in a misguided attempt at symptom management.

Because most people with depression do not shoplift, theories have been offered as to why some depressed people might engage in this behavior. One theory suggests that shoplifting is a symbolic attempt to make up for early deprivations or losses. The shoplifting may therefore be a symbolic compensation for an actual or perceived loss (Cupchick & Atcheson, 1983; Goldman, 1991). Interestingly, some support for this theory may be found in a study of parental bonding, which found that kleptomania patients reported significantly lower maternal and paternal care scores (i.e., parents' expression of affection) than normal controls (Grant & Kim, 2002d). Furthermore, a family study found that patients with kleptomania were more likely than normal controls to have a first-degree relative with an alcohol use disorder (Grant, 2003). Given evidence of increased psychiatric stress in the children of alcoholics (Sher, 1991), the examination of feelings of deprivation or loss in kleptomania patients merits further attention. Kleptomania patients may have low ego strength, and shoplifting may raise their self-esteem by creating a feeling of success when they leave a store without being apprehended. Early emotional deprivation may therefore play a role in the pathogenesis of kleptomania.

Behavioral models may also provide clues to the pathogenesis of kleptomania. From an operant standpoint, the acquisition of items without payment

that shoplifting provides creates a particularly pathogenic formulation. The quintessential positive reinforcer in kleptomania is the acquisition of items for nothing. The intermittent reinforcement (not always being able to shoplift because of store security, etc.) of kleptomanic behavior creates a schedule of reinforcement that is particularly resistant to extinction. Physiological arousal associated with the shoplifting (Goldman, 1991) may be yet another reinforcer that initiates and perpetuates the behavior.

Similarly, negative reinforcement (i.e., involving the removal of a punishing stimulus) hypothesizes that initiating but not completing a habitual behavior leads to uncomfortable states of arousal. Applied to kleptomania, this would imply that shoplifting is done to experience relief from the aversive arousal of urges. Even the self-medication theory of kleptomania may represent a negative reinforcement. This could account for the persistence of kleptomanic behavior despite being frequently apprehended.

In addition to behavioral reasons, there may be specific thinking errors that are directly linked to kleptomanic behavior: (1) believing that only shoplifting will reduce the urge or the depressive state; (2) selective memory (e.g., remembering the thrill of shoplifting while ignoring the shame and embarrassment of being apprehended; and (3) erroneous self assessment (i.e., that the person deserves to be caught stealing because he or she is intrinsically worthless). In addition, kleptomania patients score high on indices of impulsivity (Baylé et al., 2003; Grant & Kim, 2002c). Impulsive individuals may also be insensitive to internally generated cognitions focusing on restraint (McCown & Chamberlain, 2000).

These psychological theories should be understood, however, in the context of the possible biological explanations for kleptomania. Although many people shoplift sometime in their lives, it remains to be explained why all individuals who shoplift more than a few times do not succumb to disorder-inducing intermittent reinforcement contingencies (i.e., the positive reinforcement of acquiring items or the negative reinforcement of relief from aversive arousal of urges). One simple possibility is that individual differences in biological constraints surrounding reinforcement sensitivity might regulate the propensity to respond to the positive or negative reinforcement of shoplifting. That is, for some individuals, positive or negative reinforcement may have a more powerful influence on future kleptomanic behavior. A search for such individual *moderating variables* may allow us to refine both our psychological and biological understanding of operant processes in the etiology and maintenance of kleptomania.

Pharmacotherapy

No medication is currently approved by the Food and Drug Administration (FDA) for the treatment of kleptomania. Therefore, it is important to inform patients of any "off-label" use of medications for this disorder, as well as the empirical basis for considering medication treatment.

CASE REPORTS

Only case reports, two small case series, and one open-label study of pharmacotherapy have been performed for kleptomania. Various medications—tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs; Lepkifker et al., 1999), mood stabilizers, and opioid antagonists—have been examined for the treatment of kleptomania (Kim, 1998; McElroy et al., 1989). McElroy and coauthors (1991b) reported a treatment response in 10 of 20 patients with the following single agents: fluoxetine, nortriptyline, trazodone, clonazepam, valproate, and lithium. Other agents used successfully as monotherapy for kleptomania include fluvoxamine (Chong & Low, 1996) and paroxetine (Kraus, 1999).

Combinations of medications have also been effective in case reports: lithium plus fluoxetine (Burstein, 1992), fluvoxamine plus buspirone (Durst et al., 1997), fluoxetine plus alprazolam (McElroy et al., 1991b), fluvoxamine plus valproate (Kmetz et al., 1997), and fluoxetine plus imipramine (McElroy et al., 1991b).

The findings from case reports, however, have not been consistent. In fact, seven cases of fluoxetine, three cases of imipramine, two cases of lithium as monotherapy and two cases of lithium augmentation, four cases of tranylcypromine, and carbamazepine in combination with clomipramine all failed to reduce kleptomania symptoms (McElroy et al., 1991b). Additionally, some evidence suggests that SSRIs may actually induce kleptomania symptoms (Kindler et al., 1997).

One case series found that kleptomania symptoms respond to topiramate (Dannon, 2003). In a series of three patients treated with doses of 100 to 150 mg/day, all three patients achieved remission of kleptomania symptoms. Two of the patients were also taking an SSRI concomitantly with topiramate, and two had comorbid diagnoses of attention deficit hyperactivity disorder and panic disorder. In another

case series examining two subjects treated with naltrexone, both responded to the medication (Dannon et al., 1999).

OPEN-LABEL STUDIES

There have been two open-label trials of medication for kleptomania. In a 12-week open-label study, 10 subjects self-referred with kleptomania and free from other Axis I comorbid disorders were treated with naltrexone (dose range, 50 to 200 mg/day). All subjects had at least moderate urges to steal at the time of study entry. Naltrexone resulted in a significant decline in the intensity of urges to steal, stealing thoughts, and stealing behavior. The mean effective dose of naltrexone was 150 mg/day (Grant & Kim, 2002a).

In the second open-label study, 24 subjects received open-label escitalopram. After 7 weeks of treatment, escitalopram was shown to reduce shoplifting urges in 19 (79%) of them (Koran et al., 2007). Responders were then randomized to a double-blind discontinuation phase in which they received either active medication or placebo. At the end of this portion of the study, no significant differences were found between active medication and placebo; 50% of the subjects on placebo and 43% of those on active medication maintained their improvement from the open-label portion of the study.

DOUBLE-BLIND, PLACEBO-CONTROLLED STUDIES

Because of the hypothesized mechanism of action of naltrexone (i.e., modulation of mesolimbic dopamine; Matthews & German, 1984; Stewart, 1984) and the previous findings of naltrexone's ability to reduce urges in kleptomania (Grant & Kim, 2002a), one double-blind, placebo-controlled study was conducted to examine the possible efficacy of naltrexone. In this study, 25 subjects with kleptmania were enrolled. Significantly better results were observed for those assigned to naltrexone on the primary efficacy variable, the Yale-Brown Obsessive Compulsive Scale modified for Kleptomania (K-YBOCS) total scores (p = .001). A significant interaction of treatment effect by visit contrast was first detected after 6 weeks on active medication (p=.013). Cohen's effect size after 8 weeks (study endpoint) was 1.14. The mean effective dose of naltrexone was 116.7 (± 44.4) mg/day. A placebo response was detected during the first 4 weeks of the study, which then diminished. Remission of kleptomania symptoms was seen in eight naltrexone-treated subjects (66.7%) and in one (7.7%) subject on placebo (Fisher's exact = .003) (Grant et al., 2009). Although the study was only 8 weeks long, a naturalistic outpatient study demonstrated that subjects treated with naltrexone monotherapy for up to 3 years continued to report overall improvement (Grant, 2005).

PSYCHOTHERAPY

Psychoanalysis has resulted in some limited success for kleptomania symptoms, but usually with the addition of medications (Fishbain, 1988; Schwartz, 1992). Insight-oriented psychotherapy, however, has been unsuccessful in treating this disorder in 11 published cases (McElroy et al., 1991b).

Behavioral therapy appears to have resulted in successfully treated cases of kleptomania. Using covert sensitization combined with exposure and response prevention, Guidry (1975) reported on a young man who was able to reduce his stealing frequency. In a total of seven sessions over a 4-month period, the man imagined stealing as well as the consequences of stealing (being seen, caught, handcuffed, taken before a judge, embarrassment). In addition, the man went to stores and was asked to imagine that the store manager was observing him. He reduced his stealing behavior, although his urges to steal went unchanged.

In another case of covert sensitization, a young woman underwent five weekly sessions in which she was instructed to practice covert sensitization whenever she had urges to steal. She was able to then go for 14 months with only a single lapse in behavior and with no reported urges to steal (Gauthier & Pellerin, 1982). Another woman was instructed to have increasing nausea when tempted to steal, with imagery of vomiting associated with actual stealing (Glover, 1985). After four sessions over 8 weeks, the woman was able to go with only a single lapse in behavior over the next 19 months. And finally, aversive breath holding was used whenever a patient reported urges to steal (Keutzer, 1972). In combination with keeping a diary of urges to steal and six weekly sessions of therapy, the woman was able to significantly reduce the frequency of stealing.

Imaginal desensitization uses the idea of imagining the steps of stealing while maintaining a relaxed state. The patient then imagines the potential scene of stealing but also imagines her ability to not steal in that context. Undergoing fourteen 15-minutes sessions over 5 days, two patients reported complete remission of symptoms for a 2-year period (McConaghy & Blaszczynski, 1988).

And finally, learning to substitute alternative sources of satisfaction and excitement when the urges to steal occur has been successful in a single case report. The case involved a woman treated weekly for 5 months to assist her in finding alternative sources of excitement, pleasure, and self-fulfillment. She was able to report a 2-year period of symptom remission (Gudjonsson, 1987).

PHARMACOTHERAPY AND COMBINATION TREATMENTS

Although there have been no studies evaluating the efficacy of combined psychotherapy and medication for kleptomania, case reports have illustrated the benefit of combining medication with various therapies, such as aversion therapy, covert sensitization, and systematic desensitization. Successful examples of combined psychotherapy and pharmacology for the treatment of kleptomania include fluoxetine 40 mg/day combined with supportive psychotherapy; fluoxetine 40 mg/day combined with problem-oriented psychotherapy; fluoxetine 20 mg/day plus cognitive therapy; a combination of cognitive behavioral therapy, sertraline 50 mg/day, and a self-imposed shopping ban; and a combination of cognitive behavioral therapy and citalopram 40 mg/day (Aizer et al., 2004; Lepkifker et al., 1999; McNeilly & Burke, 1998).

Recommendations Based on Treatment Outcome Literature

The outcome data for the treatment of kleptomania are inconclusive. Only two controlled trials of pharmacotherapy have been reported. Based on this limited research, it appears that opioid antagonists, rather than SSRIs, should be the first-line treatment. Also, there is slightly more evidence supporting pharmacotherapy in the treatment of kleptomania than psychotherapy, but these data are extremely limited.

The research on treatment outcome in kleptomania contrasts sharply with the quantity and quality of studies in other impulse control disorders (e.g., pathological gambling). This state of affairs is probably attributable to the low prevalence of kleptomania and to clinical difficulties in treating individuals involved in illegal activities. Nevertheless, there is a great need for systematic studies for the treatment of this disorder. Such studies may need to involve collaboration across multiple treatment centers in view of the disorder's low prevalence. Given the existing data, it is not possible to construct evidence-based clinical recommendations regarding treatment.

Future Directions

Although there is an increasing understanding that a behavior such as shoplifting may reflect a distinct pathophysiology, evidence-based treatment options are limited. The current trend of using cognitive measures and brain imaging to better understand the pathophysiology of other psychiatric disorders needs to be more rigorously applied to elucidate the underlying pathophysiology of kleptomania. As pharmacotherapy and psychosocial treatment options advance to target more specific areas of brain dysfunction, the cognitive and imaging data will allow us to be more successful in treating this behavior. The neurobiological and psychological nature of kleptomania necessitates collaborative efforts among psychologists, psychiatrists, and the criminal justice system to improve early identification and treatment of this disorder.

References

Abelson, E. S. (1989). *When ladies go a-thieving: Middle-class shoplifters in the Victorian department store.* New York: Oxford University Press.

Aizer, A., Lowengrub, K., & Dannon, P. N. (2004). Kleptomania after head trauma: Two case reports and combination treatment strategies. *Clinical Neuropharmacology*, *27*, 211–215.

Allen, A. (1965). Stealing as a defense. Psychoanalytic Quarterly, *34*, 572–583.

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (DSM-I). Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.; DSM-II). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed., revised; DSM-III-R). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision; DSM-IV-TR). Washington, DC: Author.

Baylé, F. J., Caci, H., Millet, B., Richa, S., & Olié, J. P. (2003). Psychopathology and comorbidity of psychiatric disorders in patients with kleptomania. *American Journal of Psychiatry*, *160*(8), 1509–1513.

Blum, K., Braverman, E. R., Holder, J. M., Lubar, J. F., Monastra, V. J., Miller, D., Lubar, J. O., Chen, T. J., & Comings, D. E. (2000). Reward deficiency syndrome: A biogenetic model for the diagnosis and treatment of impulsive, addictive, and compulsive behaviors. *Journal of Psychoactive Drugs*, *32*(Suppl 1), 1–68.

Burstein, A. (1992). Fluoxetine-lithium treatment for kleptomania. *Journal of Clinical Psychiatry*, *53*(1), 28–29.

Chambers, R. A., Taylor, J. R., & Potenza, M. N. (2003). Developmental neurocircuitry of motivation in adolescence: A critical period of addiction vulnerability. *American Journal of Psychiatry*, *160*(6), 1041–1052.

Chong, S. A., & Low, B. L. (1996). Treatment of kleptomania with fluvoxamine. *Acta Psychiatrica Scandinavica*, *93*(4), 314–315.

Coccaro, E. F., Siever, L. J., Klar, H. M., Maurer, G., Cochrane, K., Cooper, T. B., Mohs, R. C., & Davis, K. L. (1990). Serotonergic studies in patients with affective and personality disorders: Correlates with suicidal and impulsive aggressive behavior. *Archives of General Psychiatry*, *46*(7), 587–599.

Cox, D., Cox, A., & Moschis, G. (1990). When consumer behavior goes bad: An investigation of adolescent shoplifting. *Journal of Consumer Research*, *17*, 149–159.

Cupchick, W., & Atcheson, J. D. (1983). Shoplifting: An occasional crime of the moral majority. *The Bulletin of the American Academy of Psychiatry and the Law*, *11*(4), 343–354.

Dannon, P. N. (2003). Topiramate for the treatment of kleptomania: A case series and review of the literature. *Clinical Neuropharmacology*, *26*(1), 1–4.

Dannon, P. N., Iancu, I., & Grunhaus, L. (1999). Naltrexone treatment in kleptomanic patients. *Human Psychopharmacology: Clinical and Experimental*, *14*, 583–585.

Durst, R., Katz, G., & Knobler, H. Y. (1997). Buspirone augmentation of fluvoxamine in the treatment of kleptomania. *Journal of Nervous and Mental Disease*, *185*(9), 586–588.

Esquirol, E. (1838). *Des Maladies Mentales*. Paris: Bailliere.

Feeney, D. J., & Klykylo, W. M. (1997). Treatment for kleptomania. *Journal of the American Academy of Child and Adolescent Psychiatry*, *36*(6), 723–724.

Fishbain, D. A. (1987). Kleptomania as risk-taking behavior in response to depression. *American Journal of Psychotherapy, 41*(4), 598–603.

Gauthier, J., & Pellerin, D. (1982). Management of compulsive shoplifting through covert sensitization. *Journal of Behavior Therapy and Experimental Psychiatry*, *13*(1), 73–75.

Glover, J. H. (1985). A case of kleptomania treated by covert sensitization. *British Journal of Clinical Psychology*, *24*(Pt 3), 213–214.

Goldman, M. J. (1991). Kleptomania: Making sense of the nonsensical. *American Journal of Psychiatry*, *148*(8), 986–996.

Grant, J. E. (2003). Family history and psychiatric comorbidity in persons with kleptomania. *Comprehensive Psychiatry*, *44*(6), 437–441.

Grant, J. E. (2004). Co-occurrence of personality disorders in persons with kleptomania: A preliminary investigation. *Journal of the American Academy of Psychiatry and the Law*, *34*, 395–398.

Grant, J. E. (2005). Outcome study of kleptomania patients treated with naltrexone: A chart review. *Clinical Neuropharmacology*, *28*(1), 11–14.

Grant, J. E., Brewer, J. A., & Potenza, M. N. (2006a). The neurobiology of substance and behavioral addictions. *CNS Spectrums*, *11*(12), 924–930.

Grant, J. E., Correia, S., & Brennan-Krohn, T. (2006b). White matter integrity in kleptomania: A pilot study. *Psychiatry Research*, *147*(2–3), 233–237.

Grant, J. E., & Kim, S. W. (2002a). An open label study of naltrexone in the treatment of kleptomania. *Journal of Clinical Psychiatry*, *63*(4), 349–356.

Grant, J. E., & Kim, S. W. (2002b). Clinical characteristics and associated psychopathology of 22 patients with kleptomania. *Comprehensive Psychiatry*, *43*(5), 378–384.

Grant, J. E., & Kim, S. W. (2002c). Adolescent kleptomania treated with naltrexone: A case report. *European Child and Adolescent Psychiatry, 11*, 92–95.

Grant, J. E., & Kim, S. W. (2002d). Temperament and early environmental influences in kleptomania. *Comprehensive Psychiatry*, *43*(3), 223–229.

Grant, J. E., Kim, S. W., & McCabe, J. (2006c). Structured Clinical Interview for Kleptomania (SCI-K): Preliminary validity and reliability testing. *International Journal of Methods in Psychiatric Research*, *15*(2), 83–94.

Grant, J. E., Kim, S. W., & Odlaug, B. L. (2009). A double-blind, placebo-controlled study of the opiate antagonist, naltrexone, in the treatment of kleptomania. *Biological Psychiatry*, *65*(7), 600–606.

Grant, J. E., Levine, L., Kim, D., & Potenza, M. N. (2005). Impulse control disorders in adult psychiatric inpatients. *American Journal of Psychiatry*, *162*(11), 2184–2188.

Grant, J. E., Odlaug, B. L., & Woziak, J. R. (2007). Neuropsychological functioning in kleptomania. *Behaviour Research and Therapy*, *45*(7), 1663–1670.

Grant, J. E., & Potenza, M. N. (2004). Impulse control disorders: Clinical characteristics and pharmacological management. *Annals of Clinical Psychiatry*, *16*(1), 27–34.

Grant, J. E., & Potenza, M. N. (2006). Compulsive aspects of impulse control disorders. *Psychiatric Clinics of North America*, *29*(2), 539–551.

Gudjonsson, G. H. (1987). The significance of depression in the mechanism of "compulsive" shoplifting. *Medicine, Science, and the Law, 27*(3), 171–176.

Guidry, L. S. (1975). Use of a covert punishing contingency in compulsive stealing. *Journal of Behavior Therapy and Experimental Psychiatry*, *6*(2), 169.

Hyman, S. E. (1993). Molecular and cell biology of addiction. *Current Opinion in Neurology and Neurosurgery*, *6*(4), 609–613.

Keutzer, C. (1972). Kleptomania: A direct approach to treatment. *British Journal of Medical Psychology*, *45*(2), 159–163.

Kim, S. W. (1998). Opioid antagonists in the treatment of impulse-control disorders. *Journal of Clinical Psychiatry*, *59*(4), 159–164.

Kim, S. W., & Grant, J. E. (2001). An open naltrexone treatment study in pathological gambling disorder. *International Clinical Psychopharmacology*, *16*(5), 285–289.

Kindler, S., Dannon, P. N., Iancu, I., Sasson, Y., & Zohar, J. (1997). Emergence of kleptomania during treatment for depression with serotonin selective reuptake inhibitors. *Clinical Neuropharmacology*, *20*(2), 126–129.

Kmetz, G. F., McElroy, S. L., & Collins, D. J. (1997). Response of kleptomania and mixed mania to valproate. *American Journal of Psychiatry*, *154*(4), 580–581.

Koob, G. F. (1992). Drugs of abuse: Anatomy, pharmacology and function of reward pathways. *Trends in Pharmacological Sciences*, *13*, 177–184.

Koob, G. F., & Bloom, F. E. (1988). Cellular and molecular mechanisms of drug dependence. *Science*, *242*(4879), 715–723.

Koran, L. M., Aboujaoude, E. N., & Gamel, N. N. (2007). Escitalopram treatment of kleptomania: An open-label trial followed by double-blind discontinuation. *Journal of Clinical Psychiatry*, *68*(3), 422–427.

Kuhar, M. J., Ritz, M. C., & Boja, J. W. (1991). The dopamine hypothesis of the reinforcing properties of cocaine. *Trends in Neurosciences*, *14*(7), 299–302.

Kraus, J. E. (1999). Treatment of kleptomania with paroxetine. *Journal of Clinical Psychiatry*, *60*(11), 793.

Lepkifker, E., Dannon, P. N., Ziv, R., Iancu, I., Horesh, N., & Kotler, M. (1999). The treatment of kleptomania with serotonin reuptake inhibitors. *Clinical Neuropharmacology*, *22*(1), 40–43.

Levy, E. (1934). Psychoanalytic treatment of a child with a stealing compulsion. *American Journal of Orthopsychiatry*, *4*, 1–12.

Linnoila, M., Virkunnen, M., Scheinen, M., Nuutila, A., Rimon, R., & Goodwin, F. K. (1983). Low cerebrospinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from non-impulsive violent behavior. *Life Sciences*, *33*, 2609–2614.

Marazziti, D., Presta, S., Pfanner, C., Di Nasso, E., & Cassano, G. B. (2000). The biological basis of kleptomania and compulsive buying. *Scientific Abstracts, 39th annual meeting of the American College of Neuropsychopharmacology*, San Juan, Puerto Rico.

Matthews, R. T., & German, D. C. (1984). Electrophysiological evidence for excitation of rat ventral tegmental area and dopamine neurons by morphine. *Neuroscience*, *11*(3), 617–625.

McConaghy, N., & Blaszczynski, A. (1988). Imaginal desensitization: A cost-effective treatment in two shoplifters and a binge-eater resistant to previous therapy. *Australia and New Zealand Journal of Psychiatry*, *22*, 78–82.

McCown, W., & Chamberlain, L. (2000). *Best possible odds: Contemporary treatment strategies for gambling disorders*. New York: Wiley.

McElroy, S. L., Hudson, J. I., Pope, H. G., Jr., & Keck, P. E. (1991a). Kleptomania: Clinical characteristics and associated psychopathology. *Psychological Medicine*, *21*(1), 93–108.

McElroy, S. L., Keck, P. E., Pope, H. G., Jr., & Hudson, J. I. (1989). Pharmacological treatment of kleptomania and bulimia nervosa. *Journal of Clinical Psychopharmacology*, *9*(5), 358–360.

McElroy, S. L., Pope, H. G., Jr., Hudson, J. I., Keck, P. E., Jr., & White, K. L. (1991b). Kleptomania: A report of 20 cases. *American Journal of Psychiatry*, *148*(5), 652–657.

McNeilly, D. P., & Burke, W. J. (1998). Stealing lately: A case of late-onset kleptomania. *International Journal of Geriatric Psychiatry*, *13*(2), 116–121.

Mogenson, G. J., Jones, D. J., & Yim, C. Y. (1980). From motivation to action: Functional interface between the limbic system and motor system. *Progress in Neurobiology*, *14*(2–3), 69–97.

Moreno, I., Saiz-Ruiz, J., & López-Ibor, J. J. (1991). Serotonin and gambling dependence. *Human Psychopharmacology*, *6*, 9–12.

National Association for Shoplifting Prevention. (2009). *Shoplifting statistics*. Retrieved July 31, 2009, from http://www.shopliftingprevention.org/

Potenza, M. N., & Hollander, E. (2002). Pathological gambling and impulse control disorders. In J. T. Coyle, C. Nemeroff, D. Charney, & K. L. Davis. (Eds.), *Neuropsychopharmacology: The 5th generation of progress* (pp. 1725–1741). Baltimore: Lippincott Williams & Wilkins.

Presta, S., Marazziti, D., Dell'Osso, L., Pfanner, C., Pallanti, S., & Cassano, G. B. (2002). Kleptomania: Clinical features and comorbidity in an Italian sample. *Comprehensive Psychiatry*, *43*(1), 7–12.

Rado, S. (1933). Fear of castration in women. *Psychoanalytic Quarterly*, *2*, 424–475.

Sarasalo, E., Bergman, B., & Toth, J. (1987). Theft behavior and its consequences among kleptomaniacs and shoplifters: A comparative study. *Forensic Science International*, *86*(3), 193–205.

Schwartz, J. H. (1992). Psychoanalytic psychotherapy for a woman with diagnoses of kleptomania and bulimia. *Hospital and Community Psychiatry*, *43*(2), 109–110.

Sher, K. (1991). Psychological characteristics of children of alcoholics: Overview of research methods and findings. *Recent Developments in Alcoholism*, *9*, 301–326.

Stewart, J. (1984). Reinstatement of heroin and cocaine self-administration behavior in the rat by intracerebral application of morphine in the ventral tegmental area. *Pharmacology, Biochemistry, and Behavior, 20*(6), 917–923.

Virkunnen, M., Rawlings, R., Tokola, R., Poland, R. E., Guidotti, A., Nemeroff, C., Bissette, G., Kalogeras, K., Karonen, S. L., & Linnoila, M. (1994). CSF biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholic violent offenders, fire setters, and healthy volunteers. *Archives of General Psychiatry*, *51*(1), 20–27.

CHAPTER 25

Assessment and Treatment of Intermittent Explosive Disorder

Michael S. McCloskey, Mitchell E. Berman, *and* Kurtis Noblett

Abstract

Intermittent explosive disorder (IED), a DSM-IV-TR disorder characterized by significant acts of aggression and violence, is being increasingly recognized as a prevalent and chronic disorder. Given the personal, social, and economic costs associated with IED, there is a clear need for well-validated assessment measures and efficacious treatments. However, there are currently no published, well-validated diagnostic measures of IED. With regard to treatment, preliminary evidence from a few randomized clinical trials suggests that selective serotonin reuptake inhibitors and cognitive behavioral psychotherapy may be effective in treating IED. However, more research is needed before either can be considered an empirically supported treatment for IED. In this chapter, we discuss (1) challenges in developing reliable and valid assessments for IED and (2) issues relevant to developing and testing psychological and pharmacological treatment interventions for IED.

Keywords: intermittent explosive disorder, aggression, assessment, psychotherapy, pharmacotherapy, selective serotonin reuptake inhibitor (SSRI), cognitive behavioral therapy

Introduction

Intermittent explosive disorder (IED) is the DSM-IV-TR diagnostic category used to classify individuals who engage in repeated acts of impulsive aggression that are disproportionate to any provocation and are not better accounted for by the effects of a substance, medical condition, or other psychological disorder (APA, 2000). Importantly, IED is the only disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; APA, 2000) for which affective aggressive behavior is the defining feature. Though historically IED was thought to be quite rare (APA, 2000), recent clinical and epidemiological findings support the notion that it is an underdiagnosed disorder with lifetime prevalence rates ranging from 4% to 7% (Coccaro et al., 2005; Kessler et al., 2006; Ortega et al., 2008). Furthermore, IED is associated with significant impairments in interpersonal and occupational functioning that can include loss of work, troubled relationships, and legal problems (McElroy et al. 1998). This burden is compounded by the chronic nature of the disorder when untreated (Kessler et al., 2006; McElroy et al., 1998). In addition, IED is associated with significant medical problems including coronary heart disease, hypertension, and stroke (McCloskey et al., 2010). Despite its prevalence and severity, there is a relative dearth of information on the assessment and treatment of IED.

Assessment of IED

IED Diagnostic Issues

The DSM-IV criteria for IED (APA, 2000) require "A) several discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property, B) the degree of aggressiveness expressed during the episodes is grossly out of proportion to any precipitating psychosocial stressors, and C) the aggressive episodes are not better accounted for by another mental disorder (e.g., antisocial personality

disorder, borderline personality disorder, a psychotic disorder, a manic episode, conduct disorder, or attention-deficit/hyperactivity disorder) and are not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition (e.g., head trauma, dementia of the Alzheimer's type)" (p. 667). This criteria set presents several challenges in assessing and diagnosing IED. The frequency and severity of aggressive acts addressed in the criteria are not fully defined. Does *several* refer to two serious assaultive acts? Three? Five? Does it matter if these acts occurred over a 2-year period versus a 20-year period? It is not clear from the information provided in the DSM. There is also some ambiguity with regard to what constitutes a *serious* assaultive act or *destruction* of property. However, some guidance is provided in the DSM–IV-TR (APA, 2000), which states, "Examples of serious assaultive acts include striking or otherwise hurting another person or verbally threatening to physically assault another individual. Destruction of property entails purposeful breaking of an object of value" (p. 663).

Criterion B requires the aggression to be *grossly* out of proportion to any provocation or psychosocial stressors, with no further information on how to define this. In the absence of an objective definition, the assessor is left to his or her clinical judgment. With respect to provocation, the first author and several of his research colleagues have adopted the guideline of considering an aggressive act to be grossly disproportionate (1) if the person being assessed initiates the aggressive episode by being verbally or physically aggressive toward an individual who had not been verbally or physically aggressive or (2) if the person being assessed escalated an aggressive encounter by responding to an act of verbal or physical aggression with an act of greater aggression. Examples of such escalation would include responding to a minor verbal act of aggression such as a terse or rude comment with a threat of physical harm or responding to a more severe act of verbal aggression such as threats and/or insults with physical aggression. However, even this guideline is admittedly subjective.

The final exclusionary criterion (C) specifies that the aggressive acts are not due to another mental disorder, a substance, or a general medical condition. This can be complicated, as individuals with IED often have a history of several other comorbid disorders, including an increased rate of substance use disorders (Coccaro et al., 2005; Kessler et al., 2006; Ortega et al., 2008). For more acute disorders, a temporal comparison of the aggressive episodes and the comorbid condition is usually sufficient to determine if IED should be diagnosed. For example, if an individual being evaluated for IED also has a history of major depression with three depressive episodes, the assessor would determine if the level of aggression frequency and severity *outside* of the three depressive episodes was sufficient to meet criteria for IED. If not—that is, if the person's aggression occurred primarily when clinically depressed, the individual would not be given an additional diagnosis of IED. However, this becomes more complicated when the exclusionary disorders are chronic. For example, antisocial personality disorder and borderline personality disorder are explicitly listed in the DSM as two potential exclusionary diagnoses. However, both disorders (much like IED) become evident early in life and tend to follow a chronic course, making it extremely difficult to determine whether the aggression is better accounted for by the existing personality disorder or IED.

Concerns about limitations of the DSM diagnostic criteria for IED led to an alternative integrated research IED (IED-IR) criteria set (Coccaro, 2003). In brief, the IED-IR criteria set provided an objective definition of minimal aggression frequency for IED as either twice-weekly acts of verbal aggression for 4 consecutive weeks or three acts of physical aggression within a 1-year period. The IED-IR criteria set also explicitly required the aggressive acts to be predominantly affective (occurring in response to anger) and to result in distress or impairment, both of which are implied but not explicitly stated in the DSM IED criteria set. The IED-IR criteria set also eliminated borderline and antisocial personality disorders from the group of potential disorders that may better explain aggressive behavior. This was done in part because of the difficulty of making this determination and in part due to data suggesting that patients with borderline and antisocial personality disorder who also meet criteria for IED have higher aggression levels and more severe psychosocial deficits than patients with borderline and antisocial personality disorder without IED (Coccaro, 2003). In contrast, patients with antisocial and borderline personality disorder who did not meet criteria for IED were not more aggressive than patients with other (nonborderline or antisocial) personality disorders. In other words, the mere presence of antisocial or borderline personality disorder did not explain the presence of aggressiveness in the individual. While these changes made the diagnoses of IED easier, they also complicated the picture by

providing a separate, partially overlapping criteria set for IED.

IED Assessment

There are no published diagnostic measurement instruments for IED. Diagnostic issues surrounding IED, the use of multiple criteria sets to diagnose the disorder, and the relative paucity of IED research (compared to borderline personality disorder, antisocial personality disorder, and major mood disorders) are some of the potential reasons for the failure of researchers to develop and publish assessment instruments for IED. However, at least one clinical interview and one unpublished screening questionnaire have been developed to diagnose IED. Also, several well-validated measures of anger and aggression have been used to assess aggressive tendencies and history in studies of IED. These are described below.

intermittent explosive disorder module (ied-m)

The IED-M (Coccaro, unpublished instrument) is a 20- to 30-minute structured diagnostic interview designed to obtain systematic information sufficient to make research diagnoses of current and lifetime IED by both DSM-IV and IED-IR criteria. The IED-M obtains quantitative information about lifetime and current verbal aggression, aggression against property, and physical aggression. Contextual descriptions of the three most serious episodes of each type of aggression during the 1-year period in which the aggression occurred most frequently (e.g., "What was the provocation?" and "What were the consequences of this outburst?") provide information about the proportionality of the aggressive response. Additional phenomenological information about aggressive acts is also obtained, including, but not limited to, age of onset and offset of each type of aggression, the effects of the aggressive behaviors on relationships with family and friends, subjective level of distress, emotions and physical symptoms before and after an outburst, and frequency of substance use during aggressive outbursts.

The IED-M has been used in several published studies to diagnose IED (Coccaro et al., 2004; McCloskey et al., 2008a). These studies have provided indirect evidence of the measure's construct validity. Specifically, individuals diagnosed as having IED based on the IED-M are more aggressive on self-report and behavioral aggression measures than comparison groups with other psychiatric disorders (McCloskey et al., 2006; McCloskey et al., 2008a). Unpublished data from Coccaro also show that IED diagnoses using the IED-M have strong interrater reliability (k = .84) when used with adults as part of a full diagnostic battery that includes structured clinical interviews for other Axis I and Axis II disorders. The only published validation study of the IED-M showed the measure to have strong interrater reliability (k = .76–.87) but less impressive test-retest reliability (k = .43–.63) when administered to a group of 34 children and adolescents in treatment and community settings (Olvera et al., 2001). This study also provided evidence for the construct validity for the IED-M, with the IED group reporting higher levels of lifetime verbal and physical aggression than community or psychiatric controls (Olvera et al., 2001).

intermittent explosive disorder diagnostic questionnaire (ied-dq)

The IED-DQ (McCloskey, unpublished instrument) is a 7-item self-report measure that can be used to diagnose IED according to either DSM-IV-TR or IED-IR research criteria. The IED-DQ contains items that assess aggression frequency, severity, distress associated with the aggressive behavior, and exclusionary mental health or medical conditions. Unpublished results from an initial developmental study using the IED-DQ suggest that the measure has acceptable psychometric properties with good interrater reliability (k = .77–.80), test-retest stability (k = .70–.71), and evidence of construct validity provided by differences between IED and controls on self-report measures of anger and aggression. The IED-DQ also shows high concordance with the IED-M interview diagnoses, with sensitivity and specificity for the IED-DQ of .86 and .91, respectively, for the DSM-IV IED and of .85 and .95 for the IED-IR (McCloskey, unpublished manuscript).

Aggression Assessment

Given that the cardinal symptom of IED is frequent acts of affective aggression, well-validated measures of anger and aggression can be employed to assist in the assessment of IED, including assessing changes in aggression among IED patients in response to treatment.

life history of aggression (lha)

The LHA (Coccaro et al., 1997) is a brief 11-item semistructured interview that assesses the frequency of aggressive (5 items), self-aggressive (2 items), and antisocial (4 items) behavior. All items are rated

on a 6-point scale based on the total number of occurrences of the behavior since the age of 13. Scores are coded as follows: 0 (no occurrences), 1 (1 event), 2 (2 or 3 events), 3 (4 to 9 events), 4 (10 or more events), or 5 (more events than can be counted). The 5-item aggression scale from the LHA has been shown to be a valid and reliable measure of aggressive behavior that includes both verbal and physical aggression (Coccaro et al., 1996, 1997; Dougherty et al., 1999). Furthermore, individuals with IED typically score higher than other clinical groups on the LHA aggression scale (McCloskey et al., 2006). Preliminary (unpublished) data suggest that, when the LHA is used as a screening measure, a score of 13 on the aggression scale best discriminates IED from non-IED patients (McCloskey, 2009).

BUSS PERRY AGGRESSION QUESTIONNAIRE (BPAQ)

The BPAQ (Buss & Perry, 1992) is a 29-item questionnaire that assesses trait tendencies toward physical aggression, verbal aggression, anger, and hostility by having respondents rate items on a 1 ("extremely uncharacteristic of me") to 5 ("extremely characteristic of me") scale. The BPAQ is extensively used in anger and aggression research due in part to its strong psychometric properties (Archer et al., 1995; Buss & Perry, 1992; Harris, 1997). Previous research with the BPAQ suggests that individuals with IED score, on average, between 19 and 22 on both the physical and verbal aggression scales of the BPAQ (McCloskey et al., 2006, 2008a).

STATE-TRAIT ANGER EXPRESSION INVENTORY-II (STAXI-II)

The STAXI-II (Spielberger, 1999) is a 57-item multidimensional anger and aggression questionnaire that consists of six scales: state ("right now") anger, trait anger, anger expression-out (aggression), anger expression-in, anger control-out, and anger control-in. The Anger Expression Index, a measure of total anger expression, is derived by subtracting the two anger control scales from the two anger expression scales. All items are statements that participants rate using a 4-point scale, ranging from 1 (not at all/almost never) to 4 (very much so/almost always). Like the BPAQ, the STAXI-II has been extensively used in anger and aggression research. Furthermore, the trait anger, anger expression, and anger control scales have been shown to be sensitive to treatment effects among individuals with IED (McCloskey et al., 2008b).

OVERT AGGRESSION SCALE-MODIFIED FOR OUTPATIENT USE (OAS-M)

The OAS-M (Coccaro et al., 1991) is a semi-structured interview that assesses four clusters of aggressive behavior: verbal assault, assault against objects, assault against others, and assault against self. Within each behavior cluster, the OAS-M distinguishes five levels of aggression severity in each group that are weighted 1 (e.g., "snapped or yelled") through 5 (e.g., "threatened to hit a stranger"). The OAS-M obtains the frequency of aggressive episodes for each class of aggressive behavior. The frequency of each behavior within an aggression category is multiplied by its weight and then summed. This number is multiplied by the category's weight (1 for verbal assault, 2 for assault against objects, and 3 for both assault against others and assault against self) to derive a weighted category score. The four weighted category scores are summed to arrive at an aggression score. The OAS-M also includes scales for irritability and suicidality. The OAS-M takes about 10 minutes to administer. The aggression and irritability scales show strong interrater reliability (Coccaro et al., 1991) and are sensitive to changes in aggression associated with pharmacotherapy (Coccaro & Kavoussi, 1997) and psychosocial treatment (McCloskey et al., 2008b) among IED patients.

Treatment of IED

Though many interventions (both pharmacological and psychological) have been used to treat anger and aggression with varying degrees of success, little research has examined the efficacy of interventions for IED. Preliminary evidence, however, supports the efficacy of selective serotonin reuptake inhibitors (SSRIs) and cognitive behavioral therapy (CBT) in treating IED.

Pharmacological IED Treatments

Only a few clinical trials have examined the efficacy of psychotropic medications in the treatment of IED. However, several classes of psychopharmacological agents have been evaluated for their effects on affective aggression as a general symptom. The SSRIs arguably have the strongest evidence supporting their use in reducing aggression. Serotonergic dysfunction has been associated with emotion dysregulation and aggression (Canli & Lesch, 2007; Frankle et al., 2005; New et al., 2004). The SSRIs increase serotonin concentrations by acutely binding to the serotonin transporter and inhibiting presynaptic reuptake. Early open-label studies showed

that the SSRI fluoxetine (Prozac) reduced anger and aggression among patients with borderline personality disorders (Coccaro et al., 1990; Cornelius et al., 1990). Another SSRI, sertraline (Zoloft), was also found to reduce aggressive behavior in a small sample of aggressive patients with a personality disorder when given open label (Kavoussi et al., 1994).

More rigorous double-blind, randomized controlled trials (RCTs) generally supported the antiaggressive properties of SSRIs. Fluoxetine was shown to decrease anger among 22 subjects with borderline personality disorder relative to placebo (Salzman et al., 1995). A second RCT found fluoxetine to decrease both anger and aggressive behavior in a sample of 40 aggressive patients, all of whom had a personality disorder (Coccaro & Kavoussi, 1997). These findings were consistent with experimental studies showing that SSRIs reduce aggressive responding on laboratory measures when given repeatedly (Cherek et al., 2002) or even in a single dose (Berman et al., 2009). Other RCTs have been less supportive. For example, fluvoxamine (Luvox) was not shown to decrease angry-aggressive symptoms compared to placebo after 6 weeks among a group of subjects with borderline personality disorder (Rinne et al., 2002). The antiaggressive effects of SSRI treatment often emerge after approximately 8 weeks (Coccaro & Kavoussi, 1997).

Though not specifically a study of IED, the large majority of patients in the double-blind, placebo-controlled trial of fluoxetine in impulsively aggressive subjects (Coccaro & Kavoussi, 1997) met criteria for IED-IR (Coccaro, personal communication), providing preliminary evidence of SSRI efficacy in treating IED. More recently, a double-blind RCT of 100 IED-IR patients showed fluoxetine to be superior to placebo in reducing anger and aggression, with almost one-third of IED subjects in the fluoxetine condition displaying no aggression by the end of treatment (Coccaro et al., 2009).

Mood stabilizers have also been evaluated for their antiaggressive effects. An early RCT showed that lithium reduced violent infractions relative to placebo among prisoners with chronic aggressive behavior (Sheard et al., 1976). More recently, studies have looked at anticonvulsant mood stabilizers such as carbamazepine and divalproex. A small RCT showed that divalproex reduced "explosive temper" relative to placebo in children with a disruptive behavior disorder (Donovan et al., 2000). These results paralleled an open-label trial of divalproex on personality-disordered adults in which the medication was found to decrease irritability and aggressiveness over the e8-week trial period (Kavoussi & Coccaro, 1998), as well as a small crossover study comparing carbamazepine to placebo in 11 women with borderline personality disorder (Gardner & Cowdry, 1986). However, the antiaggressive effects of these mood stabilizers may not extend to all IED subjects. A large-scale RCT of divalproex in aggressive patients suggested that the medication was not more efficacious in reducing aggression than placebo (Hollander et al., 2003). A subanalysis showed that divalproex did reduce aggression among IED-IR subjects with a comorbid Cluster B personality disorder, but not among IED-IR subject without a comorbid Cluster B personality disorder (Hollander et al., 2003).

To summarize, preliminary evidence suggests that SSRIs may be efficacious in the treatment of IED, with effects emerging after about 2 months of treatment. This is consistent with the theory that affective aggression in general (Berman et al., 2009; Carrillo et al., 2009), and IED specifically, (Coccaro, 2000) are associated with central serotonergic dysfunction. Divalproex may also be efficacious in treating IED subjects with (but not without) a comorbid Cluster B personality disorder.

Behavioral IED Interventions

There is a dearth of research on the use of psychosocial interventions to treat IED. However, the efficacy of treatments that address the related construct of anger dyscontrol (which may or may not include problems with aggressive behavior) has been extensively evaluated. Numerous studies and at least four meta-analytic reviews (Beck & Fernandez, 1998; Bowman-Edmondson & Cohen-Conger, 1996; Del Vecchio & O'Leary, 2004; DiGuiseppe & Tafrate, 2003; Tafrate, 1995) suggest that cognitive-behavioral interventions such as relaxation training, skill training, cognitive therapy, and multicomponent treatments have moderate to large effects in the treatment of anger (anger, in contrast to aggression, is an emotional experience rather than a behavior); furthermore, the anger-reducing effects of these treatment remain at follow-up. Cognitive behavioral approaches also showed a large effect in reducing aggression in this population (DiGuiseppe & Tafrate, 2003). Additional factors that were specifically associated with aggression reduction among angry patients included the use of a therapy treatment manual as well as monitoring of adherence and

compliance with the treatment used. Finally, individual (compared to group) treatment also results in greater increases in positive behaviors as well as more consistent decreases in aggression. This last result was striking enough for the authors (DiGuiseppe & Tafrate, 2003) to suggest that practitioners working with aggressive clients should choose "structured interventions, delivered in an individual format" (p. 81). However, the anger treatment literature often fails to discriminate between clinical anger problems without aggression and pathological aggression. For example, in the meta-analyses discussed earlier, subjects included angry students, angry volunteers, angry medical patients, aggressive drivers, criminals, abusive parents, and abusive partners, with the majority of samples coming from the first three categories.

Evidence suggests that individuals with physical aggression may be more resistant to treatment than angry patients without a history of significant physical aggression. A meta-analysis of treatments specifically for interpersonal (domestic) violence found that both cognitive behavioral and feminist (Deluth model) interventions had only small effects (Cohen's d = .18–.35) in reducing aggression (Babcock et al., 2004), suggesting that such treatments may be of limited use in treating IED. However, though batters and individuals diagnosed with IED both engage in repeated acts of aggression, only a small proportion of individuals diagnosed with IED have a history of domestic violence, suggesting that the two populations are not identical. For example, many batterers use aggression largely as a means to gain power and control (Jasinski, 2005; Leone et al., 2004). In contrast, retaliation for a perceived slight or injustice is often the motivation for aggression in IED (McCloskey et al., 2008b). Accordingly, while previous work on anger and aggression interventions may inform the treatment of IED, neither population is equivalent to individuals with IED.

Two published studies tested behavioral treatments for individuals with IED. The first study examined the efficacy of a brief (four 90-minute sessions) cognitive behavioral program for 28 aggressive drivers (Galovski & Blanchard, 2002). The primary results supported the notion that cognitive behavioral treatment is more effective than self-monitoring in reducing anger and aggressive driving behavior. However, a subanalysis comparing IED and non-IED drivers showed a trend (p = .06) for IED drivers to improve less than drivers without IED, suggesting that individuals with IED may require a longer, more intensive therapy regimen than subclinical aggressive populations (Galovski & Blanchard, 2002).

Most recently, a small RCT compared a 12-week multicomponent cognitive behavioral intervention delivered in either an individual or group format to a wait list control group for subjects meeting IED-IR criteria (McCloskey et al., 2008b). The intervention, modeled after the Cognitive, Relaxation and Coping Skills Training (CRCST) anger treatment manual (Deffenbacher & Mckay, 2000), consisted of three primary components. The first three sessions focused on increasing awareness of physiological cues and teaching forms of relaxations (i.e., progressive muscle relaxation, relaxation imagery, diaphragmatic breathing, relaxation without tension, and cued-controlled relaxation). During the third session, subjects were given a rationale for the use of a time- out to prevent impulsive-aggressive behaviors. During the fourth and fifth sessions, the rationale for cognitive restructuring was presented via the A-B-C model of cognition. Six types of cognitive distortions were presented (i.e., misattribution, overgeneralization, labeling, blaming, demanding/commanding and magnifying/catastrophizing), with examples and strategies for responding to each distortion. The second half of the treatment focused on implementing and generalizing previously learned relaxation and cognitive skills via imaginal exposure. The final session also included relapse prevention strategies.

A total of 45 subjects were assigned to three conditions. Results showed that subjects with IED who were randomized into either of the two (individual or group) cognitive behavioral treatments showed greater reduction in anger and aggressive behavior compared to subjects in the wait list condition. Specifically, subjects with IED reduced their aggressive behavior from pretreatment to posttreatment by over 55% in the group CBT condition and by over 75% in the individual CBT condition. The treatment gains were maintained at 3-month follow-up. Subjects in the individual CBT condition also reported a greater decrease in hostile thoughts and a greater improvement in quality of life relative to wait list subjects. Finally, almost half (7 of 15) of the subjects in the individual CBT condition achieved remission status (no physical or aggression in the past 2 weeks) at the end of treatment. In comparison, only two of the subjects in the group CBT condition and one of the subjects in the wait list condition met these remission criteria. This study

provided initial support for the efficacy of a cognitive behavioral intervention in the treatment of IED.

Conclusions

Despite its inclusion as a DSM diagnosis for over 20 years, there has been a relative paucity of research on IED. This is due in part to limitations of the DSM diagnostic criteria and the subsequent use of separate research criteria. However, recent studies identifying the prevalence and severity of IED (Kessler et al., 2006), have highlighted the need for valid assessment measures and the development of efficacious interventions to both study and treat the disorder. Diagnostic assessments of IED have yet to be published. More established measures of anger and aggression have utility for assessing aggressive history and potentially symptom change, but they are insufficient to diagnose the disorder. Treatments for IED are in their infancy. The SSRIs (most notably fluoxetine) have some support for reducing aggression in IED but may require 2 months or more to reach clinical levels of effectiveness. The limited evidence for mood stabilizers is mixed, with divalproex reducing aggression only for IED subjects with a comorbid Cluster B personality disorder. Evidence for behavioral interventions is limited to one small RCT showing CBT to be more efficacious than a wait list in reducing anger, aggression, and associated IED deficits.

Future Directions

Research on IED is still in its early stages. In order to make progress in understanding and treating this disorder, there will first have to be consistency in defining the disorder. There already appears to be some movement toward harmonizing DSM and research criteria, with the latest text revision of the DSM including some forms of verbal aggression (e.g., threats) within the definition of "serious assaultive acts" required for the diagnosis. However, clear criteria for both the severity and the frequency of aggression required for a diagnosis of IED are needed to make comparisons across IED studies meaningful. Ideally, these criteria will be based on research findings relating to the frequency and severity of aggression typically required to cause distress and impairment in psychosocial functioning.

A related area of importance will be to disentangle IED from other comorbid disorders. Intermittent explosive disorder is comorbid with several psychological disorders that are associated with an increased risk of aggression (e.g., substance use disorders, mood disorders, borderline personality disorder). This distinction is most difficult for chronic disorders such as borderline personality disorder. Research looking into the neurobiological underpinnings of IED may help discriminate these disorders. Whereas borderline personality disorder is associated with generalized limbic hyperactivation to negative stimuli, early research suggests that patients with IED have limbic activity that is specific to anger stimuli (Coccaro et al., 2007).

Because the limited amount of research on IED has largely focused on supporting its existence, little research has been conducted on potential subtypes of IED. For example, approximately 70% of individuals with IED exhibit clinically significant physical and verbal aggression, while about 20% show only physical aggression and 10% show only verbal aggression (Coccaro, 2003). It is not clear if these groups represent different subtypes of IED with unique risk factors and trajectories.

Despite preliminary evidence for the efficacy of SSRIs and CBT, there is no intervention for IED that meets the threshold for an empirically supported treatment (Chambless & Hollon, 1998). Additional RCTs of these most promising interventions are needed. Once these treatments are established as efficacious (if indeed they are), future efforts should be targeted to identifying the mechanisms of change in order to produce more efficient and efficacious treatments. For example, if SSRIs and CBT work via different mechanisms, combining the two treatments may have an additive effect. There is anecdotal evidence to suggest that this may be the case (Coccaro & McCloskey, 2006), but no clinical trials have evaluated the combined effects of SSRIs and CBT on IED.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.; DSM-IV-TR). Washington, DC: American Psychiatric Association.

Archer, J., Kilpatrick, G., & Bramwell, R. (1995). Comparison of two aggression inventories. *Aggressive Behavior, 21*, 371–380.

Babcock, J. C., Green, C. E., & Robie, C. (2004). Does batterers' treatment work? A meta-analytic review of domestic violence treatment. *Clinical Psychology Review, 23*(8), 1023–1053.

Beck, R., & Fernandez, E. (1998). Cogntive-behavioral therapy in the treatment of anger. *Cognitve Therapy and Research, 22*, 62–75.

Berman, M. E., McCloskey, M. S., Fanning, J. R., Schumacher, J. A., & Coccaro, E. F. (2009). Serotonin augmentation reduces response to attack in aggressive individuals. *Psychological Science, 20*(6), 714–720.

Bowman-Edmondson, C., & Cohen-Conger, J. (1996). A review of treatment efficacy for individuals with anger problems: Conceptual, assessment and methodological issues. *Clinical Psychology Review, 16*, 251–275.

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology, 63*(3), 452–459.

Canli, T., & Lesch, K. P. (2007). Long story short: The serotonin transporter in emotion regulation and social cognition. *Nature Neuroscience, 10*(9), 1103–1109.

Carrillo, M., Ricci, L. A., Coppersmith, G. A., & Melloni, R. H., Jr. (2009). The effect of increased serotonergic neurotransmission on aggression: A critical meta-analytical review of preclinical studies. *Psychopharmacology* (*Berlin*)*, 205*(3), 349–368.

Chambless, D. L., & Hollon, S. D. (1998). Defining empirically supported therapies. *Journal of Consulting and Clinical Psychology, 66*(1), 7–18.

Cherek, D. R., Lane, S. D., Pietras, C. J., & Steinberg, J. L. (2002). Effects of chronic paroxetine administration on measures of aggressive and impulsive responses of adult males with a history of conduct disorder. *Psychopharmacology* (*Berlin*)*, 159*(3), 266–274.

Coccaro, E. F. (2000). Intermittent explosive disorder. *Current Psychiatry Reports, 2*(1), 67–71.

Coccaro, E. F. (2003). Intermittent explosive disorder. In E. F. Coccaro (Ed.), *Aggression: Psychiatric assessment and treatment* (pp. 149–199). New York: Marcel Dekker.

Coccaro, E. F., Astill, J. L., Herbert, J. L., & Schut, A. G. (1990). Fluoxetine treatment of impulsive aggression in DSM-III-R personality disorder patients. *Journal of Clinical Psychopharmacology, 10*(5), 373–375.

Coccaro, E. F., Berman, M. E., & Kavoussi, R. J. (1997). Assessment of life history of aggression: Development and psychometric characteristics. *Psychiatry Research, 73*(3), 147–157.

Coccaro, E. F., Harvey, P. D., Kupsaw-Lawrence, E., Herbert, J. L., & Bernstein, D. P. (1991). Development of neuropharmacologically based behavioral assessments of impulsive aggressive behavior. *Journal of Neuropsychiatry and Clinical Neuroscience, 3*(2), S44–51.

Coccaro, E. F., & Kavoussi, R. J. (1997). Fluoxetine and impulsive aggressive behavior in personality-disordered subjects. *Archives of General Psychiatry, 54*(12), 1081–1088.

Coccaro, E. F., Kavoussi, R. J., Cooper, T. B., & Hauger, R. L. (1996). Hormonal responses to *d*- and *d,l*-fenfluramine in healthy human subjects. *Neuropsychopharmacology, 15*(6), 595–607.

Coccaro, E. F., Lee, R. J., & Kavoussi, R. J. (2009). A double-blind, randomized, placebo-controlled trial of fluoxetine in patients with intermittent explosive disorder. *Journal of Clinical Psychiatry, 70*(5), 653–662.

Coccaro, E. F., & McCloskey, M. S. (2006). Hothead Harry, gnome assassin: Combined treatment of intermittent explosive disorder. In R. L. F. Spitzer, M. B. Williams, & J. B. W. Gibbon M. (Eds.), *DSM-IV-TR® casebook: Experts tell how they treated their own patients* (Vol. 2, pp. 364–375). Arlington, VA: American Psychiatric Publishing.

Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry, 62*(2), 168–178.

Coccaro, E. F., Posternak, M. A., & Zimmerman, M. (2005). Prevalence and features of intermittent explosive disorder in a clinical setting. *Journal of Clinical Psychiatry, 66*(10), 1221–1227.

Coccaro, E. F., Schmidt, C. A., Samuels, J. F., & Nestadt, G. (2004). Lifetime and 1-month prevalence rates of intermittent explosive disorder in a community sample. *Journal of Clinical Psychiatry, 65*(6), 820–824.

Cornelius, J. R., Soloff, P. H., Perel, J. M., & Ulrich, R. F. (1990). Fluoxetine trial in borderline personality disorder. *Psychopharmacology Bulletin, 26*(1), 151–154.

Deffenbacher, J. L., & Mckay, M. (2000). *Overcoming situational and general anger*. Oakland, CA: New Harbinger Publications.

Del Vecchio, T., & O'Leary, K. D. (2004). Effectiveness of anger treatments for specific anger problems: A meta-analytic review. *Clinical Psychology Review, 24*(1), 15–34.

DiGuiseppe, R., & Tafrate, R. C. (2003). Anger treatment for adults: A meta-analytic review. *Clinical Psychology: Science and Practice, 10*(1), 70–84.

Donovan, S. J., Stewart, J. W., Nunes, E. V., Quitkin, F. M., Parides, M., Daniel, W., et al. (2000). Divalproex treatment for youth with explosive temper and mood lability: A double-blind, placebo-controlled crossover design. *American Journal of Psychiatry, 157*(5), 818–820.

Dougherty, D. M., Bjork, J. M., Marsh, D. M., & Moeller, F. G. (1999). Influence of trait hostility on tryptophan depletion-induced laboratory aggression. *Psychiatry Research, 88*(3), 227–232.

Frankle, W. G., Lombardo, I., New, A. S., Goodman, M., Talbot, P. S., Huang, Y., et al. (2005). Brain serotonin transporter distribution in subjects with impulsive aggressivity: A positron emission study with [11C]McN 5652. *American Journal of Psychiatry, 162*(5), 915–923.

Galovski, T., & Blanchard, E. B. (2002). The effectiveness of a brief psychological intervention on court-referred and self-referred aggressive drivers. *Behaviour Research and Therapy, 40*(12), 1385–1402.

Gardner, D. L., & Cowdry, R. W. (1986). Positive effects of carbamazepine on behavioral dyscontrol in borderline personality disorder. *American Journal of Psychiatry, 143*(4), 519–522.

Harris, J. A. (1997). A further evaluation of the aggression questionnaire: Issues of validity and reliability. *Behaviour Research and Therapy, 35*, 1047–1053.

Hollander, E., Tracy, K. A., Swann, A. C., Coccaro, E. F., McElroy, S. L., Wozniak, P., et al. (2003). Divalproex in the treatment of impulsive aggression: Efficacy in cluster B personality disorders. *Neuropsychopharmacology, 28*(6), 1186–1197.

Jasinski, J. L. (2005). Trauma and violence research: Taking stock in the 21st century. *Journal of Interpersonal Violence, 20*(4), 412–417.

Kavoussi, R. J., & Coccaro, E. F. (1998). Divalproex sodium for impulsive aggressive behavior in patients with personality disorder. *Journal of Clinical Psychiatry, 59*(12), 676–680.

Kavoussi, R. J., Liu, J., & Coccaro, E. F. (1994). An open trial of sertraline in personality disordered patients with impulsive aggression. *Journal of Clinical Psychiatry, 55*(4), 137–141.

Kessler, R. C., Coccaro, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. (2006). The prevalence and correlates of DSM-IV intermittent explosive disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry, 63*(6), 669–678.

Leone, J. M., Johnson, M. P., Cohan, C. L., & Lloyd, S. E. (2004). Consequences of male partner violence for low-income minority women. *Journal of Marriage and Family, 66*(2), 472–490.

McCloskey, M. S., Berman, M. E., Noblett, K. L., & Coccaro, E. F. (2006). Intermittent explosive disorder–integrated research diagnostic criteria: Convergent and discriminant validity. *Journal of Psychiatric Research, 40*(3), 231–242.

McCloskey, M. S., Kleabir, K., Berman, M. E., Chen, E. Y., & Coccaro, E. F. (2010). Unhealthy aggression: The association between intermittent explosive disorder and negative health outcomes. *Health Psychology*, *29*(3), 324–332.

McCloskey, M. S., Lee, R., Berman, M. E., Noblett, K. L., & Coccaro, E. F. (2008a). The relationship between impulsive verbal aggression and intermittent explosive disorder. *Aggressive Behavior, 34*(1), 51–60.

McCloskey, M. S., Noblett, K. L., Deffenbacher, J. L., Gollan, J. K., & Coccaro, E. F. (2008b). Cognitive-behavioral therapy for intermittent explosive disorder: A pilot randomized clinical trial. *Journal of Consulting and Clinical Psychology, 76*(5), 876–886.

McElroy, S. L., Soutullo, C. A., Beckman, D. A., Taylor, P., Jr., & Keck, P. E., Jr. (1998). DSM-IV intermittent explosive disorder: A report of 27 cases. *Journal of Clinical Psychiatry, 59*(4), 203–210; quiz 211.

New, A. S., Trestman, R. F., Mitropoulou, V., Goodman, M., Koenigsberg, H. H., Silverman, J., et al. (2004). Low prolactin response to fenfluramine in impulsive aggression. *Journal of Psychiatric Research, 38*(3), 223–230.

Olvera, R. L., Pliszka, S. R., Konyecsni, W. M., Hernandez, Y., Farnum, S., & Tripp, R. F. (2001). Validation of the Interview Module for Intermittent Explosive Disorder (M-IED) in children and adolescents: A pilot study. *Psychiatry Research, 101*(3), 259–267.

Ortega, A. N., Canino, G., & Alegria, M. (2008). Lifetime and 12-month intermittent explosive disorder in Latinos. *American Journal of Orthopsychiatry, 78*(1), 133–139.

Rinne, T., van den Brink, W., Wouters, L., & van Dyck, R. (2002). SSRI treatment of borderline personality disorder: A randomized, placebo-controlled clinical trial for female patients with borderline personality disorder. *American Journal of Psychiatry, 159*(12), 2048–2054.

Salzman, C., Wolfson, A. N., Schatzberg, A., Looper, J., Henke, R., Albanese, M., et al. (1995). Effect of fluoxetine on anger in symptomatic volunteers with borderline personality disorder. *Journal of Clinical Psychopharmacology, 15*(1), 23–29.

Sheard, M. H., Marini, J. L., Bridges, C. I., & Wagner, E. (1976). The effect of lithium on impulsive aggressive behavior in man. *American Journal of Psychiatry, 133*(12), 1409–1413.

Spielberger, C. D. (1999). *STAXI-2 state trait anger expression inventory-2, professional manual*. Lutz, FL: Psychological Assessment Resources.

Tafrate, R. C. (1995). Evaluation of strategies for adult anger disorders. In H. Kassinove (Ed.), *Anger disorders: Definition, diagnosis and treatment* (pp. 109–130). Washington, DC: Taylor & Francis.

CHAPTER

26 Assessment and Treatment of Pyromania

Jon E. Grant *and* Brian L. Odlaug

Abstract

Pyromania, also referred to as *pathological fire setting*, is a disorder defined by the deliberate and purposeful fire setting that has occurred on more than one occasion and that cannot be attributed to another psychiatric disorder. Although juvenile or adolescent fire-setting behavior or match play is a fairly common occurrence, clinical pyromania is a rare disorder associated with shame and embarrassment, significant psychosocial dysfunction, and legal consequences. Case reports and small controlled clinical trials have reported the successful treatment of fire-setting behavior through both pharmacotherapeutic and psychotherapeutic means. Pyromania, however, is a largely misunderstood, unrecognized, and untreated disorder.

Keywords: adolescent, arson, clinical characteristics, fire setting, impulse control disorder juvenile, legal, pyromania, treatment

Introduction

Pyromania has been described in the medical literature for at least two centuries. One of the first descriptions was provided by Jean-Etienne Esquirol, who referred to the behavior as "incendiary monomania" (Esquirol, 1838). Esquirol included pyromania with kleptomania and erotic monomania as examples of irresistible behaviors. Esquirol differentiated pyromania from simple fire setting by claiming that pyromania was due to an instinctive impulse independent of will. Since that time, although people have debated the validity of the disorder (Lindberg et al., 2005; Pilgrim, 1885), surprisingly little has been written about pyromania.

The first *Diagnostic and Statistical Manual of Mental Disorders* (DSM-I) included pyromania as a supplementary term rather than as a formal diagnostic entity (APA, 1952), and pyromania was omitted entirely from DSM-II (APA, 1968). DSM-III categorized pyromania as an impulse control disorder not elsewhere classified (APA, 1980), the same clinical diagnostic category it currently holds in the DSM-IV-TR (APA, 2000). As an impulse control disorder, pyromania is currently classified with pathological gambling, kleptomania, intermittent explosive disorder, and trichotillomania (APA, 2000). Although included in DSM-IV-TR, pyromania is still a poorly understood disorder and has received very little empirical study.

Diagnosis

Pyromania, also referred to as *pathological fire setting*, is defined by the following criteria according to DSM-IV-TR: (1) deliberate and purposeful fire-setting that has occurred on more than one occasion; (2) feelings of tension or arousal preceding a fire-setting act; (3) pleasure, gratification, or relief when setting fires or when watching/participating in the aftermath of the fire; (5) the act of fire-setting is not done out of vengeance or for monetary gain; and (6) fire-setting cannot be directly attributed to another mental condition such as conduct or bipolar disorder or impairment due to substance use (APA, 2000, p. 671).

Only one published instrument is used to screen for pyromania: the Minnesota Impulse Disorders Interview (MIDI; Grant, 2008). The MIDI includes five questions that screen for pyromania. These questions have demonstrated excellent classification accuracy based on structured clinical interviews for pyromania in adults (sensitivity 100%, specificity 100%; Grant et al., 2005) and in adolescents (sensitivity 85.7%, specificity 98.9%; Grant et al., 2007). Using this instrument, researchers found lifetime pyromania rates of 5.9% in a sample of 204 adult psychiatric inpatients (Grant et al., 2005) and 6.9% in a sample of 102 adolescent psychiatric inpatients (Grant et al., 2007). Furthermore, a modified self-report version of the MIDI found a lifetime pyromania prevalence rate of 1.01% in a sample of 791 college students (Odlaug & Grant, 2010).

Failure to Diagnose

Although pyromania may be fairly common among certain populations (e.g., psychiatric patients), it usually goes undiagnosed. Many reasons exist for the failure to diagnose this disorder.

Shame and Secrecy

Shame and secrecy are aspects of pyromania, largely due to the illegal nature of the behavior. Many people are also embarrassed because of the lack of control inherent in pyromania. Shame and secrecy may explain to some extent why so few patients will volunteer information regarding this behavior unless specifically asked (Grant et al., 2005). Patients suffering from pyromania may also be worried that the clinician is required to report their behavior. Clinicians therefore may want to inform patients at the beginning of the evaluation about what they do and do not have to report.

Clinician Lack of Knowledge

Few health care professionals have education or training in pyromania. Clinicians may instead diagnose fire setting as antisocial personality disorder or as a symptom of mania. In addition, clinicians may have many biases regarding fire setting. For example, clinicians may see themselves as potential victims of the behavior. Therefore, they may see fire setting as purely criminal, without further assessment of the underlying motivations.

Misdiagnosis

Fire setting may be a symptom of other disorders. Some of the more common misdiagnoses are discussed below.

Bipolar Disorder

Bipolar manic episodes are characterized by impulsive behaviors (APA, 2000). These manic behaviors may include fire setting (Gunderson, 1974). In addition, individuals with pyromania have high rates of co-occurring bipolar disorder (14.3%; Grant & Kim, 2007). During a manic episode, however, people often exhibit multiple symptoms of mood dysregulation—excess energy, distractibility, mood elevation, or irritability (APA, 2000)—whereas the behavior of pyromania usually has none of these associated symptoms. The DSM-IV excludes a diagnosis of pyromania when the fire setting is due to another mental condition such as bipolar disorder (APA, 2000). When individuals with pyromania have co-occurring bipolar disorder, it is important for the clinician to determine if the fire setting exists only during mania or possible simply worsens during a manic episode.

Substance Use Disorders

Substance use disorders frequently co-occur in individuals with pyromania (33.3%; Grant & Kim, 2007). It is important for clinicians to determine (1) whether the substance use problem results in the fire setting, perhaps through disinhibition (e.g., fire setting only when intoxicated), or (2) whether they are related but independent problems. One study of 90 arson recidivists found that only 3 (3.3%) met criteria for pyromania but that an additional 9 subjects met criteria for pyromania when intoxicated at the time of the fire setting (Lindberg et al., 2005). Additionally, a survey of 34 adult arsonists found that 100% were under the influence of alcohol prior to or during the act of setting fires (Jayaraman & Frazer, 2006).

Personality Disorders

One study found that the rate of borderline personality disorder is elevated in individuals with pyromania (9.5%; Grant & Kim, 2007) compared to the community at large (5.9%; Grant et al., 2008). The DSM-IV-TR diagnosis of pyromania excludes those who set fires out of anger (which will exclude fire setting only as a symptom of borderline personality disorder) or those who set fires for monetary gains or criminal purposes (which will exclude individuals with antisocial personality disorder who set fires only for criminal reasons; APA, 2000). The clinician must therefore determine if the fire setting merits an independent diagnosis of pyromania or is only secondary to a personality disorder.

Psychotic Disorders

In one study of 54 fire setters, psychotic disorders were seen in 21 (38.9%) of the cases (O'Sullivan & Kelleher, 1987). In a similar study, 20%–30% of arsonists reported symptoms consistent with psychosis (Taylor & Gunn, 1984). Fire setting that is performed only during a psychotic episode would not meet DSM-IV-TR criteria for pyromania (APA, 2000). Clinicians therefore need to screen for possible thought disorders at the time of the fire setting.

Distinguishing Pyromania from Criminal Fire Setting

Most individuals who set fires are not pyromaniacs. In fact, the most common motives for fire setting appear to be anger and revenge (Rix, 1994). People without pyromania who set fires have been found to have poor interpersonal skills, and their fire-setting behavior is thought to reflect their inability to cope with feelings of anger and frustration (Rix, 1994). For these individuals, there may be no psychiatric disorder driving the behavior. One study of 191 psychiatric inpatients found that approximately 26% had indulged in some kind of fire-setting behavior, although no relationship could be found between this behavior and a psychiatric illness (Geller & Bertsch, 1985). A review of 79 psychiatric outpatient children revealed that 18.9% were fire setters (Fitzgerald & O'Hanlon, 1991). Consequently, it is important that the clinical interview examine the motivations behind the fire setting and not simply assess the behavior. Individuals with pyromania have urges to set fires and experience a "rush" from this behavior (Grant & Kim, 2007).

There is often confusion about the terms used to describe the various types of fire setting. *Pyromania* is the proper term for fire-setting behavior only when the DSM-IV criteria have been met. In contrast to pyromania, arson is not a diagnosed psychiatric disorder or medical illness and has a distinct definition that differs from that of pyromania. Although state statutes may differ on the explicit language, *arson* is generally defined as a crime of maliciously, voluntarily, and willfully setting fire to a building or other property of another person or burning one's own property for an improper purpose (i.e., insurance fraud). Generally, an arsonist is motivated by some type of gain, whether it is revenge for a wrongdoing, for insurance fraud, or for sociopolitical reasons. Pyromania, on the other hand, is an impulse or urge-driven behavior that affects, among other things, the social and occupational lives of the individuals suffering from the illness. These urges have been described as addictive, and the act of setting a fire produces a sense of calm for the individual (Grant & Kim, 2007).

Unlike pyromania, arson appears to be a common occurrence that affects many people financially and emotionally. In 2004 according to statistics from the Federal Bureau of Investigation, there were 68,245 reported arson offenses in the United States, with an average dollar loss per incident of $12,017 (Federal Bureau of Investigation, 2004). Recent data provided through the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) found that 1.13% of a large community sample acknowledged having started a fire in their lifetime for the purpose of destroying someone else's property or just to see it burn (Vaughn et al., 2010). Very few arsonists, however, suffer from pyromania. One study found that only 3 (3.3%) of 90 arson recidivists had pure pyromania (Lindberg et al., 2005). In addition, not everyone who suffers from pyromania is an arsonist. A person meeting DSM-IV criteria for pyromania may also meet the legal definition of arson, but the DSM-IV criteria for pyromania do not require that a person ever set fire to another person's property.

Assessing Juvenile Fire Setting

Fascination with fire has been documented for centuries. Usually beginning at around 2 or 3 years of age, fire's attraction remains constant throughout an individual's life. In fact, behaviors such as playing with matches have been noted in 24.4% of child psychiatric outpatients, and a fire-setting rate of 19.4% has been found in this sample (Kolko & Kazdin, 1988). Another study of 99 elementary school boys found that 45% played with matches (Kafry, 1980). Although it is presently unknown how common this behavior is among children in the general population, its consequences are clear. Burn injuries account for 40% of accidental deaths in children under the age of 5, making these injuries the second leading cause of death in this age group (National Fire Data Center, 2001).

Juvenile fire setters are responsible for approximately 60% of all fires set in large cities (Mieszala, 1981). Local fire departments detect more children who set fires than do mental health professionals (Kolko, 1988), and on most occasions they are responsible for referring children suspected of concerning/serious fire-setting behavior to the various services for appropriate intervention. Therefore, various state fire departments have developed screening measures to help identify fire-setting

behavior in children (Fineman, 1980). Screening tools help fire department officials assess the severity of fire-setting behavior and then implement appropriate intervention and follow-up. Appropriate and adequate follow-up is important because the index fire-setting behavior is often preceded by rejection by the individual's care providers or social services departments (Koson & Dvoskin, 1982), and may contribute to high rates of suicide and self-injurious behavior among arsonists (O'Sullivan & Kelleher, 1987).

Based on an initial screening interview, a fire marshal may classify a child with fire-setting behavior into a category (Federal Emergency Management Agency, 1983) of either little (the fire department intervenes through an educational approach and provides information on safety), definite, or extreme concern. Children falling into the definite or extreme-risk category are referred to mental health services for further assessment and treatment of the behavior. Children with relatively less concerning fire-setting behavior (e.g., "curiosity" fire setters with little or no parental supervision) do not necessarily go through a mental health referral and benefit most from fire safety education provided by trained fire department officials (Fineman, 1995). This approach, along with educating the parents, has been shown to improve outcomes in children (with less concerning behavior) in terms of decreasing future fire-setting behavior (Fineman, 1995). Parents of children who set fires may find it difficult to accept such behavior as needing the attention of mental health professionals. This may be a huge barrier to the treatment of a child with serious/extreme fire-setting behavior. In such circumstances, it is vital to understand the social situation of the family (which itself may have contributed to the fire-setting behavior) and take aggressive steps to get the family involved in treatment and appropriate follow-up, even if that means forceful intervention on the part of the fire department by threatening a report to the Child Protection Services.

Mental health evaluations consist of a psychosocial assessment and a psychiatric diagnosis. A screening procedure developed by Sakheim and colleagues (1985, 1986) has helped identify certain variables that, when present in the assessment/interview of a child, correlate positively with the fire-setting behavior. Some of these variables are strong feelings of anger/resentment over neglect/abandonment and/or abuse by parents, strong feelings of revenge for the wrong done by the parents, poor impulse control and poor judgment, sexual maladjustment (which may be the result of abuse itself), absence of the father, upbringing in a conflicted family (Fineman, 1980), and a history of previous fire setting (by matches or lighters). Other positive correlates of fire-setting behavior are a psychiatric diagnosis of conduct disorder and dysthymic disorder (Fineman, 1980) and a positive response to the test item "I get quite excited when I see a fire burning."

Factors that were found to decrease the risk of (future) fire setting were feelings of guilt and depression after setting the fire, the ability to verbally express frustration/anger, the ability to empathize with other people, and good relations with peers/desire to feel close and intimate (Sakheim & Osborn, 1986; Sakheim et al., 1985).

Both typical fire setting and pyromania may start at a relatively early age. While child and adolescent fire setting is fairly common, true pyromania is quite rare in this age group. Researchers have identified revenge on parents or other persons in authority as the primary motivator for juvenile fire setters (Räsänen et al., 1995). There is, however, little information on how pyromania presents in adolescents and whether it presents differently than in adults. Based on reports of adolescent pyromania (Grant, 2006), there is some evidence that the current DSM-IV criteria apply to adolescents who report being unable to control their fire setting. More research, however, is needed to understand the presentation of fire setting among adolescents.

Treatment

Etiology

Understanding the possible etiology or etiologies for pyromania may better target treatment options. The underlying biological mechanism of urge-based disorders may involve the processing of incoming reward inputs by the ventral tegmental area-nucleus accumbens-orbitofrontal cortex circuit. This circuit influences behavior by modulating animal and human motivation (e.g., urges, cravings; Kalivas et al., 1999). Dopamine and glutamate may play major roles in the regulation of this region's functioning. One hypothesis is that differences in this region may result in the urges seen in pyromania and other impulse control disorders. The efficacy of topiramate (see below) lends some support to this hypothesis. Topiramate is thought to modulate dopaminergic and glutamatetgic neurons in this area.

Urges linked to the experience of reward and pleasure represent an important potential clinical target in pyromania. Patients with pyromania report urges to set fires. Most report fairly frequent urges

that result in fire setting. Many indicate that the act of setting a fire reduces the urges or the tension these urges produce. The act of fire setting also often provides a "rush" for some patients, producing a pleasurable feeling (Grant & Kim, 2007).

Frontal serotonergic systems have been implicated in impaired impulse regulation, and serotonin dysregulation may also be a clue to the pathophysiology of pyromania (as well as other impulse control disorders). Pharmacological intervention for pyromania has produced some promising results from selective serotonin reuptake inhibitors (SSRIs).

Some support for frontal inhibitory dysfunction comes from a study of a single individual with pyromania. Neuroimaging using single photon emission computed tomography (SPECT) found an association between left inferior frontal perfusion deficits and pyromania in a young man with pyromania (Grant, 2006). Other support comes from neuropsychological assessment of one subject with pyromania that found impairments in attention (Continuous Performance Test), verbal/visual memory (Rey Auditory Verbal Learning and Complex Figure Tests), and executive functions (Verbal Fluency); by contrast, visuospatial skills (Parietal Lobe Test) were intact (Parks et al., 2005).

New-onset fire setting has also been described in an individual with an arachnoid cyst of the cerebellar vermis (Heidrich et al., 1996). Neuroimaging studies examining other impulse control disorders, such as trichotillomania (TTM), have reported reduced volumes of the total, right, and left cerebellum cortex in TTM (Keuthen et al., 2007). Keuthen et al. (2007) found that these patients had significantly reduced volumes of the emotional functional cluster compared to controls, potentially underscoring the affective symptoms often reported in TTM subjects as well as those with other impulsive behaviors such as pyromania.

From a behavioral standpoint, fire setting may raise self-esteem by creating a feeling of power from the destruction of property, and this feeling may be particularly reinforcing when a person sets a fire without being apprehended. Some evidence supporting this theory may be found in the fact that individuals with pyromania report rates of abuse in childhood that are higher than those found in the population at large (Grant & Kim, 2007). Other behavioral models that have been used to explain other impulse control disorders may also apply to pyromania. Physiological arousal associated with fire setting (the "rush" reported by individuals with pyromania; Grant & Kim, 2007) may be a reinforcer that initiates and perpetuates the behavior.

Treatment Options

There are no randomized, controlled clinical trials examining either pharmacotherapy or psychotherapy for the treatment of pyromania. No medications have been approved by the Food and Drug Administration (FDA) for the treatment of this disorder. Therefore, it is important to inform patients of any "off-label" use of medications for this condition, as well as about the empirical basis for considering medication treatment.

Several different classes of medications have been described in case reports as demonstrating benefit in the treatment of pyromania. These include SSRIs such as escitalopram, sertraline, and fluoxetine; the mood stabilizer lithium; the antiepileptic medication topiramate; and a combination of the neuroleptic olanzapine and the antiepileptic sodium valproate (Grant & Kim, 2007; Parks et al., 2005). An equal number of medications, however, have also shown no benefit in the treatment of pyromania in case reports: fluoxetine, valproic acid, lithium, sertraline, olanzapine, escitalopram, citalopram, and clonazepam.

One case report, illustrating the treatment of an 18-year-old male with pyromania, described the use of a combination of topiramate with 3 weeks of daily cognitive behavioral therapy (CBT). The CBT consisted of fifteen 1-hour sessions that included imaginal exposure with response prevention and cognitive restructuring of fire-setting urges (Grant, 2006).

Other treatments have been developed for fire-setting behavior without limiting the intervention to pyromania. One case report detailed the use of relaxation therapy, overt sensitization, and awareness training in a 10-year-old male who set fires. One-year follow-up results indicated successful abstinence from all fire-setting behavior (Koles & Jenson, 1985). Other studies describing behavioral treatments of fire setting include methods such as fire safety education, coping and relaxation skills, aversive therapy, positive reinforcement, stimulus satiation, and operant structured fantasies and prevention programs designed for pyromania (Bennett et al., 2004; Bumpass et al., 1983; Clare et al., 1992; Franklin et al., 2002; Kolko, 1988; Rice & Quinsey, 1980; Taylor et al., 2002).

A treatment intervention for children developed by Bumpass and colleagues (1983) using a line-graphing technique with 29 children who set fires

helped the children become aware of the factors that triggered the behavior and identify the feelings that culminated in their fire setting (Bumpass et al., 1983). An average 2.5-year follow-up reported that 27 (93.1%) of the 29 children continued to set fires.

Bennett and colleagues (2004) report the successful use of fire education provided by firefighters to 42 children (29 court-referred) with fire-setting behaviors. After an assessment and subsequent fire-setting consequence education were provided, the researchers found a 100% success rate in quelling fire-setting behavior.

Group therapy methods have also been utilized with some success. Rice and Chaplin (1979) describe the use of assertion training in a group therapy setting for 10 fire-setting patients detained in a maximum security psychiatric institution (Rice & Chaplin, 1979; Rice & Quinsey, 1980). Along with social skills training already provided in the hospital, this training consisted of teaching patients how to express anger appropriately and how to say "no" to unreasonable requests made by others. Role playing and modeling the behavior of both therapists and other group members were also employed. At an 18-month follow-up, researchers noted no fire-setting behavior among the nine completers of the therapy.

Taylor and colleagues (2002) reported the use of 40-session CBT-based group therapy for 14 subjects with below average- or borderline intelligence (IQ=64-84) convicted of arson. Through a focus on reducing interest in fire and changing beliefs and attitudes about fire setting, clinically significant improvements on fire setting attitudes and interest were found in 10 (71.4%) subjects at the conclusion of treatment (Taylor et al., 2002).

Using preventive methods, Franklin and colleagues (2002) found that after 132 juvenile subjects (ages 4–17) who were arrested and convicted of fire setting were subjected to a full day of fire education, including the various financial, legal, and societal impacts of fire setting and an emphasis on individual accountability for their actions, the rate of recidivism was less than 1% for those in the active treatment group. A control group of 102 offenders fared less well, with a recidivism rate of 36% (Franklin et al., 2002).

Although there is no standard treatment method for pyromania at this time, based on the very limited literature, an approach using both CBT and pharmacological treatment may be most beneficial.

Conclusions

Pyromania is a largely unrecognized disorder that causes significant psychological, social, and legal repercussions. Because few individuals volunteer information regarding their fire setting, it is important that clinicians recognize the disorder and screen patients appropriately. Various treatments have been helpful in case studies, but more research on etiology and treatment is needed.

Future Directions

In an effort to better understand and subsequently treat this often disabling disorder, future research should strive to identify the neurobiological underpinnings of pyromania. Neuroimaging, neuropsychological testing, and randomized, placebo-controlled clinical trials are sorely needed and widely absent from the current literature for pyromania.

References

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (DSM-I). Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental disorders* (2nd ed.; DSM-II). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.; DSM-III-R). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision; DSM-IV-TR). Washington, DC: Author.

Bennett, B. K., Gamelli, R. L., Duchene, R. C., Atkocaitis, D., & Plunkett, J. A. (2004). Burn Education Awareness Recognition and Support (BEARS): A community-based juvenile firesetters assessment and treatment program. *Journal of Burn Care Rehabilitation, 25*(3), 324–327.

Bumpass, E. R., Fagelman, F. D., & Brix, R. J. (1983). Interventions with children who set fires. *American Journal of Psychotherapy*, *37*, 328–345.

Clare, I. C. H., Murphy, G. H., Cox, D., & Chaplin, E. H. (1992). Assessment and treatment of firesetting: A single-case investigation using a cognitive-behavioural model. *Criminal Behaviour and Mental Health*, *2*, 253–268.

Esquirol, E. (1838). *Des Maladies Mentales*. Paris: Bailliere.

Federal Bureau of Investigation. (2004). *Crime in the United States 2004*. Retrieved September 1, 2009 from *http://www.fbi.gov/ucr/ucr.htm*

Federal Emergency Management Agency. (1983). *Juvenile firesetting handbook: Dealing with children ages 7 to 14*. Washington, DC: U.S. Government Printing Office.

Fineman, K. R. (1980). Firesetting in childhood and adolescence. *Psychiatric Clinics of North America, 3*(3), 483–500.

Fineman, K. R. (1995). A model for the qualitative analysis of child and adult fire deviant behavior. *American Journal of Forensic Psychology, 13*(1), 31–60.

Fitzgerald, M., & O'Hanlon, M. (1991). The incidence of fire setting and associated psychopathology of children attending a child psychiatric outpatients. *Irish Journal of Medical Science, 160*(5), 128–129.

Franklin, G. A., Pucci, P. S., Arbabi, S., Brandt, M. M., Wahl, W. L., & Taheri, P. A. (2002). Decreased juvenile arson and firesetting recidivism after implementation of a multidisciplinary prevention program. *Journal of Trauma*, *53*(2), 260–264.

Geller, J. L., & Bertsch, G. (1985). Fire-setting behavior in the histories of a state hospital population. *American Journal of Psychiatry*, *142*(4), 464–468.

Grant, B. F., Chou, S. P., Goldstein, R. B., Huang, B., Stinson, F. S., Saha, T. D., Smith, S. M., Dawson, D. A., Pulay, A. J., Pickering, R. P., & Ruan, W. J. (2008). Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: Results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry*, *69*(4), 533–545.

Grant, J. E. (2006). SPECT imaging and treatment of pyromania. *Journal of Clinical Psychiatry*, *67*(6), 998.

Grant, J. E. (2008). *Impulse control disorders: A clinician's guide to understanding and treating behavioral addictions.* New York: W. W. Norton.

Grant, J. E., & Kim, S. W. (2007). Pyromania: Clinical characteristics and psychiatric comorbidity. *Journal of Clinical Psychiatry*, *68*(11), 1717–1722.

Grant, J, E., Levine, L., Kim, D., & Potenza, M. N. (2005). Impulse control disorders in adult psychiatric inpatients. *American Journal of Psychiatry*, *162*(11), 2184–2188.

Grant, J. E., Williams, K. A., & Potenza, M. N. (2007). Impulse-control disorders in adolescent psychiatric inpatients: Co-occurring disorders and sex differences. *Journal of Clinical Psychiatry*, *68*(10), 1584–1592.

Gunderson, J. G. (1974). Management of manic states: The problem of fire setting. *Psychiatry*, *37*(2), 137–146.

Heidrich, A., Schmidtke, A., Lesch, K. P., Hofmann, E., & Becker, T. (1996). Cerebellar arachnoid cyst in a firesetter: The weight of organic lesions in arson. *Journal of Psychiatry and Neuroscience, 21*(3), 202–206.

Jayaraman, A., & Frazer, J. (2006). Arson: A growing inferno. *Medicine, Science, and Law*, *46*(4), 295–300.

Kafry, D. (1980). Playing with matches: Children and fire. In D. Canter (Ed.), *Fires and human behavior* (pp. 47–61). Chichester, England: Wiley.

Kalivas, P. W., Churchill, L., & Romanides, A. (1999). Involvement of the pallidal-thalamocortical circuit in adaptive behavior. *Annals of the New York Academy of Sciences, 877*, 64–70.

Keuthen, N. J., Makris, N., Schlerf, J. E., Martis, B., Savage, C. R., McMullin, K., Seidman, L. J., Schmahmann, J. D., Kennedy, D. N., Hodge, S. M., & Rauch, S. L. (2007). Evidence for reduced cerebellar volumes in trichotillomania. *Biological Psychiatry*, *61*(3), 374–381.

Koles, M. R., & Jenson, W. R. (1985). Comprehensive treatment of chronic fire setting in a severely disordered boy. *Journal of Behavior Therapy and Experimental Psychiatry*, *16*(1), 81–85.

Kolko, D. J. (1988). Community interventions for juvenile firesetters: A survey of two national programs. *Hospital and Community Psychiatry*, *39*(9), 973–979.

Kolko, D. J., & Kazdin, A. E. (1988). Prevalence of fire-setting and related behaviors in child psychiatric inpatients. *Journal of Consulting Clinical Psychology*, *56*(4), 628–630.

Koson, D. F., & Dvoskin, J. (1982). Arson: A diagnostic study. *Bulletin of the American Academy of Psychiatry and the Law*, *10*(1), 39–49.

Lindberg, N., Holi, M. M., Tani, P., & Virkkunen, M. (2005). Looking for pyromania: Characteristics of a consecutive sample of Finnish male criminals with histories of recidivist fire-setting between 1973 and 1993. *BMC Psychiatry*, *5*, 47.

Mieszala, P. (1981). Juvenile firesetters. *Rekindle*, August, 11–13.

National Fire Data Center. (2001). Fire in the United States 1989–1998. Emmitsburg, Maryland: United States Fire Administration, Federal Emergency Management Agency.

Odlaug, B. L., & Grant, J. E. (2010). Impulse control disorders in a college sample. *Primary Care Companion Journal of Clinical Psychiatry*, *12*(2), e1–e5.

O'Sullivan, G. H., & Kelleher, M. J. (1987). A study of firesetters in the south-west of Ireland. *British Journal of Psychiatry*, *151*, 818–823.

Parks, R. W., Green, R. D. J., Girgis, S., Hunter, M. D., Woodruff, P. W., & Spence, S. A. (2005). Response of pyromania to biological treatment in a homeless person. *Neuropsychiatric Diseases and Treatment*, *1*(3), 277–280.

Pilgrim, C. W. (1885). Pyromania (so-called), with report of a case. *American Journal of Insanity*, *41*, 456–465.

Räsänen, P., Hirvenoja, R., Hakko, H., & Väisänen, E. (1995). A portrait of the juvenile arsonist. *Forensic Science International*, *73*(1), 41–47.

Rice, M. E., & Chaplin, T. C. (1979). Social skills training for hospitalized male arsonists. *Journal of Behavior Therapy and Experimental Psychiatry,* 10, 105–108.

Rice, M. E., & Quinsey, V. L. (1980). Assessment and training of social competence in dangerous psychiatric patients. *International Journal of Law and Psychiatry*, *3*(4), 371–390.

Rix, K. J. (1994). A psychiatric study of adult arsonists. *Medicine, Science, and Law*, *34*(1), 21–34.

Sakheim, G. A., & Osborn, E. (1986). A psychological profile of juvenile firesetters in residential treatment: A replication study. *Child Welfare*, *65*(5), 495–503.

Sakheim, G. A., Vigdor, M. G., Gordon, M., & Helprin, L. M. (1985). A psychological profile of juvenile firesetters in residential treatment. *Child Welfare*, *64*(5), 453–476.

Taylor, P. J., & Gunn, J. (1984). Violence and psychosis. I. Risk of violence among psychotic men. *British Medical Journal* (*Clinical Research and Education*), *288*(6435), 1945–1949.

Taylor, J. L., Thorne, I., Robertson, A., & Avery, G. (2002). Evaluation of a group intervention for convicted arsonists with mild and borderline intellectual disabilities. *Criminal Behavior and Mental Health*, *12*(4), 282–293.

Vaughn, M. G., Fu, Q., DeLisi, M., Wright, J. P., Beaver, K. M., Perron, B. E., & Howard, M. O. (2010). Prevalence and correlates of fire-setting in the United States: Results from the National Epidemiological Survey on Alcohol and Related Conditions. *Comprehensive Psychiatry*, *51*(3), 217–223.

CHAPTER 27

Assessment and Treatment of Pathological Skin Picking

Jedidiah Siev, Hannah E. Reese, Kiara Timpano, *and* Sabine Wilhelm

Abstract

Pathological skin picking (PSP) refers to chronic skin picking or scratching that causes tissue damage and distress. It is a heterogeneous category of behaviors and may be manifest in the context of various psychological disorders. This chapter presents an overview of the empirical literature on the assessment and treatment of PSP, including (1) a cognitive-behavioral model as heuristic for conceptualizing treatment, (2) assessment tools, (3) a review of the pharmacological and psychosocial treatment outcome literatures, (4) cognitive-behavioral treatment techniques, and (5) future directions. The chapter is intended to introduce the clinician to the assessment and psychological tools used to treat PSP, as well as to provide impetus to advance research in this understudied domain.

Keywords: skin picking; body-focused repetitive behavior; cognitive-behavior therapy; excoriation; habit reversal training

Assessment and Treatment of PSP

Introduction

Pathological skin picking (PSP) refers to chronic picking or scratching of skin lesions that causes tissue damage and distress, sometimes quite severe (Simeon et al., 1997). In the literature it is referred to in many ways, including *neurotic, psychogenic*, or *psychocutaneous excoriation*; *dermatillomania*; *neurodermatitis*; *neurotic or self-inflicted dermatosis*; and *acne excoriée*, all of which may or may not describe exactly the same phenomenon. Not currently classified as a unique psychiatric disorder (American Psychiatric Association, 2000), PSP is a heterogeneous condition that is similar to other body-focused repetitive behaviors (BFRBs) but is manifest in the context of numerous disorders and clinical presentations. Individuals engage in PSP for many reasons, and the behavior itself varies considerably from individual to individual (e.g., site and instrument of picking). The phenomenology and treatment of PSP are relatively understudied, and although there is budding evidence for the efficacy of psychological and psychiatric interventions, the empirical literature is limited. The clinician wishing to develop a treatment approach may be flummoxed when consulting a small literature based on a heterogeneous patient population.

The purpose of this chapter is to provide an overview of the assessment and treatment of PSP and, in so doing, (1) to introduce the clinician to the assessment and psychological (particularly cognitive-behavioral) tools used to treat PSP and (2) to provide an impetus to advance research on PSP. We begin with a simplified model, as directly relevant to assessment and treatment, highlighting the diverse and multiply determined behaviors under the umbrella term *pathological skin picking*. With attention to the consequent importance of careful and functional assessment, we then present assessment tools with demonstrated utility for PSP. Thereafter, we review the treatment outcome literature and then describe cognitive-behavioral techniques in treating PSP. Finally, we end with directions for future research.

Cognitive-Behavioral Model of PSP

Historically, the model for understanding PSP has centered on the notion of urge reduction. That is, picking was thought to occur in direct response to an internal tension, urge, or discomfort, which was then relieved by picking. Thus, picking was negatively reinforced each time it successfully rid the individual of the unpleasant sensation. The growing literature on BFRBs, however, suggests that this contingency is likely only one of many factors involved in the development and maintenance of PSP. The current conceptualization incorporates a range of biological, psychological, and situational features that can play a role in the presentation of this phenomenon. This newer model importantly accounts for the heterogeneous presentation of PSP, which includes the varied pathways through which the behavior could result and be maintained. Figure 27.1 presents a simplified version of the models presented by Franklin and Tolin (2007) and Mansueto et al., (1997) for trichotillomania and other BFRBs. We delineate the different components of this model below and also illustrate how a careful functional analysis is imperative when selecting the most helpful treatment avenue for any given individual. An important caveat to consider with regard to the current model is that it should be viewed as a work in progress, as future research will most certainly elucidate the specific factors involved, as well as the relationships that exist between these factors.

The model outlines three areas in which the factors that may contribute to the development and maintenance of PSP lie: general vulnerability factors, antecedents, and consequents. General vulnerability factors themselves do not necessarily lead to the onset of PSP, but rather function as a backdrop and occasionally help explicate reasons for initial bouts of the behavior. For example, biological factors, including a potential genetic predisposition (Bienvenu et al., 2000; Wilhelm et al., 1999), altered neurobiological circuits (Grachev, 1997; Swedo et al., 1991), and altered pain sensitivity (Christenson et al., 1991; Stanley et al., 1992) could all render an individual more vulnerable to the development of PSP. Family dynamics, such as learning or socialization, along with life experiences and comorbidity, may also increase an individual's

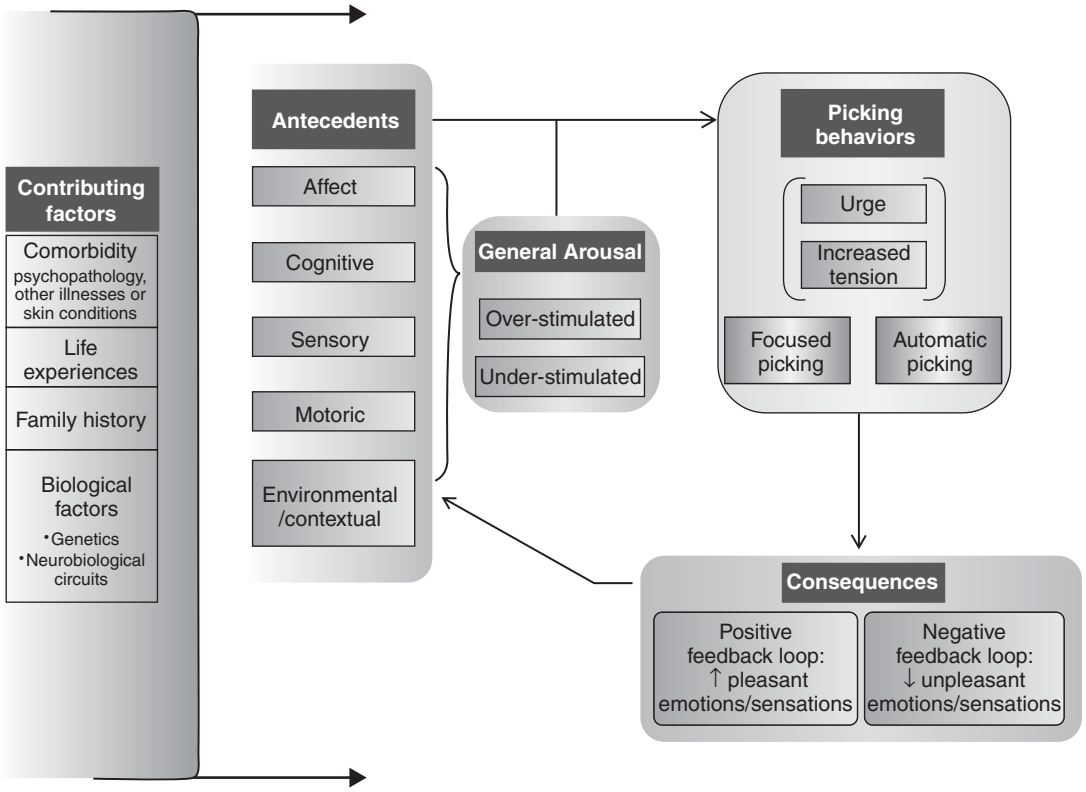

Fig. 27.1 Biopsychosocial model of PSP.

general level of risk for PSP. In particular, the presence of additional conditions or syndromes is important to consider since they may help explain initial occurrences of PSP (e.g., acne) or because the PSP may actually be a symptom of the other condition (e.g., picking as a symptom of body dysmorphic disorder [BDD] or Praeder-Willi syndrome).

The second building block of the model consists of a number of direct antecedents that can increase the chance of in-the-moment PSP. Several authors have discussed how antecedents can be classified as either internal or external in nature (Franklin & Tolin, 2007; Mansueto et al., 1997). Internal cues include affective (e.g., agitation, worry, boredom), cognitive (e.g., "I will feel satisfied if I just pick once" or "People will judge me if they see my scab"), and sensory (e.g., itchiness) phenomena. By contrast, external cues may include motoric triggers (e.g., touching or feeling for skin imperfections) and environmental or contextual cues (e.g., specific situations or visual signs). These factors are often established as triggers via classical conditioning (Azrin & Nunn, 1973; Franklin & Tolin, 2007), wherein the drive to perform the behavior is strengthened when the individual is exposed to something that has previously been linked with PSP. Antecedents may also trigger picking by producing a state of dysregulated arousal in which the individual becomes either hyper- or hypoaroused (Christenson et al., 1993; Franklin & Tolin, 2007). Research has found that feeling both over- and understimulated is linked to PSP (Bohne et al., 2002) and that the behavior may take on the role of a specific emotion regulation strategy (Begotka et al., 2003). Finally, it is possible, though not necessary, for the antecedents to be associated with a building tension in the individual, which results in a subsequent urge to pick.

Once picking begins, it can be classified as either automatic or focused picking. The former often occurs outside the person's awareness, whereas the latter is more likely to be associated with a specific goal (e.g., removing blemishes or regulating emotional distress). The consequences of picking are the final component of the model and represent the reinforcing agent. Within the negative feedback loop, picking serves to remove unpleasant emotions and/or sensations associated with the antecedents of the picking episode. Indeed, many individuals with PSP report that the behavior reduces negative emotions, such as sadness, anxiety, or boredom, along with removing or altering unpleasant sensations or cognitions (Bohne et al., 2002; Diefenbach et al., 2008; Wilhelm et al., 1999). At the same time, it is also possible for a positive feedback loop to be active. In this pathway, picking behaviors are reinforced by increasing pleasant emotions and/or sensations (Bohne et al., 2002; Meunier et al., 2009). These immediate consequences, which act as explicit behavioral contingencies that maintain the behavior, are distinct from the more long-term consequences of PSP. The delayed outcome of these behaviors often includes a series of detrimental repercussions, such as negative self-evaluation, mood states, social consequences, and possibly even physical scarring (Keuthen et al., 2000; Wilhelm et al., 1999).

Given the complexity of the model, it is clear that very few individuals with PSP will look exactly alike. As such, specific treatment strategies (discussed below in greater detail) may be helpful for one individual and completely ineffectual for another. It is therefore imperative for the clinician to conduct a thorough functional analysis with the patient to identify the most appropriate treatment strategies. For example, an individual with a skin disease (e.g., acne, eczema) may benefit from treatment of the physical condition in conjunction with treatment aimed at the PSP behaviors. In instances where features of BDD are driving the PSP, an individual may need supplementary cognitive-behavioral therapy (CBT) focused on BDD and instruction in more classical response prevention for the PSP. Considering the specific antecedents a given individual may experience, different techniques (e.g., stimulus control or alternative behaviors) could be implemented. With regard to consequences, if the therapist and the client determine that PSP functions primarily to modulate negative affect, techniques focused on emotion regulation and increasing distress tolerance may be most effective. As these examples indicate, each component of the model represents an instance where the cognitive-behavioral techniques for PSP can be uniquely tailored to a given individual's needs so that the most effective treatment is delivered.

Assessment Tools

In what follows, we provide an overview of the most commonly used assessment tools for PSP. These include self-monitoring and functional analysis, photographs, clinician-rated measures, and self-report measures. In light of the diversity of PSP, we again stress the necessity of careful assessment before intervention.

Self-Monitoring and Functional Analysis

Assessment begins with self-monitoring of the frequency and duration of the picking behavior. Particular attention should be paid to the relevant antecedents and consequences of the picking. Although a number of self-monitoring forms exist in the literature, Mansueto and colleagues have developed a particularly useful and detailed monitoring form for BFRBs and especially PSP (Figure 27.2; see also Mansueto et al., 1999). On this form, the patient records the date, times that picking began and ended, site of picking (e.g., the face, arm), intensity of the urge to pick, and efforts to resist picking. The form also allows the patient to note where he or she is and what he or she is doing, feeling, and thinking at the time the picking begins. After the picking has ended, the patient is prompted to write any resultant thoughts, feelings, or physical consequences. Mansueto and colleagues (1999) suggest that assessment should include evaluation of the following five types of antecedents and consequents: cognitive (e.g., having the thought "That hair is out of place"), affective (e.g., frustration, boredom), motoric (e.g., sitting in a certain position), sensory (e.g., an itch or tingling of the skin), and environmental (e.g., certain places or activities). Particular attention should also be paid to negative (e.g., relief from tension) and positive (e.g., pleasure, sensory stimulation) reinforcers of the behavior. As highlighted in the CBT model, these negative and positive feedback loops may play a crucial role in maintaining the picking behavior. Given the heterogeneous nature of PSP and the high levels of comorbidity, a careful functional analysis of the picking is essential to develop treatment interventions tailored to the patient's unique patterns of behavior.

Photographs

Photographs can provide an objective measure of the degree of picking and associated tissue damage. Twohig and colleagues (Twohig & Woods, 2001; Twohig et al., 2006) have used photographs as primary treatment outcome measures by having blind raters assess photographs of the affected areas for

Self-Monitoring Form for Body Focused Repetitive Behaviors

Day and Date:_____ **Time Began:**_____ **Time Ended:**_____

Location (where were you?)	
Activity (What activity were you engaged in? (E.g., watching TV, night time bathroom routine, etc.)	
Strength of Urges (0–10)	Degree of Awareness (0–10)
Notable Feelings Prior to Picking	
Notable Thoughts Prior to Picking	
Site(s) of Picking	
Strength of Effort to Resist (0–10)	
Interventions/efforts used to delay, distract, or use substitutes and success of each (0–10)	
Why did you stop when you did?	
Consequences: -Thoughts and Feelings: -Degree of visible damage (0–10)	
What did you do after the picking episode ended?	
Comments and observations	

Note: This form is based on the Comprehensive Behavior (ComB) Model, which was developed by Dr. Charles Mansueto, Ph.D., and his colleagues at the Behavior Therapy Center of Greater Washington.

Fig. 27.2 Self-monitoring form for BFRBs, including pathological skin picking. This form is reproduced with the generous permission of Dr. Charles Manseuto, Ph.D.

degree of damage. Photographic evidence can also provide a concrete measurement of treatment progress for the patient. Of course, clinicians should remain sensitive to any feelings of shame and embarrassment that being photographed may induce.

Clinician-Rated Measures

YALE-BROWN OBSESSIVE COMPULSIVE SCALE MODIFIED FOR NEUROTIC EXCORIATION

The Yale-Brown Obsessive Compulsive Scale Modified for Neurotic Excoriation (NE-YBOCS) is based on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), a widely used clinician-rated measure of obsessive-compulsive disorder (OCD) symptom severity (Goodman et al., 1989). Like the original Y-BOCS, the NE-YBOCS includes 10 items. Items 1–5 have been modified to correspond to urges to pick or thoughts about picking, and items 6–10 correspond to the picking behavior (Arnold et al., 1999; Bloch et al., 2001; Grant et al., 2007). Each item is rated on a 0 to 4 scale yielding total scores that range from 0 to 40. Although no psychometric papers have been published on the NE-YBOCS, it has demonstrated good test-retest reliability (ρ = .83) and good construct validity when compared with other outcome measures in one treatment outcome study (Grant et al., 2007).

SKIN PICKING TREATMENT SCALE

Simeon et al. (1997) also modified the Y-BOCS to create a clinician-rated measure of PSP. The Skin Picking Treatment Scale (SPTS) comprises five items that assess the frequency and duration of picking, the severity of picking, the intensity of the urge to pick, the degree of perceived control over picking, and interference due to picking. As with the original Y-BOCS, each item is rated on a 0 to 4 scale. Total scores range from 0 to 20. Presently, no psychometric data have been reported on this measure, although it has been used in two treatment outcome trials (Bloch et al., 2001; Simeon et al., 1997).

Self-Report Measures

HABIT QUESTIONNAIRE

The Habit Questionnaire is a self-report measure developed by Teng, Woods, Twohig, and Marcks (2002) to assess for the presence of a range of BFRBs including skin picking, mouth chewing, nail biting, skin biting, and skin scratching. For each endorsed behavior, the respondent is asked to rate the frequency and duration of the behavior as well as any associated impairment, injury, medical attention, or intervention. Respondents also report whether the behavior only occurs under the influence of alcohol or another substance and whether they have ever been diagnosed with any of the following comorbid conditions: OCD, Tourette's syndrome, autism, Asperger's syndrome, or developmental disability. The authors defined the presence of a BFRB as occurring more than five times a day for 4 weeks or longer and resulting in impairment in functioning, injury, medical attention, or intervention. In a sample of 105 undergraduate students, the Habit Questionnaire demonstrated moderate test-retest reliability ($\varnothing$ = .69).

MILWAUKEE INVENTORY FOR THE DIMENSIONS OF ADULT SKIN PICKING

The most recently developed measure of skin picking is the Milwaukee Inventory for the Dimensions of Adult Skin Picking (MIDAS; Walther et al., 2009). Building on previous work in PSP and trichotillomania (Arnold et al., 2001; Christenson & Mackenzie, 1994; Flessner et al., 2008b), Walther et al. sought to develop a measure that would distinguish between automatic and focused picking. The self-report measure includes six items assessing automatic picking (e.g., "I am usually not aware of picking my skin during the picking episode") and six items assessing focused picking (e.g., "I pick my skin when I am experiencing a negative emotion such as stress, anger, frustration, or sadness"). Each item is rated on a 1 (*not true of any of my skin picking*) to 5 (*true for all of my skin picking*) scale. Thus, scores on each subscale range from 6 to 30. Initial testing of the MIDAS has demonstrated adequate internal consistency (automatic subscale: α = .77; focused subscale: α = .81) and good construct validity.

SKIN PICKING IMPACT SCALE

The Skin Picking Impact Scale (SPIS; Keuthen et al., 2001a) is a 10-item self-report measure of the emotional (e.g., "I feel embarrassed because of my skin picking"), social (e.g., "I think my social life would be better if I didn't pick my skin"), and behavioral (e.g., "It takes me longer to go out because of my skin picking") consequences of skin picking. Respondents rate how much each item applies to them in the previous week on a 0 (*none*) to 5 (*severe*) scale. Thus, total scores range from 0 to 50. The SPIS has shown high internal consistency (α = .93) and good construct validity in a sample of self-injurious skin pickers. A cutoff score of 7 or above was shown to reliably distinguish self-injurious skin pickers from non-self-injurious skin pickers.

SKIN PICKING SCALE

The Skin Picking Scale (SPS; Keuthen et al., 2001b) is a six-item self-report measure modeled after the Y-BOCS (Goodman et al., 1989), assessing the frequency and intensity of the urge to pick and the time spent picking, as well as interference, distress, and avoidance due to skin picking. Each item is rated on a 0 (*none*) to 4 (*extreme*) scale. Thus, total scores range from 0 to 24. Initial testing revealed moderate internal consistency (α = .80) and good construct validity. Similar to the SPIS, a cutoff score of 7 or above was shown to reliably distinguish self-injurious skin pickers from non-self-injurious skin pickers.

SKIN PICKING SYMPTOM ASSESSMENT SCALE

The Skin Picking Symptom Assessment Scale (SP-SAS; Grant et al., 2007) is a 12-item self-report measure of skin picking modeled after two earlier measures of kleptomania (Grant & Kim, 2002) and pathological gambling (Kim et al., 2001). Respondents are asked to rate each symptom over the past week on a 0 to 4 scale. Total scores range from 0 to 48. As with the NE-YBOCS, no psychometric papers have been published on the SP-SAS. It has, however, demonstrated adequate test-retest reliability (ρ = .74) and satisfactory construct validity when compared with other outcome measures in one treatment outcome study (Grant et al., 2007).

Review of the Treatment Outcome Literature

Clinical trials designed specifically to treat PSP are few in number, and methodological ambiguities complicate interpretation. The literature is dominated by case reports, along with a few open trials and even fewer controlled and/or double-blind trials, all with small samples. Although definitive conclusions are difficult to draw, the studies suggest the utility of pharmacological and psychosocial treatments, including serotonergic and glutamatergic medications and CBT in treating PSP. In the following sections, we review the treatment outcome literature for PSP.

Pharmacotherapy and Biological Treatment

Simeon and colleagues (1997) conducted a double-blind pharmacotherapy trial in which they randomized 21 individuals with PSP to a 10-week course of fluoxetine (20–80 mg/day; *M* = 53–55 mg/day) or placebo. In completer analyses, fluoxetine led to greater improvement on two of three outcome measures: the Clinical Global Impression-Improvement Scale (CGI-I) and a self-report visual analog scale rating change in picking, but not the clinician-rated SPTS. Intent-to-treat analyses revealed significantly greater improvement only on the self-report measure. The magnitudes of some nonsignificant effects (SPTS in the completer analyses and CGI-I in the intent-to-treat analyses) were moderate or large, however, suggesting that the differences are meaningful, especially in light of the small sample size and resultant low statistical power. Furthermore, all 6 completers who received fluoxetine (and 8 of 10 who were randomized to receive fluoxetine) achieved clinical responder status compared with 3 of 11 on placebo. Of note, four participants dropped out of the fluoxetine condition: two had improved considerably (CGI-I = 2) and two had worsened considerably (CGI-I = 6), at least one of whose deterioration was clearly caused by the medication.

Four open-label trials of selective serotonin reuptake inhibitors (SSRIs) have been conducted for the treatment of PSP. Bloch and colleagues (2001) investigated fluoxetine in a sample of 15 subjects, all of whom received the study medication (20–60 mg/day) for 6 weeks. Responders (*n* = 8) were then randomized, double-blind, to receive continued fluoxetine at the dosage reached by week 6 or placebo for 6 more weeks. Overall, responders who received continued fluoxetine were considerably improved from baseline to week 12 on measures of skin picking (NE-YBOCS and a 7-item, then-unpublished version of the SPS), whereas those who were randomized to placebo returned to their baseline functioning by week 12. These results are promising, but conclusions regarding the efficacy of fluoxetine should be tempered by the finding that nearly half of the original 15 subjects did not respond to fluoxetine in the first arm of the study. Arnold and colleagues (1999) administered fluvoxamine (25–300 mg/day; *M* = 112.5 mg/day) to 14 subjects over 12 weeks and found improvement in picking behavior and global symptoms (but not in symptoms of depression) on both observer (NE-YBOCS) and self-report (visual analog scales) measures. However, half of the subjects dropped out of the study, four (29%) because of medication-related side effects. Keuthen and colleagues (2007) treated 27 individuals with escitalopram (5–30 mg/day; *M* = 25 mg/day) for 18 weeks. Following treatment, the sample was generally improved on measures of skin picking (SPTS, SPIS, and SPS, as well as the CGI), mood, and quality of life. Of the 27 participants, 20 were classified as full or partial responders and 4 discontinued treatment due to

side effects. Finally, Kalivas and colleagues (1996) report a case series of 31 patients with various self-injurious skin-directed disorders (26 of whom were described as having "neurotic excoriations") who were treated with sertraline (25–200 mg/day; *M* = 95 mg/day in responders and 72 mg/day in nonresponders) for an unspecified length of time. Three patients were not evaluated due to "intolerable adverse effects" (p. 589). Among the 28 participants who were evaluated, 19 experienced at least 50% reduction in open skin lesions.

Recently, Grant and colleagues (2007) conducted an open-label trial of lamotrigine, an anticonvulsant thought to affect glutamate. Twenty-four subjects were enrolled to receive lamotrigine (12.5–300 mg/day) for 12 weeks. As a group, subjects improved notably on measures of time spent picking as well as on secondary measures. Of the 20 subjects who completed the course of treatment, 16 were deemed responders. Only one of the four dropouts discontinued treatment because of a medication side effect. This study is notably the first to examine a non-SSRI in the treatment of PSP.

In addition to these few clinical trials, numerous case reports document the apparent utility of SSRIs, including fluoxetine (e.g., Gupta & Gupta, 1993), fluvoxamine (e.g., O'Sullivan et al., 1999), escitalopram (e.g., Pukadan et al., 2008), and paroxetine (e.g., Biondi et al., 2000) in treating at least some patients suffering from PSP (for reviews, see Arnold et al., 2001; Grant & Odlaug, 2009). Other authors, however, have published case reports suggesting that SSRIs may actually exacerbate skin picking in some patients (Denys et al., 2003). In addition to SSRIs, other pharmacological agents have also reportedly been effective in treating individual patients, including antidepressants such as clomipramine (e.g., Gupta et al., 1986) and doxepin (Harris et al., 1987); atypical or typical antipsychotics such as olanzapine (e.g., Blanch et al., 2004) and pimozide (Duke, 1983); the putative glutamate modulator, *N*-acetylcysteine (Odlaug & Grant, 2007); and naltrexone (Lienemann & Walker, 1989). Researchers have also reported success in augmenting previously inefficacious courses of some medications (e.g., citalopram, fluoxetine, venlafaxine) by adding other treatments, including atypical antipsychotics such as olanzapine (e.g., Christensen, 2004) and aripiprazole (Carter & Shillcutt, 2006; Curtis & Richards, 2007; Ginsberg, 2006); the antiglutamatergic agent riluzole (Sasso et al., 2006); inositol (Seedat et al., 2001); and CBT (Sahin et al., 2004).

A recent case report suggests that neurosurgery may be effective in reducing dangerous skin picking in patients who do not respond to other forms of treatment. Kondziolka and Hudak (2008) documented the effectiveness of bilateral anterior capsulotomies using gamma knife radiosurgery in a patient with treatment-refractory skin picking in the context of severe OCD. The patient had not responded to numerous medication trials and courses of behavior therapy, and had received multiple skin grafts, many of which he picked through. By 7 months postsurgery, his skin picking and OCD symptoms had decreased somewhat, and he was accepted for 2 months of inpatient therapy. By 17 months postsurgery, the skin picking was sufficiently reduced to allow the wound to begin to heal, and the OCD symptoms were greatly improved.

Psychosocial Treatment

PSYCHODYNAMIC AND ECLECTIC THERAPIES

In 1953, Seitz published a case series of brief psychodyanamic psychotherapy in 25 patients with "psychocutaneous excoriation syndromes" who did not respond to dermatological treatments. Based on earlier case reports and psychological studies of skin disorders, Seitz suggested that scratching is a conversion reaction stemming from "unexpressed rage and guilt, as well as the unsatisfied wishes for love," and that excoriation releases "tension associated with repressed rage; it atones for guilt by means of mutilating self-punishment; and it provides regressive gratification of the need for love through cutaneous-erotic masturbatory pleasure" (p. 201). Treatment itself consisted of 12 weekly sessions designed to elicit the verbal expression of rage and inferiority and "dilution" (p. 201) of the associated guilt and shame. Seitz reported that all 25 patients experienced temporary exacerbations of the cutaneous disorder, usually at the eighth session, and that 12 of the patients dropped out of treatment. For 12 of the 13 completers, however, the excoriation had resolved by acute posttreatment (i.e., "symptomatic cure," defined as "clearing of the lesions and absence of itching" [p. 201]), and all 12 maintained their gains at 3-month follow-up. Of those patients available for longer-term follow-up, six of seven were still in remission after 6 months and four of five after 1 year (different patients had relapsed at 6 months and 1 year). Participants who completed treatment differed at baseline from those who discontinued. They had significantly more mild skin lesions and were rated as more motivated. Although uncontrolled, this case series suggests that brief psychodynamic psychotherapy may be efficacious for individuals with mild skin picking who

are motivated. Other reports indicate lower rates of treatment success with psychodynamic or eclectic therapy (e.g., Fruensgaard, 1991a, 1991b).

COGNITIVE-BEHAVIOR THERAPY

Six studies and a number of case reports document the utility of various cognitive-behavioral techniques in treating PSP. These techniques are often implemented in combination with each other and are described in more detail below (see "Cognitive-Behavioral Techniques: A Clinician's Toolbox").

Only one between-groups controlled trial of CBT for PSP has been conducted. Teng, Woods, and Twohig (2006) compared brief habit reversal training (HRT; Azrin & Nunn, 1973; Azrin & Peterson, 1988) to no treatment (waiting list control; WL) in a sample of 19 chronic skin pickers. In this study, HRT consisted of three weekly meetings: a single 1-hour session of awareness and competing response training, followed by two half-hour booster sessions. Competing response training involved "participants [learning] to clench their fists for 1 min . . . contingent on the skin picking and its antecedents" (p. 416). Teng and colleagues found that HRT outperformed WL on self-monitored tallies of skin-picking frequency (77% versus 16% reduction in self-reported skin picking in the HRT and WL groups, respectively) and photograph ratings, and noted that the gains from HRT were maintained for at least 3 months. None of the participants had entirely ceased picking at posttreatment, however. Nevertheless, these results suggest that HRT can be an effective and fast-acting intervention for individuals with chronic PSP.

In addition to the study by Teng and colleagues (2006), two other studies have examined HRT for PSP using experimental designs. Using a multiple baseline across participants design, Twohig and Woods (2001) treated two brothers who engaged in damaging picking of their fingers with brief HRT similar in form and length to the treatment used by Teng et al. The frequency of picking and skin damage decreased for both participants after implementation of treatment; however, only one of the participants fully maintained his gains at follow-up. Lane and colleagues (2006) reported on the use of "competing activities" for a 9-year-old boy with PSP and comorbid attention deficit hyperactivity disorder (ADHD), low IQ, and learning and speech-language difficulties. At the beginning of each day, the boy was given a choice of three plastic balls "to keep his hands occupied so as to avoid skin picking" (p. 460). Using an ABCBAB design, Lane and colleagues measured the percentage of time spent picking over the course of 29 days during which the child received medication only (Adderall for ADHD), medication with the intervention, or the intervention alone. The use of competing activities decreased the time spent picking (measured via direct observation) and appeared most effective when combined with medication for ADHD. No follow-up data or measures of long-term improvement in picking or the urge to pick were reported.

Two studies have examined acceptance-based forms of CBT using multiple-baseline designs. Twohig and colleagues (2006) examined the treatment of five chronic skin pickers with eight weekly 1-hour sessions of acceptance and commitment therapy (ACT; Hayes et al., 1999). Four of the five participants evidenced considerable treatment gains (i.e., low levels of self-monitored skin picking frequency and improvement in visible skin damage) at acute posttreatment; however, only one maintained the gains at 3-month follow-up. The results of this study suggest that ACT alone can be helpful, at least in the short term, but that "additional treatment development is needed particularly in the maintenance of gains" (p. 1520). It is noteworthy that the use of HRT techniques was explicitly proscribed in the aforementioned study. To evaluate the utility of combining techniques of ACT and HRT, Flessner and colleagues (2008a) conducted a pilot study of acceptance-enhanced behavior therapy in five individuals with chronic PSP or trichotillomania. The two participants with PSP improved (50% reduction in skin-picking severity), although somewhat less than did those with trichotillomania (65% reduction). Both participants with PSP initially received HRT but did not evince improvement until the addition of ACT techniques. It is impossible to know whether the addition of ACT techniques per se was responsible for the improvement (e.g., as opposed to continued time and effort in treatment).

There is evidence, as well, for the effectiveness of an Internet-based self-help program that follows the principles of HRT for PSP. In the only large-scale study of CBT for PSP ($N = 372$), Flessner and colleagues (2007) conducted an uncontrolled program evaluation of the computer-based intervention at www.StopPicking.com. The intervention lasted an average of 12 weeks and comprised an assessment phase, during which individuals monitored picking behavior and its associated antecedents and consequences; an intervention phase, which included various cognitive-behavioral coping strategies

(e.g., stimulus control, use of competing responses, relaxation, cognitive restructuring); and a maintenance phase. Postintervention, 63% of participants were deemed responders on the basis of achieving a 25% reduction in scores on the SPS (Keuthen et al., 2001b).

A few other case reports have added to the literature suggesting that PSP can be treated successfully and relatively quickly with HRT. In four patients with neurodermatitis, Rosenbaum and Ayllon (1981) found that a single treatment session induced a marked decrease in skin picking for at least 6 months. In another report, a woman with acne excoriée was able to reduce the time she spent picking from 14 hours to less than 10 minutes per week after 1 month of HRT, and the gains were still evident after 4 months (Kent & Drummond, 1989). Noting the difficulty of making inferences from treatment outcomes in skin picking associated with a dermatological condition to cases of picking without any such condition, Deckersbach and colleagues (2002) reported three cases of people with chronic skin picking in the absence of a dermatological condition and in patients with psychiatric comorbidity. All were treated with CBT that included HRT but was supplemented with other techniques, such as those to increase emotion regulation skills. In two cases, relatively brief courses of treatment (four and seven sessions) were associated with considerable behavioral gains, and in the third, the patient ceased picking after 2 years of therapy, six sessions of which were focused on skin picking. Deckersbach et al. (2003) treated a woman with severe PSP (automatic and focused) related to BDD with eight sessions of CBT that included HRT, cognitive restructuring, and emotion regulation training. Over the course of treatment, the patient decreased the frequency of skin-picking episodes from 15–20 per day to fewer than 3 per day, and her self-report symptom scores decreased by approximately 50%. These gains were maintained at 3-month follow-up. Of note, she evinced improvements in automatic skin picking more rapidly than in picking that served an emotion regulation function. Finally, Welkowitz et al. (1989) described the successful treatment of a man with excoriations at multiple sites with an intervention that included various behavioral strategies similar to elements of HRT.

Summary of Outcome Literature

Although large-scale studies are lacking, one double-blind study and five open-label trials suggest that SSRIs or glutamatergic agents may be efficacious in treating PSP for at least a subgroup of individuals. Enthusiasm is reduced by high or equivocal rates of nonresponse in some studies as well as notable dropout rates. Furthermore, the lack of clear diagnostic criteria and the use of variable outcome measures complicate interpretation (limitations common to studies of psychosocial treatments as well). Moreover, it is not known whether the efficacy of continued pharmacological treatment wanes over time. Case reports document the possibility that various other pharmacological agents may improve treatment outcome when used instead of, or to augment, serotonergic medications. These await further investigation.

One between-groups study and several small experimental paradigms suggest that CBT has utility in treating PSP and has the potential to induce gains quickly. Cognitive-behavior therapy treatments for PSP include HRT, acceptance and mindfulness, and various other techniques that are now described in more detail.

Cognitive-Behavioral Techniques: A Clinician's Toolbox

As is evident from the treatment outcome literature, a number of CBT techniques are helpful for patients with PSP, often in combination with each other. They include HRT, stimulus control, acceptance and mindfulness, cognitive strategies, relaxation training, emotion regulation, and various other tools. The population of individuals who engage in PSP is quite heterogeneous with respect to the underlying pathology, and the formulation of a treatment plan as well as the selection of intervention techniques must be tailored to the individual (see "Cognitive-Behavioral Model of Skin Picking"). In the following section, we describe various CBT techniques with utility in treating individuals with PSP.

Habit Reversal Training

Habit reversal training is an approach to treating habitual behaviors of many types (Azrin & Nunn, 1973; Azrin & Peterson, 1988) that incorporates a number of specific techniques at least some of which are present in many variations of CBT for PSP. The techniques include awareness training, the use of competing responses, and relaxation (described separately below).

Chronic skin pickers are frequently not aware of their picking, especially automatic pickers. Even those who engage in focused picking may not be cognizant of the psychological and environmental antecedents or consequences of picking, or the

specific sequence and nature of the picking behavior itself. Hence, prerequisite to choosing an appropriate intervention strategy and, of course, implementing it in real time, is that the patients identify and recognize when and precisely how they pick. Awareness training is typically conducted both in and out of session and involves monitoring the occurrence of picking episodes (e.g., by tallying the number of such episodes and/or urges during set periods of time), as well as describing, frame by frame, what transpires before, during, and following picking. Ultimately, automaticity in detecting warning signs of picking is a critical antidote to the automaticity of habitual picking.

Competing responses are behavioral alternatives that are used to contravene episodes of picking, and involve engaging in behavior opposite to, and incompatible with, the patient's idiosyncratic picking behavior. For example, a patient who picks with her fingernails might make a fist or hold an object whenever she experiences the urge to pick. The patient is taught to select a competing response that can be maintained long enough for the urge to pass (minimum 1 minute) and that does not disrupt necessary activities during potential picking episodes (e.g., socially inconspicuous for someone who picks at work). Targeted practice and reinforcement are used to facilitate the application and generalization of competing responses.

Stimulus Control

To the extent reasonable, environmental antecedents and triggers are manipulated to decrease the likelihood of picking or the intensity of the urge to do so. Patients who engage in automatic picking can avoid behaviors and situations during which they mindlessly pick. For example, someone who watches television or reads with his chin resting in his palm, which leads to mindless skin touching and ultimately picking, may intentionally keep his hands away from his face (e.g., in his pockets or otherwise occupied) during those activities. Similarly, he may engage in prophylactic use of competing responses upon entering situations of risk. Patients who engage in focused picking can also find ways to disrupt the cycle of picking. For example, a patient who picks in the mirror may remove the mirror, alter the lighting in the room, or smear petroleum jelly on the mirror.

Acceptance and Commitment Therapy

Acceptance and commitment therapy (ACT; Hayes et al., 1999) is a form of CBT that emphasizes the roles of experiential avoidance and behavioral rigidity in maintaining dysfunctional behavior. Acceptance and commitment therapy is highly compatible with HRT in treating PSP, and some have suggested that ACT may be particularly well suited for individuals who engage in focused picking to regulate emotion, as opposed to automatic picking (Flessner et al., 2008a). The full ACT treatment protocol is available on the Internet (http://www.contextualpsychology.org/treatment_protocols); it is written for OCD, but can be applied to skin picking by changing the term *obsession* to *urge to pick* (M. Twohig, personal communication, August 24, 2009).

As implemented by Twohig et al. (2006), ACT for PSP involves five steps that are learned and practiced over the course of 8 weeks:

1. Efforts to eliminate urges to pick, as well as other internal events (e.g., affective or motivational), are not effective. Hence, whereas many individuals who engage in pathological habitual behavior struggle to resist the negative affective experience of the urge itself, efforts to do so are highly unlikely to succeed.
2. Moreover, attempts to control urges themselves create or maintain the problem because in many cases they paradoxically exacerbate or magnify the internal experience or perceived lack of efficacy in managing it.
3. There is a difference between uncontrollable urges and controllable behaviors. Whereas efforts to resist the experience of an urge are likely unhelpful, those to resist the behavioral response to the urge (e.g., picking) are feasible, provided that one is willing to experience the discomfort of the urge. The mechanism of treatment is therefore not to decrease the urge, but rather to eliminate the picking.
4. Withstanding the urge to pick is difficult, especially if the urge itself is perceived as harmful or threatening in some way. Various exercises are introduced and practiced to alter the perception of verbal events (e.g., thoughts, urges) as threatening or overly meaningful.
5. Ultimately, rather than succumb to urges by choosing unwanted behavior designed to avoid or neutralize internal experiences such as urges or affective states, the individual learns to pursue valued action whatever the immediate internal cost. That is, the patient learns to act rather than react. In the case of PSP, the patient chooses not to pick because he or she does not wish to pick, without regard to whether that choice is likely to decrease the experience of negative internal states.

Cognitive Strategies

In contrast to the use of HRT and ACT strategies, which are fundamentally suited to most individuals with PSP, the choice to supplement CBT with cognitive techniques is particular to the patient's clinical manifestation. For example, Deckersbach et al. (2002) describe patients whose picking episodes were often triggered by negative emotions such as sadness, some of which were fueled by dysfunctional beliefs (e.g., "My face looks horrible" [p. 371]; "I will never find a job" [p. 371]; "I cannot handle this" [p. 372]; "I have to get rid of this scab" [p. 372]). In such cases, cognitive restructuring (e.g., Beck, 1995) can be helpful to address underlying beliefs and assumptions. In general, CBT approaches to skin picking typically incorporate cognitive techniques as a flexible and optional module when appropriate. Often these strategies are most relevant when the urge to pick is triggered or accompanied by dysfunctional beliefs about oneself or one's ability to manage distress and regulate emotions.

Relaxation Training

Picking behavior may exacerbate and be exacerbated by generalized stress and tension. Hence, HRT frequently incorporates relaxation training, which can be used regularly as a means of lowering baseline stress, as well as strategically as a complement to competing responses to resist acute urges to pick.

There are numerous variations of relaxation training that differ in terms of complexity and refinement. Progressive muscle relaxation (Bernstein & Borkovec, 1973) and applied relaxation (Öst, 1987) are formal protocols through which an individual learns to relax by systematically tensing and then relaxing various muscle groups. Ultimately, the individual learns to engage in cued (without tensing, and in response to a self-generated cue) and differential (while performing another activity) relaxation and to apply relaxation in vivo. As such, relaxation training is a bona fide treatment with demonstrated efficacy for a number of disorders (e.g., Manzoni et al., 2008; Öst, 1987). However, less comprehensive relaxation training can be used as an adjunct to other CBT techniques. This can include simplified versions of muscle relaxation (e.g., focusing on four muscle groups; cf. Wolpe & Lazarus, 1966) as well as diaphragmatic breathing. Implementation of relaxation should be tailored to the individual, who may find utility in using it to lower generalized tension, resist the urge to pick, and/or regulate emotion.

Emotion Regulation

Clinicians have noted that some individuals engage in focused PSP to modulate negative affect or emotion dysregulation. Moreover, anecdotal evidence and studies of trichotillomania suggest that self-injurious BFRBs with an emotion regulation quality may respond less well or more slowly to straightforward behavioral treatment (e.g., Deckersbach et al., 2002, 2003; Flessner et al., 2008a). Therefore, individuals for whom picking is triggered by affective distress or who pick to self-sooth (common, for example, in individuals with borderline personality disorder) may benefit from the development of emotion regulation and distress tolerance skills. Specific techniques include relaxation, exercise, participation in enjoyable activities, acting opposite to the distressing emotion, distraction, and other forms of self-soothing (for a comprehensive list of emotion regulation and distress tolerance strategies, see Linehan, 1993).

Other CBT Techniques

At times, PSP is manifest as an epiphenomenon in the context of another disorder. When it is symptomatic of another underlying disorder, intervention strategies that focus exclusively on the picking behavior are unlikely to be sufficient. In such cases, developing a treatment plan requires attention not only to the behavior itself, but to the underlying symptoms and motivation to pick as well. For example, more than one-third of patients with BDD engage in skin picking, often to smooth or remove perceived blemishes (Grant et al., 2006). For those individuals, the BDD treatment protocol would likely incorporate cognitive restructuring, as well as exposure and response prevention, HRT, and stimulus control techniques. Habit reversal training would be useful to facilitate response prevention (i.e., resisting the urge to pick during exposures) but would not adequately address the BDD by itself.

Future Directions

Research on PSP has grown in recent years; however, treatment studies are notably lacking in number and size. Considering the widely discrepant manifestations of PSP behavior, this significantly limits the ability to make clinical inferences on the basis of the empirical literature. Certainly, larger comprehensive investigations are warranted. In addition, future research would benefit from attention to a number of issues that follow.

We have emphasized throughout this chapter the heterogeneity of PSP. Indeed, examination of the treatment studies and case reports reveals samples drawn from a range of populations that differ on factors one might presume to predict treatment outcome, including type of picking (e.g., automatic versus focused) and diagnosis (e.g., the presence or absence of a dermatological condition, psychiatric disorder, and comorbidity). The severity of picking itself is not consistent, ranging from minor finger picking to dangerous self-mutilation. The extent to which these factors affect treatment is virtually unknown. Considering a condition with such variability, Paul's fundamental question of psychotherapy research is particularly salient: "*What* treatment, by *whom*, is most effective for *this* individual with *that* specific problem, and under *which* set of circumstances?" (1967, p. 111; emphasis in the original).

Larger studies would not only engender greater confidence in the efficacy of treatments for PSP (or lack thereof), but would permit examination of the clinical observation that subtypes of PSP respond differentially to various treatments (Grant & Odlaug, 2009). For example, a number of researchers have suggested that automatic picking is more responsive to behavioral interventions than is focused picking designed to regulate emotion. Therefore, mindfulness and emotion regulation skills may be particularly helpful for individuals who engage in the latter (Deckersbach et al., 2002, 2003; Flessner et al., 2008a). Similarly, a subset of patients with PSP seem not to respond to SSRIs (Keuthen et al., 2007) or even to deteriorate (Denys et al., 2003). Keuthen and colleagues (2007) offer the possibility that such differential outcome depends on whether the PSP behavior is impulsive or compulsive (see also Arnold et al., 2001). Without larger, systematic studies, however, such accounts are speculative.

Even to the extent that studies that directly examine moderation are not feasible, efforts to articulate and standardize diagnostic criteria would markedly facilitate the conduct and comparative interpretation of treatment outcome studies, as well as the quality of empirically grounded clinical care (Bohne et al., 2002). Likewise, the field would benefit from further evaluation (e.g., psychometric properties) of measures that permit careful assessment of PSP and, ultimately, treatments better tailored to the individual. Existing studies are difficult to compare in part because they utilize a variety of outcome measures, many with unknown psychometric properties. This complicates even the evaluation of severity.

Finally, efforts to improve treatment outcome are generally warranted. Wilhelm et al. (1999) report retrospective data that suggest rather limited benefit from both behavioral and pharmacological treatments for most individuals with PSP. In addition, rates of nonresponse remain high in some pharmacological studies, and little is known about psychosocial treatment response rates. Future research should examine whether, as suggested by numerous case reports, combining medications and/or psychotherapy techniques can increase treatment success (Grant & Odlaug, 2009).

Author Note

The authors wish to thank Nancy Keuthen, Ph.D., for suggestions and guidance.

References

American Psychiatric Association (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision). Washington, DC: Author.

Arnold, L. M., Auchenbach, M. B., & McElroy, S. L. (2001). Psychogenic excoriation: Clinical features, proposed diagnostic criteria, epidemiology, and approaches to treatment. *CNS Drugs, 15*, 351–359.

Arnold, L. M., Mutasim, D. F., Dwight, M. M., Lamerson, C. L., Morris, E. M., & McElroy, S. L. (1999). An open clinical trial of fluvoxamine treatment of psychogenic excoriation. *Journal of Clinical Psychopharmacology, 19*, 15–18.

Azrin, N. H., & Nunn, R. G. (1973). Habit-reversal: A method of eliminating nervous habits and tics. *Behaviour Research and Therapy, 11*, 619–628.

Azrin, N. H., & Peterson, A. L. (1988). Habit reversal for the treatment of Tourette Syndrome. *Behaviour Research and Therapy, 26*, 347–351.

Beck, J. S. (1995). *Cognitive therapy: Basics and beyond*. New York: Guilford.

Begotka, A. M., Woods, D. W., & Wetterneck, C. T. (2003). The relationship between experiential avoidance and the severity of trichotillomania in a nonreferred sample. *Journal of Behavior Therapy and Experimental Psychiatry, 35*, 17–24.

Bernstein, D. A., & Borkovec, T. D. (1973). *Progressive relaxation training: A manual for the helping professions*. Champaign, IL: Research Press.

Bienvenu, O. J., Samuels, J. F., Riddle, M. A., Hoehn-Saric, R., Liang, K. Y., Cullen, B. A. M., Grados, M. A., & Nestadt, G. (2000). The relationship of obsessive-compulsive disorder to possible spectrum disorders: Results from a family study. *Biological Psychiatry, 48*, 287–293.

Biondi, M., Arcangeli, T., & Petrucci, R. M. (2000). Paroxetine in a case of psychogenic pruritus and neurotic excoriations [Letter to the Editor]. *Psychotherapy and Psychosomatics, 69*, 165–166.

Blanch, J., Grimalt, F., Massana, G., & Navarro, V. (2004). Efficacy of olanzapine in the treatment of psychogenic excoriation [Letter to the Editor]. *British Journal of Dermatology, 151*, 714–716.

Bloch, M. R., Elliott, M., Thompson, H., & Koran, L. M. (2001). Fluoxetine in pathologic skin-picking: Open-label and double-blind results. *Psychosomatics, 42*, 314–319.

Bohne, A., Wilhelm, S., Keuthen, N. J., Baer, L., & Jenike, M. A. (2002). Skin picking in German students: Prevalence, phenomenology, and associated characteristics. *Behavior Modification, 26*, 320–339.

Carter, W. G., & Shillcutt, S. D. (2006). Aripiprazole augmentation of venlafaxine in the treatment of psychogenic excoriation [Letter to the Editor]. *Journal of Clinical Psychiatry, 67*, 1311.

Christensen, R. (2004). Olanzapine augmentation of fluoxetine in the treatment of pathological skin picking [Letter to the Editor]. *Canadian Journal of Psychiatry, 49*, 788–789.

Christenson, G. A., & Mackenzie, T. B. (1994). Trichotillomania. In M. Hersen & R. T. Ammerman (Eds.), *Handbook of prescriptive treatment for adults* (pp. 217–235). New York: Plenum Press.

Christenson, G. A., Mackenzie, T. B., & Mitchell, J. E. (1991). Characteristics of 60 adult chronic hair pullers. *American Journal of Psychiatry, 148*, 365–370.

Christenson, G. A., Ristvedt, S. L., & Mackenzie, T. B. (1993). Identification of trichotillomania cue profiles. *Behaviour Research and Therapy, 31*, 315–320.

Curtis, A. R., & Richards, R. W. (2007). The treatment of psychogenic excoriation and obsessive compulsive disorder using aripiprazole and fluoxetine [Letter to the Editor]. *Annals of Clinical Psychiatry, 19*, 199–200.

Deckersbach, T., Wilhelm, S., & Keuthen, N. (2003). Self-injurious skin picking: Clinical characteristics, assessment methods, and treatment modalities. *Brief Treatment and Crisis Intervention, 3*, 249–260.

Deckersbach, T., Wilhelm, S., Keuthen, N. J., Baer, L., & Jenike, M. A. (2002). Cognitive-behavior therapy for self-injurious skin picking: A case series. *Behavior Modification, 26*, 361–377.

Denys, D., van Megen, H. J. G. M., & Westenberg, H. G. M. (2003). Emerging skin-picking behaviour after serotonin reuptake inhibitor-treatment in patients with obsessive-compulsive disorder: Possible mechanisms and implications for clinical care. *Journal of Psychopharmacology, 17*, 149–151.

Diefenbach, G. J., Tolin, D. F., Meunier, S., & Worhunsky, P. (2008). Emotion regulation and trichotillomania: A comparison of clinical and nonclinical hair pulling. *Journal of Behavior Therapy and Experimental Psychiatry, 39*, 32–41.

Duke, E. E. (1983). Clinical experience with pimozide: Emphasis on its use in postherpetic neuralgia. *Journal of the American Academy of Dermatology, 8*, 845–850.

Flessner, C. A., Busch, A., Heidemann, P., & Woods, D. W. (2008a). Acceptance-enhanced behavior therapy (AEBT) for trichotillomania and chronic skin picking: Exploring the effects of component sequencing. *Behavior Modification, 32*, 579–594.

Flessner, C. A., Mouton-Odum, S., Stocker, A. J., & Keuthen, N. J. (2007). StopPicking.com: Internet-based treatment for self-injurious skin picking. *Dermatology Online Journal, 13*, 3.

Flessner, C. A., Woods, D. W., Franklin, M. E., Cashin, S. E., & Keuthen, N. J. (2008b). The Milwaukee Inventory for Subtypes of Trichotillomania (MIST-A): Development of an instrument for the assessment of "focused" and "automatic" hair pulling. *Journal of Psychopathology and Behavioral Assessment, 30*, 20–30.

Franklin, M. E., & Tolin, D. F. (2007). *Treating trichotillomania: Cognitive-behavioral therapy for hairpulling and related problems*. New York: Springer.

Fruensgaard, K. (1991a). Psychotherapeutic strategy and neurotic excoriations. *International Journal of Dermatology, 30*, 198–203.

Fruensgaard, K. (1991b). Psychotherapy and neurotic excoriations. *International Journal of Dermatology, 30*, 262–265.

Ginsberg, D. L. (2006). Addition of aripiprazole to venlafaxine useful in pathologic skin picking. *Psychiatry Weekly*. Retrieved August 14, 2009, from http://www.psychiatryweekly.com/aspx/article/ArticleDetail.aspx?articleid=312

Goodman, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill, C. L., Heninger, G. R., & Charney, D. S. (1989). The Yale-Brown Obsessive-Compulsive Scale: I. Development, use, and reliability. *Archives of General Psychiatry, 46*, 1006–1011.

Grachev, I. D. (1997). MRI-based morphometric topographic parcellation of human neocortex in trichotillomania. *Psychiatry Clinical Neuroscience, 51*, 315–321.

Grant, J. E., & Kim, S. W. (2002). An open-label study of naltrexone in the treatment of kleptomania. *Journal of Clinical Psychiatry, 63*, 349–356.

Grant, J. E., Menard, W., & Phillips, K. A. (2006). Pathological skin picking in individuals with body dysmorphic disorder. *General Hospital Psychiatry, 28*, 487–493.

Grant, J. E., & Odlaug, B. L. (2009). Update on pathological skin picking. *Current Psychiatry Reports, 11*, 283–288.

Grant, J. E., Odlaug, B. L., & Kim, S. W. (2007). Lamotrigine treatment of pathological skin picking: An open-label study. *Journal of Clinical Psychiatry, 68*, 1384–1391.

Gupta, M. A., & Gupta, A. K. (1993). Olanzapine is effective in the management of some self-induced dermatoses: Three case reports. *Cutis, 66*, 143–146.

Gupta, M. A., Gupta, A. K., & Haberman, H. F. (1986). Neurotic excoriations: A review and some new perspectives. *Comprehensive Psychiatry, 27*, 381–386.

Harris, B. A., Sherertz, E. F., & Flowers, F. P. (1987). Improvement of chronic neurotic excoriations with oral doxepin therapy. *International Journal of Dermatology, 26*, 541–543.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy: An experiential approach to behavior change*. New York: Guilford Press.

Kalivas, J., Kalivas, L., Gilman, D., & Hayden, C. T. (1996). Sertraline in the treatment of neurotic excoriations and related disorders [Letter to the Editor]. *Archives of Dermatology, 132*, 589–590.

Kent, A., & Drummond, L. M. (1989). Acne excoriée—A case report of treatment using habit reversal. *Clinical and Experimental Dermatology, 14*, 163–164.

Keuthen, N. J., Deckersbach, T., Wilhelm, S., Engelhard, I., Forker, A., O'Sullivan, R. L., Jenike, M. A., & Baer, L. (2001a). The skin picking impact scale (SPIS): Scale development and psychometric analysis. *Psychosomatics, 42*, 397–403.

Keuthen, N. J., Deckersbach, T., Wilhelm, S., Hale, E., Fraim, C., Baer, L., O'Sullivan, R. L., & Jenike, M. A. (2000). Repetitive skin-picking in a student population and comparison with a sample of self-injurious skin-pickers. *Psychosomatics, 41*, 210–215.

Keuthen, N. J., Jameson, M., Loh, R., Deckersbach, T., Wilhelm, S., & Dougherty, D. D. (2007). Open-label escitalopram treatment for pathological skin picking. *International Clinical Psychopharmacology, 22*, 268–274.

Keuthen, N. J., Wilhelm, S., Deckersbach, T., Engelhard, I. M., Forker, A. E., Baer, L., & Jenike, M. A. (2001b). The skin picking scale: Scale construction and psychometric analysis. *Journal of Psychosomatic Research, 50*, 337–341.

Kim, S. W., Grant, J. E., Adson, D. E., & Shin, Y. C. (2001). Double-blind naltrexone and placebo comparison study in the treatment of pathological gambling. *Biological Psychiatry, 49*, 914–921.

Kondziolka, D., & Hudak, R. (2008). Management of obsessive-compulsive disorder–related skin picking with gamma knife radiosurgical anterior capsulotomies: A case report [Letter to the Editor]. *Journal of Clinical Psychiatry, 69*, 1337–1340.

Lane, K. L., Thompson, A., Reske, C. L., Gable, L. M., & Barton-Arwood, S. (2006). Reducing skin picking via competing activities. *Journal of Applied Behavior Analysis, 39*, 459–462.

Lienemann, J., & Walker, F. D. (1989). Reversal of self-abusive behavior with naltrexone [Letter to the Editor]. *Journal of Clinical Psychopharmacology, 9*, 448–449.

Linehan, M. M. (1993). *Skills training manual for treating borderline personality disorder*. New York: Guilford Press.

Mansueto, C. S., Golomb, R. G., Thomas, A. M., & Stemberger, R. M. (1999). A comprehensive model for behavioral treatment of trichotillomania. *Cognitive and Behavioral Practice*, *6*, 23–43.

Mansueto, C. S., Stemberger, R. M., Thomas, A. M., & Golomb, R. G. (1997). Trichotillomania: A comprehensive behavioral model. *Clinical Psychology Review, 17*, 567–577.

Manzoni, G. M., Pagnini, F., Castelnuovo, G., & Molinari, E. (2008). Relaxation training for anxiety: A ten-years' systematic review with meta-analysis. *BMC Psychiatry, 8*, Article 41.

Meunier, S. A., Tolin, D. F., & Franklin, M. (2009). Affective and sensory correlates of hair pulling in pediatric trichotillomania. *Behavior Modification, 33*, 396–407.

Odlaug, B. L., & Grant, J. E. (2007). *N*-acetyl cysteine in the treatment of grooming disorders [Letter to the Editor]. *Journal of Clinical Psychopharmacology, 27*, 227–229.

Öst, L.-G. (1987). Applied relaxation: Description of a coping technique and review of controlled studies. *Behaviour Research and Therapy, 25*, 297–409.

O'Sullivan, R. L., Phillips, K. A., Keuthen, N. J., & Wilhelm, S. (1999). Near-fatal skin picking from delusional body dysmorphic disorder responsive to fluvoxamine. *Psychosomatics, 40*, 79–81.

Paul, G. L. (1967). Strategy of outcome research in psychotherapy. *Journal of Consulting Psychology, 31*, 109–118.

Pukadan, D., Antony, J., Mohandas, E., Cyriac, M., Smith, G., & Elias, A. (2008). Use of escitalopram in psychogenic excoriation [Letter to the Editor]. *The Australian and New Zealand Journal of Psychiatry, 42*, 435–436.

Rosenbaum, M. S., & Ayllon, T. (1981). The behavioral treatment of neurodermatitis through habit-reversal. *Behaviour Research and Therapy, 19*, 313–318.

Sahin, E., Kalyoncu, A., Pektas, Ö., Tan, D., Mirsal, H., & Beyazyürek, M. (2004). Near-fatal skin-picking due to obsessive compulsive disorder responding to combined fluoxetine and cognitive-behavioral therapy: A case report. *Bulletin of Clinical Psychopharmacology, 14*, 88–91.

Sasso, D. A., Kalanithi, P. S. A., Trueblood, K. V., Pittenger, C., Kelmendi, B., Wayslink, S., Malison, R. T., Krystal, J. H., & Coric, V. (2006). Beneficial effects of the glutamate-modulating agent riluzole on disordered eating and pathological skin-picking behaviors [Letter to the Editor]. *Journal of Clinical Psychopharmacology, 26*, 685–687.

Seedat, S., Stein, D. J., & Harvey, B. H. (2001). Inositol in the treatment of trichotillomania and compulsive skin picking [Letter to the Editor]. *Journal of Clinical Psychiatry, 62*, 60–61.

Seitz, P. F. D. (1953). Dynamically-oriented brief psychotherapy: Psychocutaneous excoriation syndromes: An experiment. *Psychosomatic Medicine, 15*, 200–242.

Simeon, D., Stein, D. J., Gross, S., Islam, N., Schmeidler, J., & Hollander, E. (1997). A double-blind trial of fluoxetine in pathologic skin picking. *Journal of Clinical Psychiatry, 58*, 341–347.

Stanley, M. A., Swann, A. C., Bowers, T. C., Davis, M. L., & Taylor, D. J. (1992). A comparison of clinical features in trichotillomania and obsessive-compulsive disorder. *Behaviour Research and Therapy, 30*, 39–44.

Swedo, S. E., Rapoport, J. L., Leonard, H. L., Schapiro, M. B., Rapoport, S. I., & Grady, C. L. (1991). Regional cerebral glucose metabolism of women with trichotillomania. *Archives of General Psychiatry, 48*, 828–833.

Teng, E. J., Woods, D. W., & Twohig, M. P. (2006). Habit reversal as a treatment for chronic skin picking: A pilot investigation. *Behavior Modification, 30*, 411–422.

Teng, E. J., Woods, D. W., Twohig, M. P., & Marcks, B. A. (2002). Body focused repetitive behavior problems: Prevalence in a nonreferred population and differences in perceived somatic activity. *Behavior Modification, 26*, 340–360.

Twohig, M. P, Hayes, S. C., & Masuda, A. (2006). A preliminary investigation of acceptance and commitment therapy as a treatment for chronic skin picking. *Behaviour Research and Therapy, 44*, 1513–1522.

Twohig, M. P., & Woods, D. W. (2001). Habit reversal as a treatment for chronic skin picking in typically developing adult male siblings. *Journal of Applied Behavior Analysis, 34*, 217–220.

Walther, M. R., Flessner, C. A., Conelea, C. A., & Woods, D. W. (2009). The Milwaukee Inventory for the Dimensions of Adult Skin Picking (MIDAS): Initial development and psychometric properties. *Journal of Behavior Therapy and Experimental Psychiatry, 40*, 127–135.

Welkowitz, L. A., Held, J. L., & Held, A. L. (1989). Management of neurotic scratching with behavioral therapy. *Journal of the American Academy of Dermatology, 21*, 802–804.

Wilhelm, S., Keuthen, N. J., Deckersbach, T., Engelhard, I. M., Forker, A. E., Baer, L., O'Sullivan, R. L., & Jenike, M. A. (1999). Self-injurious skin picking: Clinical characteristics and comorbidity. *Journal of Clinical Psychiatry, 60*, 454–459.

Wolpe, J., & Lazarus, A. A. (1966). *Behavior therapy techniques: A guide to the treatment of neuroses*. Elmsford, NY: Pergamon Press.

Further Reading

Arnold, L. M., Auchenbach, M. B., & McElroy, S. L. (2001). Psychogenic excoriation: Clinical features, proposed diagnostic criteria, epidemiology, and approaches to treatment. *CNS Drugs, 15*, 351–359.

Azrin, N. H., & Nunn, R. G. (1973). Habit-reversal: A method of eliminating nervous habits and tics. *Behaviour Research and Therapy, 11*, 619–628.

Deckersbach, T., Wilhelm, S., & Keuthen, N. (2003). Self-injurious skin picking: Clinical characteristics, assessment methods, and treatment modalities. *Brief Treatment and Crisis Intervention, 3*, 249–260.

Grant, J. E., & Odlaug, B. L. (2009). Update on pathological skin picking. *Current Psychiatry Reports, 11*, 283–288.

Grant, J. E., Odlaug, B. L., & Kim, S. W. (2007). Lamotrigine treatment of pathological skin picking: An open-label study. *Journal of Clinical Psychiatry, 68*, 1384–1391.

Simeon, D., Stein, D. J., Gross, S., Islam, N., Schmeidler, J., & Hollander, E. (1997). A double-blind trial of fluoxetine in pathologic skin picking. *Journal of Clinical Psychiatry, 58*, 341–347.

Teng, E. J., Woods, D. W., & Twohig, M. P. (2006). Habit reversal as a treatment for chronic skin picking: A pilot investigation. *Behavior Modification, 30*, 411–422.

Twohig, M. P, Hayes, S. C., & Masuda, A. (2006). A preliminary investigation of acceptance and commitment therapy as a treatment for chronic skin picking. *Behaviour Research and Therapy, 44*, 1513–1522.

CHAPTER

28 Impulsive/Compulsive Sexual Behavior: Assessment and Treatment

Eli Coleman

Abstract

There is a growing recognition among clinicians that any type of sexual behavior can become pathologically impulsive or compulsive. There is quite a bit of debate about terminology for this condition, the diagnostic criteria, assessment methods and treatment approaches. In the absence of clear consensus, clinicians are struggling with how to help the many men and women who suffer and seek help from this type of problem. This chapter will review the author's assessment and treatment approach. Clinicians will need to keep abreast of the literature as new research evolves and follow the continued debate around this controversial area.

Keywords: impulse control, sexual behavior, assessment, treatment

As with many other pleasurable and self- and other-enhancing sexual behaviors, there is growing recognition among clinicians that sexual behavior can become pathologically impulsive or compulsive (Coleman, 1991; Coleman et al., 2003). Normal, healthy sexual behavior is often impulsive, driven, and thought about continuously, and the satisfaction of sexual behavior is often accompanied by a sense of abandonment and loss of control. So, when does the sexual behavior become pathological and in need of treatment? How is this assessed, and what are the treatment approaches? These are vexing questions that are to some degree unanswered and are under intense debate. While the paraphilias have been recognized as clinical disorders, there have been serious challenges to the evidence presented to support their continued classification in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Di*sorders (DSM) and the World Health Organization's *International Classification of Diseases* (ICD; e.g, Moser, 2009). Currently, there is no diagnostic category syndrome in the DSM-IV-TR (APA, 2000) for normophilic behavior that is taken to its impulsive/compulsive extreme. Yet, many men and women suffer from this problem and seek help for it, and clinicians are in need of guidance from the best information available at this time.

A Debate Over Terminology: What Do We Call It?

There has been great debate about the terminology and etiology of this clinical syndrome (Coleman, 2003). It is not newly described in the literature, but it has been described by many different terms: *hypersexuality, hyperphilia, erotomania, satyriasis, promiscuity, Don Juanism, Don Juanitaism,* and, more recently, *sexual addiction, compulsive sexual behavior,* and *paraphilia-related disorder* (e.g., Carnes, 1983; Coleman, 1991; Goodman, 1993; Kafka & Prentky, 1994; Levine, 1982; Money, 1986). The terminology has often implied different values, attitudes, and theoretical orientations, and the debate continues regarding classification, causes, and treatment (Coleman, 1986, 1990, 1991, 1992). In the ICD (Version 10), there is a category of Excessive

Sexual Drive. Examples of this are *nymphomania* and *satyriasis*. Most recently, the committee assigned to recommend revisions to the current DSM has suggested the term and a new category of sexual disorders: *hypersexual disorder* (APA, 2010). There has never been such a category in DSM. However, since the publication of the DSM-III in 1980, the category Sexual Disorder Not Otherwise Specified has included an example of Don Juanism ("distress about a pattern of repeated sexual relationships involving a succession of lovers who are experienced by the individual only as things to be used"; APA, 2000, p. 582)

The rationale for and empirical evidence in support of the proposal of the DSM task force is elaborated in the review by Kafka (2009). He argues that there are three extant pathophysiological models to describe this syndrome: sexual arousal/desire dysregulation, sexual addiction, and sexual compulsivity.

Kafka argues that mental health consumers and providers have a strong clinical need to recognize and diagnose a distinct group of men and women who have been seeking help for this problem in various ways (psychotherapy, 12-step support groups, and pharmacotherapy), yet there is no diagnostic category at present. In the absence of a diagnostic category, these individuals have been diagnosed with Sexual Disorder Not Otherwise Specified (APA, 2010).

Besides the clinical need, Kafka and the DSM workgroup argue that significant research is needed to consolidate an operational definition. "A DSM-V-based empirically derived definition should significantly enhance research efforts to explore some of the additional diagnostic validators for which there are no current data" (APA, 2010).

Kafka and the DSM workgroup argue that the paraphilic disorders are the nearest diagnostic category and that there may be comorbidity with some of these other paraphilias. However, they state that the category Hypersexual Disorder involves normophilic behavior rather than deviant or social anomalous behavior and is distinct in and of itself.

The DSM workgroup acknowledges that much research is needed to understand this disorder but that there is sufficient evidence to move this proposed disorder into field trials and possible inclusion in DSM-V. Clinicians will need to follow this debate, and it is uncertain how this clinical syndrome will be classified in the revisions of the DSM or ICD. There is also the possibility that this syndrome could be classified under the proposed new category of Behavioral Addictions (APA, 2010; Holden, 2010). For the first time, this general category has been suggested. Some say this is a change that is long overdue; others, however, are more cautious (Holden, 2010). So far, only one behavior has been proposed for inclusion: Disordered Gambling. Pathological Gambling was included as a diagnostic category in previous versions of DSM but under Impulse Control Disorders Not Elsewhere Classified. Some argue that problematic sexual behavior should be included as well. This category is proposed to be subsumed under a newly proposed umbrella category of Addiction and Related Disorders (including Alcohol and other drug use disorders). It is unclear what the outcome of this debate will be.

The proposed category of Hypersexual Disorder at least recognizes the various pathological pathways and understands this as a complex psychosexual disorder; however, the category still connotes that the behavior may be "more than" or conveys the notion that the behavior is driven by hypersexual drive dysregulation. The problem with the category of behavioral addiction is that it assumes that it has commonalities in clinical expression, etiology, comorbidity, physiology, and treatment with Substance Use Disorders. As Kafka has pointed out, this is probably only one pathophysiological mechanism or pathway to the disordered behavior (Kafka, 2009). Treating impulse control sexual behavior as an addiction has been criticized for many reason (Coleman, 2003). The issue of nosology will probably not be resolved until at least the revision of DSM to be released in 2013, and I suspect will remain a controversial issue.

In my work and throughout this chapter, I use the term *impulsive/compulsive sexual behavior* (ICSB) to describe this syndrome. I prefer this term at this point because of its descriptive nature, and it leaves open the possibility of multiple pathological pathways and treatments. In previous work, I have used the term *compulsive sexual behavior*; however, I have seen this term as limiting,and it failed to recognize that some individuals have more problems with impulse control rather than with obsessive-compulsive mechanisms (Raymond et al, 2003). Since they could be of either type, I have chosen to add *impulsive* to describe the phenomenon.

Impulsive/compulsive sexual behavior is a putative clinical syndrome characterized by the experience of sexual urges, sexually arousing fantasies, and sexual behaviors that are recurrent, intense, and cause distressful interference in one's daily life

(Coleman, 1987, 1991, 1992; Coleman et al., 2003). Individuals with ICSB often perceive their sexual behavior to be excessive, but they are unable to control it. They act out impulsively (act on impulses and lack impulse control) and/or compulsively (being plagued by intrusive obsessive thoughts and driven behaviors; Carnes, 1991; Coleman, 1991; Coleman et al., 2003; Kafka, 1991). There are several important aspects to this behavior: it can involve fantasies and urges as well as behavior. In addition, the behavior must reach a level of clinically significant distress and interference in one's daily life. More specific criteria will be discussed in the section on assessment.

There are two different types of ICSB: paraphilic and nonparaphilic. In many ways, I share Kafka's view that nonparaphilic ICSB is more similar to the paraphilias. The only main difference is that one involves normative sexual behavior and the other involves socially anomalous behavior or that which is considered deviant.

Paraphilic ICSB

Money (1986) defined paraphilias as "a condition occurring in men and women of being compulsively responsive to and obligatively dependent upon an unusual and personally or socially unacceptable stimulus, perceived or in the imagery of fantasy, for optimal initiation and maintenance of erotosexual arousal and the facilitation or attainment of orgasm" (p. 267). He continued, "In legal terminology, a paraphilia is a perversion or deviancy; and in the vernacular it is kinky or bizarre sex" (p. 267). Thus, paraphilic ICSB involves sexual behaviors that are not only impulsive or compulsive and repetitive, but also unconventional or socially deviant (APA, 2000; Money, 1986, 1988).

The paraphilias are categorized and have distinct diagnostic criteria in the *Diagnostic and Statistical Manual of the American Psychiatric Association* (2000). In the DSM-IV-TR, paraphilias are defined as "recurrent, intense sexually arousing fantasies, sexual urges, or behaviors generally involving (1) nonhuman objects, (2) the suffering or humiliation of oneself or one's partner, or (3) children or other non-consenting persons" (p. 566). The definition goes on to explain that "The behavior, sexual urges, or fantasies cause clinically significant distress in social, occupational, or other important areas of functioning" (p. 566). Paraphilias may involve illegal behavior, such as an adult having sex with a minor. However, not all sexual offenses are committed by people who meet the diagnostic criteria for one who has a paraphilia. That said, most paraphilias do cause interference in intrapsychic and/or interpersonal functioning.

The eight paraphilias listed in the DSM-IV TR are pedophilia, exhibitionism, voyeurism, sexual sadism, sexual masochism, fetishism, transvestic fetishism, and frotteurism (see Table 28.1).

The DSM workgroup on paraphilic disorders has recommended a number of changes to be included in the next revision of the DSM (DSM-V). Most notably, the workgroup has suggested that a distinction needs to be made between a paraphilia and a paraphilic disorder. "A paraphilia by itself would not automatically justify or require psychiatric intervention. A *paraphilic disorder* is a paraphilia that causes distress or impairment to the individual or harm to others. One would *ascertain* a paraphilia (according to the nature of the urges, fantasies, or behaviors) but *diagnose* a paraphilic disorder (on the basis of distress and impairment). In this conception, having a paraphilia would be a necessary but not a sufficient condition for having a paraphilic disorder" (APA, 2010).

Table 28.1 Eight Paraphilias Defined in DSM-IV-TR

Pedophilia	Sexual arousal to an age range restricted to prepubertal or peripubertal children
Exhibitionism	Sexual excitement by evoking emotional response from a stranger by illicitly exhibiting an erotic part of the body
Voyeurism	Sexual arousal through the risk of being discovered while watching a stranger
Sadism	Sexual arousal to being the authority who demands discipline and imposes humiliation, abuse, torture, and punishment
Masochism	Sexual arousal by being the recipient of abuse, torture, punishment, discipline, humiliation, obedience, and servitude
Transvestism	Sexual arousal by wearing clothing of the other sex
Fetishism	Sexual arousal evoked by a particular object, substance, or part of a partner's body
Frotteurism	Sexual arousal through rubbing the genital area against the body of a stranger in a crowd

The DSM workgroup has also suggested a change in the classification of *pedophilia* and has suggested the term *pedohebophilic disorder*. The workgroup feels that a new term would better capture the range of ages to which pedophiles are attracted. One set of specifiers for pedohebephilic disorder would allow the clinician to record whether the patient is most attracted to prepubescent (Tanner 1) children, most attracted to pubescent (Tanner 2–3) children, or equally attracted to pubescent and prepubescent children. The workgroup has also suggested an additional category of Paraphilic Coercive Disorder. There has never been a category for people who are aroused by rape fantasies, which is common among individuals who commit rape.

The DSM workgroup has left the Paraphilia Not Otherwise Specified category for more unusual and less common paraphilias. John Money created a taxonomy of almost 50 distinct types of paraphilias including zoophilia (bestiality), asphyxiophilia (cutting off oxygen to increase or enhance sexual arousal or orgasm), and necrophilia (sex with dead people), to name a few (Money, 1986).

The DSM workgroup is obviously responding to the criticism that the current criteria may be overpathologizing normative sexual variations. The workgroup has attempted to develop clearer diagnostic criteria with more behavioral indicators rather than the more subjective criteria used in DSM-IV-TR. However, in the process, they have removed the subjective term *recurrent,* which could lead to overdiagnosis since the threshold is low—only a few occasions or a small number of victims. These proposed improvements will certainly be met with criticism and will ultimately be tested in field trials. Clinicians will have to follow these developments, as these represent significant potential revisions of classification and criteria.

Nonparaphilic ICSB

In contrast to paraphilic ICSB, nonparaphilic ICSB involves normative and conventional sexual behavior that is engaged in recurrently and intensely, yet with similar negative consequences and distress (Coleman, 1991, 1992; Coleman et al., 2003). It involves normophilic behavior. *Normophilia* is "a condition of being erotosexually in conformity with the standard as dictated by customary, religious, or legal authority" (Money, 1986, p. 266). This type of ICSB can cause considerable interference in pair-bonding relationships (e.g., Coleman, 1991, 1995). There are also high risks associated with sexually transmitted infections (STIs), including HIV infection (Coleman, et al., 2009; Kalichman & Cain, 2004; Kalichman et al., 2005). Besides STIs, unintended pregnancies can result (Henshaw, 1998; McBride et al., 2008). In addition, with the explosion of the Internet, viewing of pornography at home and at the workplace has become a serious problem (Cooper et al., 2002). This is one of the most common types of ISCB that we are seeing clinically today. Also, there are far more cybersex users who use the Internet to seek partners and who are at greater risk for STIs (Coleman et al., 2009; McFarlane et al., 2000).

There is no consensus on the types of nonparaphilic ICSB. There are at least seven subtypes: compulsive cruising and multiple partners, compulsive fixation on an unattainable partner, compulsive autoeroticism (masturbation), compulsive use of erotica, compulsive use of the Internet for sexual purposes, compulsive multiple love relationships, and compulsive sexuality in a relationship (Coleman et al., 2003; see Table 28.2).

Besides advocating for a new category of Hypersexuality Disorder, the DSM workgroup revision committee recommended a set of diagnostic criteria and specifiers for the type of "hypersexual" behavior. In their conceptualization, they have recommended the following specifiers:

Masturbation
Pornography
Sexual Behavior with Consenting Adults
Cybersex
Telephone Sex
Strip Clubs
Other

While this list is similar to the seven types mentioned, the proposed specifiers are behavioral rather than dynamic.

In the past, we have used a slight alteration of the paraphilia diagnostic criteria and this represents an amalgam of criteria we have used (Coleman et al., 2003; (Raymond et al., 2003):

A. Over a period of at least six months, recurrent intense sexually arousing behaviors, sexual urges, or fantasies that involving one or more of the following:

(1) Compulsive cruising and multiple sexual partners

(2) Compulsive fixation on an unattainable partner

(3) Compulsive autoeroticism

(4) Compulsive use of the Internet for sexual purposes

Table 28.2 Types of Nonparaphilic ICSB

Compulsive Cruising and Multiple Partners
- Constantly searching or "scanning" the environment for a potential partner. Relentless search to find, conquer, and satisfy the demand for a sexual outlet. Insatiable demand for multiple partners as part of a strategy for management of anxiety and maintenance of self-esteem
- Cruising as ritualistic and trance-inducing
- Partners as "things to be used"

Compulsive Fixation on an Unattainable Partner
- Compulsive fixation on an unattainable partner despite lack of a reciprocal response
- Elaboration on fantasies without the intrusion of reality
- Fueling the fantasy by the potential and fantasized reciprocation of love
- Idealizing and fictionalizing the love object

Compulsive Autoeroticism
- Obsessive and compulsive drive toward sexual self-stimulation of the genitalia
- Cessation of masturbation caused by exhaustion, injury, or extreme social pressure rather than sexual satisfaction
- Loneliness felt keenly after an orgasm
- Common practice of masturbating 5–15 times a day
- Common experience of physical injury
- Interference with occupational, social, interpersonal, and intimacy functioning

Compulsive Use of Erotica
- Obsessive and compulsive drive to seek sexual stimulation through erotica
- Hiding, hoarding, and/or compulsive collecting of erotic materials
- Spending excessive amounts of money seeking/buying erotica

Compulsive Use of the Internet
- Obsessive and compulsive use of the Internet in seeking sexual gratification
- Compulsive chatting, seeking of fantasized sexual partners
- Spending excessive amounts of time online, causing interference with occupational, social, interpersonal, and intimacy functioning

Compulsive Multiple Love Relationships
- Obsession and compulsion in finding the intense feeling of a new relationship
- Lack of capacity to freely choose multiple love relationships
- Fantasy and role playing essential in relationships; reality is intrusive
- Highly skilled romance artist

Compulsive Sexuality in a Relationship
- Compulsive expressions of sexuality in a relationship
- Demanding sexual expression through manipulation, coercion, or violence
- Absence of expression of sexuality that results in anxiety, depression, and anger
- Unending need for sex, expressions of love, attention, and signs of affection that temporarily relieve anxiety
- Relationships characterized by intense possessiveness, jealousy, and anger

(5) Compulsive use of erotica
(6) Compulsive multiple love relationships
(7) Compulsive sexuality in a relationship

B. The fantasies, sexual urges or behaviors cause clinically significant distress or impairment in social, occupational or other important areas of functioning.

C. Not due to another medical condition, substance use disorder, or attributable to another Axis I or II disorder such as mania or a developmental disorder. Must take into account norms of gender, sexual orientation and sociocultural groups.

D. Duration of at least 6 months.

The APA proposed diagnostic criteria for DSM-V are (APA, 2010):

A. Over a period of at least six months, recurrent and intense sexual fantasies, sexual urges, and sexual behavior in association with four or more of the following five criteria:

(1) Excessive time is consumed by sexual fantasies and urges, and by planning for and engaging in sexual behavior.

(2) Repetitively engaging in these sexual fantasies, urges, and behavior in response to dysphoric mood states (e.g., anxiety, depression, boredom, irritability).

(3) Repetitively engaging in sexual fantasies, urges, and behavior in response to stressful life events.

(4) Repetitive but unsuccessful efforts to control or significantly reduce these sexual fantasies, urges, and behavior.

(5) Repetitively engaging in sexual behavior while disregarding the risk for physical or emotional harm to self or others.

B. There is clinically significant personal distress or impairment in social, occupational or other important areas of functioning associated with the frequency and intensity of these sexual fantasies, urges, and behavior.

C. These sexual fantasies, urges, and behavior are not due to direct physiological effects of exogenous substances (e.g., drugs of abuse or medications) or to Manic Episodes.

D. The person is at least 18 years of age.

As you can see, the DSM workgroup recommends more specific criteria for "intense and recurrent." These more behavioral descriptions are probably much more useful as diagnostic criteria. They must still be considered and field tested. The most important thing that a clinician needs is a clear set of diagnostic criteria or an operational definition of the clinical syndrome. Additional issues about diagnosis are discussed below.

Assessment and Diagnosis

Impulsive/compulsive sexual behavior is unlikely to be a presenting complaint unless the clinician is attached to a specific sexual disorder clinic. Clinicians are generally uncomfortable talking about sex with their patients due to lack of training. It is more likely that the presenting complaint is an STI, an unwanted pregnancy, or marital or relationship problems. Other patients may present with anxiety, depression, alcohol or other drug abuse/dependency, or somatic complaints (Coleman, 1992; Coleman et al., 2003). There is a high comorbitity of ICSB with psychiatric disorders, particularly anxiety disorders, depression, and substance abuse disorders (Black et al., 1997; Kafka & Prentky, 1996; Raymond et al., 2003). Therefore, when patients present with these types of problems, the clinician should suspect that sexual behavior might be associated with them—as a coping mechanism, as a distressing outcome, or simply as a comorbid state. It is important to rule out such things as bipolar disorder or to differentiate between classic obsessive-compulsive disorder and this syndrome.

Men are more likely to suffer from ICSB than women. While there are no good epidemiological data on this disorder, we estimate that 5% of the population may be affected. When women are involved, they are more likely to suffer from non-paraphilic ICSB (Coleman et al., 2003).

When ICSB is suspected, an initial set of screening questions can be helpful. The following screening questions are recommended:

1. Do you, or others who know you, find that you are overly preoccupied or obsessed with sexual activity?
2. Do you find yourself compelled to engage in sexual activity in response to stress, anxiety, or depression?
3. Have serious problems developed as a result of your sexual behavior (e.g., loss of a job or relationship, sexually transmitted infections, injuries or illnesses, or sexual offenses)?
4. Do you feel guilty or shameful about some of your sexual behaviors?
5. Do you fantasize or engage in any unusual or what some would consider "deviant" sexual behavior?
6. Do you find yourself constantly searching or "scanning" the environment for a potential sexual partner?
7. Do you ever find yourself sexually obsessed with someone who is not interested in you or who does not even know you?
8. Do you think your pattern of masturbation is excessive, driven, or dangerous?
9. Do you find yourself compulsively searching for erotica for sexual stimulation?
10. Do you find yourself spending excessive amounts of time on the Internet engaging in various sexual pursuits?
11. Have you had numerous love relationships that are short-lived, intense, and unfulfilling?
12. Do you feel a constant need for sex or expressions of love in your sexual relationship?

Determining the Diagnosis

The screening questions can be helpful in determining if a problem exists. While many people have developed problematic sexual behaviors, this does not mean that they have met the threshold for ICSB.

Standardized Instruments

There are no clinically validated instruments to measure ICSB. Using standardized instruments to evaluate

for comorbid psychiatric conditions is certainly advisable. It is important to look for both Axis I and Axis II disorders, as well as to rule out any contributions from medical conditions. Some scales are under development, such as the Sexual Addiction Screening Test (Carnes, 1991; Nelson & Oehlert, 2008), the Compulsive Sexual Behavior Inventory (Coleman et al., 2001; Miner et al., 2007), the Hypersexual Scale (Reid & Garos, 2007), and the Sexual Compulsive Scale (Kalichman & Rompa, 1995; 2001; the Sexual Inhibition (SIS) and Sexual Excitation (SES) Scales (Janssen et al., 2002a, 2002b). The Compulsive Sexual Behavior Inventory has been translated into Spanish and has been found to be reliable (Coleman & Swinburne Romine, 2010a). In the absence of a fully developed and validated scale, these instruments should be used cautiously. It is helpful to have a number of nonstandardized instruments in asking screening questions, as some patients are sometimes more likely to report things in writing and others in a clinical interview (especially concerning sensitive and taboo subjects). A combined approach leads to a better assessment.

The Danger of Overpathologizing This Disorder

The overpathologizing of sexual behavior (including ICSB) can occur by failing to recognize the wide range of normal human sexual expression—not only in frequency but also in variety (Coleman, 1992, 2010). It can also occur among clinicians who have overly conservative attitudes and values regarding human sexual expression. It is important for professionals to be comfortable with a wide range of normal sexual behavior—both in type and in frequency. Another problem can be caused by lack of knowledge and training. Many clinicians lack appropriate training in human sexuality. As in evaluating any condition outside of one's area of expertise, it is good practice to seek consultation from a specialist in treating sexual disorders.

Sometimes individuals, with their own restrictive values, will diagnose themselves with ICSB, thus creating their own distress. Therefore, it is very important to distinguish between an individual whose values conflict with his or her sexual behavior and one who engages in sexual behaviors that are driven by impulsive, obsessive, and/or compulsive mechanisms.

A Conflict Over Values

There is an inherent danger in diagnosing ICSB simply because the individual's behavior does not fit the values of the individual, group, or society. There has been a long tradition of pathologizing behavior that is not normative within a culture. For example, masturbation, oral sex, homosexual behavior, sadomasochistic behavior, or an extrarelational affair could be viewed as compulsive behavior because someone might disapprove of this behavior. There is no scientific merit in viewing these behaviors as disordered, compulsive, or deviant. When individuals are distressed about these behaviors, they are most likely in conflict with their own or someone else's value system rather than with compulsive sexual behavior (Coleman, 1992, 2010b).

Problematic versus Impulsive-Compulsive Sexual Behavior

Behaviors that are in conflict with someone's value system may be problematic but not impulsive, obsessive, or compulsive. Having sexual problems is common. Problems are often caused by a number of nonpathological factors. People may make mistakes; they may be ignorant. They may, at times, act impulsively. Their behavior may cause problems in a relationship. Some people use sex as a coping mechanism similar to the use of alcohol, drugs, or eating. This pattern of sexual behavior may become problematic. Problematic sexual behavior is often remedied, however, by time, experience, education, or brief counseling. Impulsive, obsessive, and compulsive behavior, by its nature, is much more resistant to change (Coleman, 1992, 2010b)

I have found that it is very helpful to view sexual behaviors on a continuum. At one end of the continuum is healthy sexuality; at the other end is the clinical syndrome of ICSB (see Figure 28.1)

It is very important for the clinician to distinguish between problematic sexual behavior and that which may be a function of ICSB. However, this is not easily done, as there are no clear, universally accepted criteria for knowing when a patient has crossed the line between problematic behavior and the clinical syndrome. Only a trained clinician specializing in assessing and treating these individuals would be able to determine the difference. And, in many cases, it is difficult to make that assessment initially. Often in the process of addressing the behavior, it becomes clear that more serious underlying pathological mechanisms are at work (Coleman, 1992, 2010b)

Fig. 28.1 Continuum of ICSB.

Developmental Process versus Compulsive Sexual Behavior

Some sexual behaviors might be viewed as impulsive, obsessive, or compulsive if they are not considered within their developmental context. Adolescents, for example, can become obsessed with sex for long periods of time. They can act impulsively. In adulthood, it is common for individuals to go through periods in which sexual behavior may take on impulsive, obsessive, and compulsive characteristics. In the early stages of romance, there is a natural developmental period in which individuals might be obsessed with their partners and compelled to seek out their company and to express affection. These are normal, healthy developmental processes of sexual development and must be distinguished from ICSB (Coleman, 1992, 2010b)

Summary

It is important to distinguish between the clinical syndrome of ICSB and behaviors that might be better explained by conflicts in intrapersonal or interpersonal values or as a developmental process. For example, a young single man who thinks masturbation is a sin might consider his behavior obsessive or compulsive. A wife who finds that her husband has viewed some erotica might see this as an addiction.

When ICSB is suspected, the health care professional is advised to refer to a specialist in this area for further assessment and possible treatment. One source of referral is the American Association of Sex Educators, Counselors and Therapists (http://www.aasect.org) or the Society for Sex Therapy and Research (http://www.sstarnet.org/). There are professional organizations in many countries with standards for training and experience that determine acceptance for membership or certification.

Treatment

Treatment of ICSB usually involves a combination of psycho- and pharmacotherapies (Coleman, 1995; Coleman et al., 2003).

Brief Treatment and Psychoeducation

Many behaviors can be described as problematic or subclinical. Often these behaviors can be readily addressed through brief psychoeducation or solution-focused therapy. Sometimes people simply need to understand the patterns of their behavior, and its negative consequences, and be more motivated to engage in sexually healthier behaviors. In cases where individuals feel that normative sexual behavior is somehow pathological, the clinician can educate them about the normative range of sexual expression. Violating one's value system is normative, and individuals' mistakes can be used to guide them back to their valued path in life. Errors in judgment can be recognized as such and reaffirm good decision making. Disagreements in value systems between couples are common. These disagreements can be acknowledged and the couple can work to find compromise solutions for accommodating their disparate value systems, as is done with value conflicts in raising children, dealing with family members, or managing finances.

Psychological Treatment

In serious cases, it is more likely that a course of psychotherapy will be needed, often with adjunct pharmacotherapy (Coleman, 1995; Coleman et al., 2003). In ICSB, the patient is suffering from a psychosexual disorder. This is something that is deep-rooted and stems from a failure of healthy psychosexual development. Many of these patients have grown up in dysfunctional family environments and have serious underlying identity and intimacy problems. Many of their needs for nurturance, love, acceptance, and positive role models have not been met. As a result, there has often been a disruption in their psychosexual development that interferes with their ability to feel good about themselves as sexual beings and/or their ability to be intimate with another human being (Coleman, 1987). It is very helpful to conceptualize their problem as an identity disorder, an intimacy disorder, or an attachment disorder.

Therapeutic Modalities

A preferred modality is group therapy combined with adjunct individual and family therapy. Weekly group therapy becomes a laboratory for examining underlying mechanisms, learning from others' coping strategies, and learning new intimacy skills. It is a vehicle to explore the underlying dynamics and see them played out in the interactions with other group members. Group therapy reduces the intense feeling of shame that these individuals experience due to their aberrant sexual histories and their generalized feeling of shame about their basic identity. It also motivates patients to complete the rigorous treatment. In an ongoing group, new members are able to learn from other patients who have completed therapeutic tasks and are better able to understand the whole course of therapy. Group therapy is an opportunity to provide peer support

as patients start to build outside support systems for better coping. They are all able to celebrate when one member has completed the course and is ready to "graduate."

Individual therapy allows the therapist to address more individualized concerns, strengthen the therapeutic alliance, examine issues in more depth, and guide the patient on how to use the other modes of treatment more effectively (e.g, group or family therapy). It allows for ongoing treatment planning and determining therapeutic tasks at different developmental stages of the therapeutic process.

Family therapy is designed to address family-of-origin issues and conflicts in current interpersonal functioning. This could involve a partner or spouse, parents, siblings, or other significant persons in the patient's life.

Gaining Control Over Sexual Behavior

The first step in the therapy process is to work toward gaining some control over the problematic sexual behaviors. By the nature of the problem, it is difficult to gain control over all problematic behaviors at once. It is naturally helpful to gain control over the most problematic behaviors (e.g., engaging in sexual behavior with a high likelihood of developing STIs, including HIV, viewing erotica on work computers where work policies might result in loss of a job, engaging in dangerous sexual activities like autoerotic asphyxiation, or engaging in sexual behaviors that might lead to arrest and incarceration). It is helpful for for therapists to understand that developing healthy sexual expression is often a long process. Setting unrealistic goals at the beginning of therapy can set the patient up for failure and discouragement. Therefore, it is helpful to define the boundaries with the patient and to ensure that the boundaries are not overly restrictive. Patients tend to want to restrict their sexual behavior to a narrowly defined script, since they are driven by an intense and immediate desire to be rid of all unhealthy patterns. Some of this desire is driven by a common etiological factors—overly restrictive sexual attitudes about sexual expression.

It is best to have the sexual boundaries set by the patient. Therefore, it is helpful for the clinician to ask the patient to suggest these boundaries. The clinician may have to challenge overly restrictive (or sometimes overly liberal) boundaries set by the patient. In the process of negotiation, the sexual boundaries are clarified in behavioral terms. The patient can then set goals for staying within those boundaries, and can report sexual behaviors that have been both within the boundaries and outside of them. Violations are then monitored; the goal is to help the patient stay within the boundaries. This serves a number of functions:

1. It protects the patient from experiencing continuing serious harm as a result of the ICSB.
2. The patient develops a sense of competency by meeting his or her own goals.
3. Underlying mechanisms are more clearly identified. By ending the problematic behaviors, the patient is left without one of the main coping mechanisms for underlying etiological dynamics. Anxiety, depression, self-esteem, loneliness, and intimacy problems will become more evident. The risk is that these problems will exacerbate to a level that fuels the ICSB further. For this reason, the boundaries should not be too restrictive at this point and may need to be revised.
4. As staying within these boundaries is difficult, the patient becomes more motivated to address underlying mechanisms rather than simply thinking that he or she can gain control over the behavior by sheer will and determination.
5. Unless he or she stays within the boundaries, the patient simply remains within the cycle of ICSB and masks the underlying mechanisms that must be uncovered and addressed in therapy in order to work on long-term change and the development of healthy sexuality.

Pharmacotherapy as an Adjunct to Psychotherapy

Pharmacological agents can be extremely helpful in initially interrupting the vicious cycle of ICSB. They can be useful in assisting in the initial step of the therapeutic process: gaining control over the most serious ICSBs. Pharmacological therapy alone can be helpful to some patients when the problematic behavior is simply a function of neurotransmitter dysregulation that, when treated, allows the patient to establish healthy sexual behaviors (Coleman, 2005).

Many patients resist the idea of using medication. It is helpful to explain the nature of neurotransmitter dysregulation and its relationship to ICSB, and to familiarize the patient with the literature that has shown this therapy to be effective. One must be cautious and explain that there have been very few randomized clinical trials and that clinicians depend mostly on the case report literature. None of the medications now in use are approved by the Food and Drug Administration for ICSB;

this is "off-label" use. In addition, these medications may not be very helpful to patients with underlying personality disorders that are contributing to their ICSB. Obviously, the patient must consent to treatment.

Besides assisting in the initial stage of gaining control over some ICSBs, pharmacotherapy can assist in the entire process of therapy:

1. Because the mechanisms of ICSB often involve neurotransmitter dysregulation related to impulse control, anxiety, depression, and/or pleasure seeking, pharmacotherapy can speed up the process of therapy or improve its effectiveness.
2. Pharmacotherapy can assist in helping the patient through the rigors of the psychotherapeutic process. In psychotherapy, clinicians often ask patients with ICSB to recall their childhood abusive experiences (whether emotional, physical, or sexual). This reexperiencing of the trauma initially evokes a surprisingly calm or detached response. This is often followed by increased anxiety, which does not seem to be triggered by a particular event. Alternatively, an emotional response to a particular event (e.g., rejection) is felt far more acutely than the situation seemingly warrants. Medications can help modulate the emotional response sufficient for the patient to experience appropriate emotions and assist in resolving these issues (Coleman, 1995, 2005).
3. Learning new mechanisms for managing stress and improving coping skills is an essential part of the psychotherapeutic process. However, the lack of such mechanisms may not completely involve learned behaviors; some mechanisms are underlying maladaptive stress responses. Medications can help mediate these maladaptive stress responses while the patient is learning new behavioral strategies for managing stress and improving coping skills.

Resolving Family-of-Origin Conflicts

As ICSB is often a function of an underlying disturbance in identity, intimacy, and attachment functioning, it is helpful for the patient to understand the source of the identity and intimacy problems and work to resolve them. As a beginning step in the therapeutic process, patients are asked to begin writing their sexual autobiography (including their early experiences not only with sexual activity but also with intimate relationships). Through this process, patients become more aware of the disruptions in their psychosexual development. These conflicts are often played out in their dysfunctional relationships and sexual behavior; however, patients often do not recognize these patterns. For some patients, resolution of conflicts may be as simple as acknowledging and grieving over these losses. For others, it is helpful to review these conflicts with their family members to gain further understanding and a shared feeling of loss. The best resolution of family-of-origin conflicts is obtained when family relationships are improved in terms of intimacy functioning (Coleman, 1995). This is accomplished through family therapy. The next step is for patients to clearly see how these dynamics are played out in their cycle of ICSB, and the nature and dynamics of their intimate relationships (or lack thereof). Different patterns are learned, experimented with, and consolidated in the therapeutic process.

Understanding Triggers and Cycles of ICSB

After reviewing the patient's sexual autobiography, learned patterns of sexual behavior and, in particular, ICSB are identified. The patients gain an understanding of the events, situations, and/or emotions that often trigger their ICSB. They are encouraged to identify their *cycle* of behavior, which describes the chain or sequence of events, the dynamics that lead to their ICSB, and those that perpetuate it. Each patient has a unique cycle. Once they understand their cycle, patients are better prepared to employ tactics to interrupt it and learn healthy intimacy and sexual functioning (Coleman, 1995, 2010b) This prepares them to develop their maintenance plan. Relapse prevention strategies are employed.

Learning New Coping Mechanisms and Breaking the Cycle

Once the cycle is understood, the patient is able to learn the most appropriate mechanisms for breaking it rather than letting the cycle perpetuate itself. This can involve strategies such as stress management, anger management, communication skills, relaxation, self-nurturing, developing and utilizing a support system, and/or changing cognitions. Therapy offers patients psychological tools to better manage their stress, anxiety, and depression, which can trigger their ICSB (Coleman, 1995, 2010b).

Learning New Intimacy Skills and Healthy Sexual Functioning

Once ICSB is under control, the patient is ready to learn new intimacy skills and develop healthy sexual functioning. This may involve major shifts in the ways in which patients interact with other people,

are intimate with their partner, or approach sexual activity with themselves or with a partner. These are learned skills. Patients need guidance from the therapist on ways of improving their relationship skills and approaching sexual activity. The basic principles of sex therapy can be very useful in this stage of the therapeutic process.

Intimate relationships are often destroyed or severely damaged as a result of a patient's ICSB. Repairing a relationship is quite a task. Partners often have major issues of trust and betrayal. They need time to grieve, to be angry, and to be able to forgive before reengaging in an intimate relationship. Both the patient and the partner need to develop new intimacy skills through honest and respectful communication, empathy, and commitment to work on the relationship. Partners often have their own issues that need to be worked through independently. Repairing a broken relationship comes at a later stage of the therapeutic process when the ICSB is under control, new patterns of relating can be learned, and trust restored (Coleman, 1995, 2010b).

Aftercare

We have found that many of our patients benefit from ongoing psychotherapeutic support through a much less intensive but ongoing therapy. The goal of this aftercare is to consolidate the gains obtained in therapy, to immediately address issues of potential relapse, and to foster the idea of ongoing sexual development. Patients who have completed an intensive course of treatment and have achieved the aforementioned goals enter an aftercare group composed of other patients who have also achieved the goals of therapy. This group offers ongoing support and helpful advice, since they understand the nature of the problem and have been successful in overcoming it. For patients who are unable to attend an aftercare group on an ongoing basis, the exact form of aftercare can be tailored to the individual situation. Offering individual psychotherapy periodically can be sufficient for many patients. All patients are encouraged to develop their own support system outside of therapy, and for some patients, this is sufficient aftercare support. The bottom line is that most patients need some type of ongoing support to maintain their progress and to grow further as sexual beings.

Summary

Psychological treatment for ICSB involves a complex set of therapeutic modalities and treatment regimens. Group therapy is a preferred main modality of treatment combined with adjunct individual and family psychotherapy. Because ICSB is a deep-seated psychosexual disorder, a long process is needed not only to gain control over it but also to consolidate long-term gains and prevent relapse. The ultimate goal is not just to gain control over dysfunctional sexual behaviors but also to assist individuals to find healthy and pleasurable means of sexual expression and intimacy functioning.

Pharmacological Treatment

A number of pharmacological agents have been shown to provide helpful adjunctive therapy for ICSB. The most frequently utilized agents are the selective serotonin reuptake inhibitors (SSRIs; Bradford, 2000; Coleman, 2005, 2010b). These medications have been shown to be quite effective in treating depression and anxiety and in helping to control ICSB. They can be used to address underlying mechanisms of anxiety and depression and also to promote impulse control. They also have the side effect of decreasing the libido. The value of the SSRIs is that they seem to address the three main pathophysiological mechanisms outlined by Kafka (2009). A number of open-label studies have indicated that the SSRIs may be efficacious in the treatment of ICSB (Bradford, 2000; Coleman et al., 1992; Federoff, 1993; Kafka, 1991, 1994; Kafka & Prentky, 1992; Stein et al., 1992). These medications appear to improve mood, reduce anxiety, interrupt obsessive thinking, and help patients control urges to engage in ICSB. The older tricyclic medications and the newer atypical antidepressants can still be used; for certain patients, these may be more effective than the SSRIs (e.g., Coleman et al., 2000).

In more resistant cases, antiandrogens can be used (Bradford, 2000; Raymond et al., 2002b). These medications often do not address comorbid psychiatric disorders (especially anxiety and depressive disorders (Raymond et al., 1999), but they control libido and help moderate sexual urges. These drugs have much more severe side effects to be considered, but they remain potentially useful pharmacological agents (Bradford, 2000).

Mood stabilizers can also be used. These medications have been used in the past and remain a potential effective treatment modality (Cesnik & Coleman, 1989; Coleman et al., 2002). In patients for whom the SSRIs do not provide sufficient control for their ICSB, clinicians have augmented SSRI therapy with different types of mood stabilizers such

as lithium carbonate, carbamazapine, and valproic acid (Coleman, 2005, 2010b).

Finally, we have been encouraged by the results produced by naltrexone used with patients with ICSB. Naltrexone is an opioid antagonist that acts on opioid receptors. It effectively blocks these receptors, preventing the body from making use of opiates and endorphins. Opoid antagonists were first used to treat opium addicts; however, more recently, they have been shown to be effective in treating alcoholics, compulsive gamblers, kleptomaniacs, and smokers. Naltrexone not only seems to block the sensation and pleasure-seeking mechanisms but is also thought to have an effect on the impulse control centers of the brain as well. We reported on the first successful treatment of two individuals with ICSB with naltrexone. Both of them had severe symptoms of ICSB and had shown only mild improvement with SSRIs and other antidepressants. When naltrexone was added to the SSRI treatment, the patients lost the urge to engage in their prior problematic behavior and were able to stop their ICSB (Raymond et al., 2002a). We recently completed a case review with similar positive results (Raymond et al., 2010) and now are embarking on a double-blind, placebo-controlled study. Naltexoine can be used to augment SSRI therapy, and sometimes it can be useful on its own. These preliminary results are encouraging and, in our clinical experience, have added one more tool to our armamentarium of pharmacological treatment choices.

Summary of Pharmacological Treatment

Pharmacological treatment has been shown to be an effective adjunct to the psychological treatment of ICSB. Sometimes a single SSRI can be helpful; alternatively, other medications or the use of medications to augment the SSRI are efficacious. It takes a trained clinician who is familiar with these medications and the literature to effectively utilize these medications in treating patients with ICSB. We need controlled clinical trials in order to develop a more evidence-based clinical approach to the pharmacological treatment of ICSB. However, there is at least some evidence that a number of helpful treatment options are available (Bradford, 2000; Coleman, 2005, 2010b). Clinicians should keep abreast of the literature for future developments, as this is still an emerging field of study.

Conclusion

Impulsive/compulsive sexual behavior is a serious clinical disorder that deserves attention from health care professionals. It can be easily overlooked and yet can lead to serious distress and negative consequences. The difficulty of identifying this problem is compounded by the fact that we do not have a consensus on what to call this syndrome, the diagnostic criteria, or the methods to treat it. We rely on a case report literature for guidance in the absence of clinical trials of psychological or pharmacological treatment. Careful assessment and combined multimodal and multidisciplinary treatment can offer assistance to many individuals suffering from ICSB. Specialists are needed in assessment and treatment.

There is much to be learned about this syndrome. Clinicians will need to keep up with the emerging literature in order to provide the best evidence-based care. In the meantime, it is encouraging that we have found effective treatments that can offer hope for improved sexual and intimate lives for patients who suffer from ICSB.

References

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., Text Revision). Washington, DC: Author.

American Psychiatric Association. (2010). *DSM-V development*. Retrieved March 28, 2010, from http://www.dsm5.org

Black, D. W., Kehrberg, L. L. D., Flumerfelt, D. L., & Schlasser, S. S. (1997). Characteristics of 36 subjects reporting compulsive sexual behavior. *American Journal of Psychiatry, 154*, 243–249.

Bradford, J. (2000). Treatment of sexual deviation using a pharmacologic approach. *Journal of Sex Research, 37*(3), 248–257.

Carnes, P. (1983). *Out of the shadows: Understanding sexual addiction.* Minneapolis: CompCare Publishers.

Carnes, P. (1991). Sexual addiction screening test. *Tennessee Nurse*, *54*, 29.

Cesnik, J. A., & Coleman, E. (1989). Use of lithium carbonate in the treatment of autoerotic asphyxia. *American Journal of Psychotherapy, 43*(2), 277–286.

Coleman, E. (1986). Sexual compulsion vs. sexual addiction: The debate continues. *SIECUS Report* July, 7–11.

Coleman, E. (1987). Sexual compulsivity: Definition, etiology, and treatment considerations. *Journal of Chemical Dependency Treatment, 1*, 189–204.

Coleman, E. (1990). The obsessive-compulsive model for describing compulsive sexual behavior. *American Journal of Preventative Psychiatry and Neurology, 2*(3), 9–14.

Coleman, E. (1991). Compulsive sexual behavior: New concepts and treatments. *Journal of Psychology and Human Sexuality, 4*, 37–52.

Coleman, E. (1992). Is your patient suffering from compulsive sexual behavior? *Psychiatric Annals, 22*, 320–325.

Coleman, E. (1995). Treatment of compulsive sexual behavior. In R. C. Rosen & S. R. Leiblum (Eds.), *Case studies in sex therapy* (pp. 333–349). New York: Guilford Press.

Coleman, E. (2003). Compulsive sexual behavior: What to call it, how to treat it? *SIECUS Report, 31*(5), 12–16.

Coleman, E. (2005). Neuroanatomical and neurotransmitter dysfunction and compulsive sexual behavior. In J. S. Hyde (Ed.), *Biological substrates of human sexuality* (pp. 147–169). Washington, DC: American Psychological Association.

Coleman, E. & Swinburne Romine, R. E. (2010a). The Compulsive Sexual Behavior Inventory. In T. D. Fisher, C. M. Davis, W. L. Yarber, & S. L. Davis. (Eds.), *Handbook of sexuality-related measures* (3rd ed., pp. 144–146). New York: Routledge.

Coleman, E. (2010b). *What sexual scientists know about . . . compulsive sexual behavior.* Allentown, PA: Society for the Scientific Study of Sexuality. Retreived June 20, 2010, from http://www.sexscience.org/uploads/media/SSSS_compulsive2.pdf

Coleman, E., Cesnik, J., Moore, A., & Dwyer, S. M. (1992). An exploratory study of the role of psychotropic medications in the treatment of sex offenders. *Journal of Offender Rehabilitation, 18* (3/4), 75–88.

Coleman, E., Gratzer, T., Nescvacil, L., & Raymond, N. (2000). Nefazodone and the treatment of nonparaphilic compulsive sexual behavior: A retrospective study. *Journal of Clinical Psychiatry, 61*, 282–284.

Coleman, E., Horvath, K., Miner, M., Ross, M. W., Oakes, M., Rosser, B. R. S., & Men's INTernet Sex (MINTS-II) Team. (2009). Compulsive sexual behavior and risk for unsafe sex among Internet using men who have sex with men. *Archives of Sexual Behavior, 39*, 1045–1053.

Coleman, E., Miner, M., Ohlerking, F., & Raymond, N. (2001). Compulsive sexual behavior inventory: A preliminary study of reliability and validity. *Journal of Sex & Marital Therapy, 27*, 325–332.

Coleman, E., Raymond, N., & McBean, A. (2003). Assessment and treatment of compulsive sexual behavior. *Minnesota Medicine, 86*(7), 42–47.

Cooper, A., Golden, G. H., & Kent-Ferraro, J. (2002). Online sexual behaviors in the workplace: How can Human Resource departments and Employee Assistance programs respond effectively? *Sexual Addiction & Compulsivity, 9*, 149–165.

Federoff, J. P. (1993). Serotonergic drug treatment of deviant sexual interests. *Annals of Sex Research, 6*, 105–121.

Goodman, A. (1993). Assessment and treatment of sexual addiction. *Journal of Sex & Marital Therapy, 19*(3), 225–251.

Henshaw, S. K. (1998). Unintended pregnancy in the United States. *Family Planning Perspectives, 30*, 24–29.

Holden, C. (2010). Behavioral addictions debut in proposed DSM-V. *Science, 327*, 935.

Janssen, E., Vorst, H., Finn, P., & Bancroft, J. (2002a). The Sexual Inhibition (SIS) and Sexual Excitation (SES) Scales: I. Measuring sexual inhibition and excitation proneness in men. *Journal of Sex Research, 39*, 114–126.

Janssen, E., Vorst, H., Finn, P., & Bancroft, J. (2002b). The Sexual Inhibition (SIS) and Sexual Excitation (SES) Scales: II. Predicting psychophysiological response patterns. *Journal of Sex Research, 39*, 127–132.

Kafka, M. P. (1991). Successful antidepressant treatment of nonparaphilic sexual addictions and paraphilias in men. *Journal of Clinical Psychiatry, 52*(2), 60–65.

Kafka M. P. (1994). Paraphilia-related disorders: Common, neglected, and misunderstood. *Harvard Revew of Psychiatry, 2*, 39–40.

Kafka, M. P. (2009). Hypersexual disorder: A proposed diagnosis for DSM-V. *Archives of Sexual Behavior*, *39*, 377–400.

Kafka, M. P., & Prentky, R. (1992). Fluoxetine treatment of nonparaphilic sexual addictions and paraphilias in men. *Journal of Clinical Psychiatry, 53*, 351–358.

Kafka, M. P., & Prentky, R. (1994). Preliminary observations of DSM-III-R Axis I comorbidity in men with paraphilias and paraphilia-related disorders. *Journal of Clinical Psychiatry, 55*, 481–487.

Kalichman, S. C., & Cain, D. (2004). The relationship between indicators of sexual compulsivity and high risk sexual practices among men and women receiving services from a sexually transmitted infection clinic. *Journal of Sex Research, 41*, 235–241.

Kalichman, S. C., Cherry, C., Cain, D., Pope, H., & Kalichman, M. (2005). Psychosocial and behavioral correlates of seeking sex partners on the Internet among HIV-positive men. *Annals of Behavioral Medicine*, 30, 243–250.

Kalichman, S. C., & Rompa, D. (1995). Sexual sensation seeking and sexual compulsivity scales: Reliability, validity, and predicting HIV risk behavior. Journal of Personality Assessment, 65, 586–601.

Kalichman, S. C., & Rompa, D. (2001). The Sexual Compulsivity Scale: Further development and use with HIV-positive persons. *Journal of Personality Assessment, 76*, 379–395.

Levine, S. B. (1982). A modern perspective on nymphomania. Journal of Sex and Marital Therapy, 8, 316–324.

McBride, K. R., Reece, M., & Sanders, S. (2008). Using the Sexual Compulsivity Scale to predict outcomes of sexual behavior in young adults. *Sexual Addiction & Compulsivity, 15*, 97–115.

McFarlane, M., Sheana, S., & Rietmeijer, C. (2000). The Internet as a newly emerging risk environment for sexually transmitted diseases. *Journal of the American Medical Association, 284,* 443–446.

Miner, M., Coleman, E., Center, B., Ross, M. W., & Rosser, B. R. S. (2007). The Compulsive Sexual Behavior Inventory: Psychometric properties. *Archives of Sexual Behavior, 36,* 579–587.

Money, J. (1986). *Lovemaps: Clinical concepts of sexual/erotic health and pathology, paraphilia, and gender transposition in childhood, adolescence, and maturity*. New York: Irvington.

Money, J. (1988). *Gay, straight, and in-between: The sexology of erotic orientation*. New York: Oxford University Press.

Moser, C. (2009). When is an unusual sexual interest a mental disorder? *Archives of Sexual Behavior, 38,* 323–325.

Nelson, K. G., & Oehlert, M. E. (2008). Psychometric exploration of the Sexual Addiction Screening Test in veterans. *Sexual Addiction & Compulsivity, 15*, 39–58.

Raymond, N. C., Coleman, E., & Miner, M. H. (2003). Psychiatric comorbidity and compulsive/impulsive traits in compulsive sexual behavior. *Comprehensive Psychiatry, 44*, 370–380.

Raymond, N. C., Coleman, E., Ohlerking, F., Christenson, G. A., & Miner, M. (1999). Psychiatric comorbidity in pedophilic sex offenders. *American Journal of Psychiatry, 156*(5), 786–788.

Raymond, N. C., Grant, E., & Coleman, E. (2010). Augmentation with naltrexone to treat compulsive sexual behavior: A case series. *Annals of Clinical Psychiatry*, 22, 56–62.

Raymond, N. C., Grant, J. E., Kim, S. M., & Coleman E. (2002a). Treatment of compulsive sexual behavior with naltrexone and serotonin reuptake inhibitors: Two case studies. *International Clinical Psychopharmacology, 17*(4), 201–205.

Raymond, N. C., Robinson, B. E., Kraft, C., Rittberg, B., & Coleman, E. (2002b). Treatment of pedophilia with leuprolide acetate: A case study. *Journal of Psychology and Human Sexuality, 13* (3/4), 79–88.

Reid, R., & Garos, S. (2007, August). *A new measure of hypersexual behavior*. Presented at the meeting of the American Psychological Association, San Francisco.

Stein, D. J., Hollander, E., Anthony, D. T., Schneier, F. R., Fallon, B. A., Liebowitz, M. R., & Klein, D. F. (1992). Serotonergic medications for sexual obsessions, sexual addictions and paraphilias. *Journal of Clinical Psychiatry, 53*(8), 267–271.

CHAPTER

29 Assessment and Treatment of Problem Internet Use

Kimberly Young

Abstract

Research over the last decade has identified Internet addiction as a new and often unrecognized clinical disorder that impacts a user's ability to control online use to the extent that it can cause relational, occupational, and social problems. Symptoms of problem Internet use are compared to the criteria used to diagnose other addictions. In particular, pathological gambling is compared to problematic Internet use because of overlapping criteria. As new cases of problem Internet use are being documented, this chapter describes the diagnostic and treatment implications of the disorder. As computers are used with great frequency, detection and diagnosis of Internet addiction are often difficult. Symptoms of a possible problem may be masked by legitimate use of the Internet, and clinicians may not ask questions about computer use because problem Internet use is still a new and often unrecognized condition. This chapter outlines diagnostic conceptualizations of problem Internet use including the DSM-V proposed criteria for Pathological Computer Use, assessment techniques, and treatment considerations.

Keywords: Internet addiction, problem Internet use, pathological computer use, assessment, diagnosis, treatment

Introduction

The Internet is a new technology that has impacted the world and provided many benefits to its users. At the same time, the Internet has had negative ramifications. Some people are becoming preoccupied with the Internet, are unable to control their use, and are jeopardizing theiremployment and relationships. The concept of *Internet addiction* has been proposed as an explanation for uncontrollable, damaging use of this technology. Symptoms are compared to the criteria used to diagnose other addictions, and the literature has characterized Internet addiction as an impulse control disorder comparable to pathological gambling because of overlapping criteria.

Much of this research lacks the empirical robustness of experimental design, relying more upon survey data and self-reported data from self-selected populations. The research also lacks proper use of control groups and, in some cases, utilizes a small number of anecdotal case studies and questionnaires to draw conclusions. The research has further been complicated as studies have utilized various terms to describe Internet addiction, including *Internet dependency, problematic Internet use, excessive use, heavy use, pathological Internet use*, and *compulsive Internet use.*

Studies on Internet addiction originated in the United States/ More recently, studies have documented Internet addiction in a growing number of countries such as Italy (Ferraro et al., 2007), Pakistan (Suhail & Bargees, 2006), and the Czech Republic (Simkova & Cincera, 2004). Reports also indicate that Internet addiction has become a serious public health concern in the People's Republic of China (BBC, 2005), Korea (Hur, 2006), and Taiwan (Lee, 2007). According to

Lee (2007), about 10% of China's more than 30 million Internet gamers are said to be addicted. To battle what has been called an epidemic by some reports, Chinese authorities regularly shut down Internet cafes, many illegally operated, in crackdowns that also include huge fines for their operators. The Chinese government has also instituted laws to regulate the number of hours adolescents can play online games and has opened the first inpatient treatment center for Internet addiction in Beijing.

It is difficult to estimate how widespread the problem is. A nationwide study conducted by a team from Stanford University's School of Medicine had estimated that nearly one in eight Americans exhibit at least one possible sign of problematic Internet use (Aboujaoude et al., 2006).

Therefore, this chapter reviews conceptualizations of Internet addiction, including the new proposed criteria for the diagnosis of Pathological Computer Use to be used in the revision of the DSM-V. In addition, this chapter reviews various forms that Internet addiction can take, including online sexual preoccupations, Internet gambling, and interactive applications from chatting to games.

This chapter also examines the treatment implications for working with Internet-addicted clients. Unlike other addictions, healthy overuse of the Internet can be very productive: people can surf for information, make vacation plans, book airline seats, do research, chat with friends, and bank online. Not all Internet use is unproductive or nonlegitimate. Treatment of Internet addiction involves the attempt to moderate and control compulsive use while retaining a healthy balance between computer use and other aspects of a client's life. In addition to discussing the larger issue of conceptualizing and diagnosing the problem, once it is brought into the treatment arena, the issue becomes how to help clients find that balance when they rely on computers in their daily lives.

Diagnostic Conceptualization

According to Dr. Maressa Hecht Orzack, director of the Computer Addiction Services at McLean Hospital, a Harvard Medical School affiliate and another pioneer in the study of Internet addiction, Internet addicts demonstrate loss of impulse control in which life becomes unmanageable for them but, despite the resulting problems, they cannot moderate their Internet use. The computer becomes the primary relationship in the addict's life (Orzack, 1999).

While time is not a direct factor in diagnosing Internet addiction, early studies suggested that persons classified as dependent or addicted Internet users generally had an excessive habit, spending anywhere from 40 to 80 hours per week online, with sessions lasting up to 20 hours (Greenfield 1999; Young, 1998). Sleep patterns were disrupted due to late night log-ins and addicts generally stayed up surfing until late in the morning, with the reality of having to wake up early for work or school. In extreme cases, caffeine pills were used to facilitate longer Internet sessions. Such sleep deprivation caused excessive fatigue impairing academic or occupational performance, which also increased the risks of poor diet and insufficient exercise.

Given the popularity of the Internet, detecting and diagnosing Internet addiction is often difficult, as its legitimate business and personal use often mask addictive behavior. The best method for clinical detection of compulsive Internet use is to compare it against criteria for other established addictions. Researchers have likened Internet addiction to addictive syndromes similar to impulse control disorders on the Axis I Scale in the DSM and have utilized various forms of DSM-IV-based criteria to define Internet addiction. Of all the conditions referenced in the DSM, pathological gambling was viewed as most akin to this phenomenon. The Internet Addiction Diagnostic Questionnaire (IADQ) was developed as an initial screening instrument for diagnosis (Young, 1998). The following questions from this questionnaire conceptualize patterns associated with the disorder:

1. Do you feel preoccupied with the Internet (think about previous online activity or anticipate the next online session)?
2. Do you feel the need to use the Internet for increasing amounts of time in order to achieve satisfaction?
3. Have you repeatedly made unsuccessful efforts to control, cut back, or stop Internet use?
4. Do you feel restless, moody, depressed, or irritable when attempting to cut down or stop Internet use?
5. Do you stay online longer than originally intended?
6. Have you jeopardized or risked the loss of a significant relationship, job, educational or career opportunity because of the Internet?
7. Have you lied to family members, therapists, or others to conceal the extent of involvement with the Internet?
8. Do you use the Internet as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)?

Answers to these questions were used to evaluate nonessential computer/Internet use, such as nonbusiness or nonacademically related use. Subjects were considered dependent if they endorsed when answering five or more of the questions over a 6-month period. Associated features included ordinarily excessive Internet use, neglect of routine duties or life responsibilities, social isolation, and being secretive about online activities or suddenly demanding privacy when online. While the IADQ provides a means to conceptualize pathological or addictive use of the Internet, these warning signs can often be masked by cultural norms that encourage and reinforce online use. Even if a client meets all the criteria, signs of abuse can be rationalized by statements such as "I need this for my job" or "It's just a machine" when in reality the Internet is causing significant problems in a user's life.

Beard and Wolf (2001) further modified Young's diagnostic criteria, recommending that all of the first five criteria be required for diagnosis of Internet addiction, since these criteria could be met without any impairment in the person's daily functioning. They also recommended that at least one of the last three criteria (i.e., criteria 6, 7, or 8) be required in diagnosing Internet addiction. The last three criteria were separated from the others because they impact the pathological Internet user's ability to cope and function (e.g., depressed, anxious, escaping problems) and also affect the user's interaction with others (e.g., significant other, colleagues at work). Shapiro et al. (2003) put forth a more comprehensive approach to diagnosing Internet addiction under the general heading of impulse control disorders per the DSM-IV-TR that further broadened the diagnostic criteria for problematic Internet use:

A. Maladaptive preoccupation with Internet use, as indicated by at least one of the following: 1. Preoccupations with use of the Internet that are experienced as irresistible. 2. Excessive use of the Internet for periods of time longer than planned.

B. The use of the Internet or the preoccupation with its use causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The excessive Internet use does not occur exclusively during periods of hypomania or mania and is not better accounted for by other Axis I disorders.

Most recently, the American Psychiatric Association has considered including the diagnosis of Pathological Computer Use in the upcoming revision of the DSM-V (Block, 2007; Block, 2008). Conceptually, the diagnosis is a compulsive-impulsive spectrum disorder that involves online and/or offline computer use (Dell'Osso et al., 2006; Hollander & Stein, 2006) and consists of at least three subtypes: excessive gaming, sexual preoccupations, and e-mail/text messaging (Block, 2007). All of the variants share the following four characteristics: (1) *excessive use*, often associated with loss of the sense of time or neglect of basic drives; (2) *withdrawal*, including feelings of anger, tension, and/or depression when the computer is inaccessible; (3) *tolerance*, including the need for better computer equipment, more software, or more hours of use; and (4) *negative repercussions*, including arguments, lying, poor achievement, social isolation, and fatigue (Beard & Wolf, 2001; Block, 2008). This last criterion seems to pull together all the previous forms of classification of Internet addiction, defining it in a comprehensive manner that includes the major components associated with the compulsive behavior.

Motivation for Treatment

Once Internet addiction is diagnosed, the next step is to assess the client's motivation for treatment. In the early stages of recovery, clients will typically deny or minimize their habitual use of the Internet and the consequences of their behavior for their lives. Often, a friend, a spouse, or a parent has pushed the individual to seek help, especially in cases of addiction. The client may feel resentful and deny that use of the Internet is a problem. To break this pattern, after diagnosis, the therapist should use motivational interviewing techniques that encourage the client to commit to treatment as an integral aspect of recovery (Greenfield 1999; Orzack, 1999).

The concepts and approaches of motivational interviewing evolved from experience in the treatment of problem drinkers and were first described by Miller (1983). They were later elaborated by Miller and Rollnick (1991), who provided a more detailed description of the clinical procedures. Motivational interviewing is a goal-directed style of counseling for eliciting behavior change by helping clients to explore and resolve ambivalence. It involves asking open-ended questions, giving affirmations, and using reflective listening.

Motivational interviewing is intended to confront the client in a constructive manner to evoke change or, using external contingencies such as the potential loss of a job or relationship, to mobilize a client's values and goals to stimulate behavior change. Clients dealing with addiction or substance

abuse problems often feel ambivalent about quitting, even after they admit that they have a problem. They fear the loss of the Internet; they fear what life might be like if they were unable to chat with online friends, engage in online activities, and use the Internet as a form of psychological escape. Motivational interviewing helps clients confront their ambivalence.

Questions can be asked such as:

- When did you first begin to use the Internet?
- How many hours per week do you currently spend online (for nonessential use)?
- What applications do you use on the Internet (specific sites/groups/games)?
- How many hours per week do you spend using each application?
- How would you rank order each application from most to least important (1 = first, 2 = second, 3 = third, etc.)?
- What do you like best about each application? What do you like least?
- How has the Internet changed your life?
- How you do you feel when you log off?
- What problems or consequences have stemmed from your Internet use? (If this is difficult for the client to describe, have the client keep a log near the computer in order to document such behaviors for the next week's session.)
- Have others complained about how much time you spend online?
- Have you sought treatment for this condition before? If so, when? How much success did you have?

The answers to these questions create a clearer clinical profile of the client. The therapist can determine the types of applications that are most problematic for the client (e.g., chat rooms, online gaming, online pornography). The length of Internet use, the consequences of the behavior, a history of prior treatment attempts, and the outcomes of any such attempts are also assessed. This helps clients begin the process of examining how the Internet impacts their lives.

It is helpful for clients to gain a sense of responsibility for their behavior. Allowing clients to resolve their ambivalence in a manner that gently pushes them makes clients more inclined to acknowledge the consequences of their excessive online use and engage in treatment. Generally, the therapeutic style is quiet and eliciting rather than aggressive, confrontational, or argumentative. For therapists accustomed to confronting and giving advice, motivational interviewing may appear to be a hopelessly slow and passive process. However, the proof is in the outcome. More aggressive strategies, sometimes guided by a desire to "confront client denial," easily push clients into making changes for which they are not ready.

In cases of impulse control disorders, an individual's compulsive behavior is often associated with increasingly painful states of tension and agitation that are relieved by the completion of the act. For example, an alcoholic is driven to drink or an overeater is driven to binge on food during moments of tension and stress. The compulsive behavior serves to reduce the underlying emotional tension and serves as a reward for future behavior. Similarly, interactive online features such as chat rooms, instant messaging, or texting can be less about using the Internet as a communication tool and more about finding a psychological escape (Young, 2004).

Researchers have suggested that, like the person who craves a cigarette or a drink, Internet addicts feel a difference between online and offline emotions (Young, 1998). They may feel frustrated, worried, angry, anxious, and depressed when offline. When online, they feel excited, thrilled, uninhibited, attractive, supported, and more desirable. These strong positive emotions reinforce the compulsive behavior (Orzack, 1999). The behavior acts as a way to temporarily avoid negative or unpleasant feelings. Under the influence, the alcoholic feels as if all the other problems in life have disappeared. When eating, the overeater experiences a sense of peace and relaxation, lessening the overwhelming stress and frustration he or she feels. In the same manner, compulsive Internet users use the virtual world to escape life's problems temporarily, involving themselves deeply in virtual relationships carried on by chat, text, or e-mail. Over time, this coping mechanism can prove unproductive and potentially harmful as the issues hidden by the compulsive behavior culminate in larger problems.

Helping the client explore how he or she feels just before going online will help pinpoint the types of emotions being covered by the behavior (or how the client is using the Internet to cope or escape from problems). Answers may include issues such as a fight with a spouse, depressed mood, stress at a job, or a poor grade in school. Motivational interviewing should explore how these feelings diminish when online, looking for how the client rationalizes or justifies using the Internet (e.g., "Chatting makes me forget about the fight with my husband," "Looking at online porn makes me feel

less depressed," "Gambling online makes me feel less stressed at work," "Killing other players in an online game makes me feel better about my poor grade at school"). Motivational interviewing is also meant to help the client recognize the consequences stemming from excessive or compulsive use. Problems may consist of issues like these: "My spouse becomes angrier," "My depressed feelings return when I turn off the computer," "My job still stinks," and "I will loss my scholarship if I don't get my grades up." The therapeutic relationship is more like a partnership or companionship than one of expert/recipient roles to examine and resolve ambivalence. The operational assumption in motivational interviewing is that ambivalence is the principal obstacle to be overcome in triggering change. Overall, the specific strategies are designed to elicit, clarify, and resolve ambivalence in a client-centered and respectful therapeutic manner.

Classic and New Internet Abusers

Based upon clinical interviews with over 2000 Internet addicts in the past decade, there appear to be two types of addicts or abusers. Some clients suffer from a prior addiction, such as to sex or gambling, and they use the Internet as a new outlet to act on this addiction. These clients are referred to as *Classic Addicts*. Other clients have no history of prior addiction; their addiction to the Internet is an entirely new problem behavior. These clients are referred to as *New Addicts*. Classic Addicts are generally relapsing on the Internet into those activities that they can perform in real life, such as pathological gambling or sexually compulsive behavior.

For examples, for those Classic Addicts in recovery from sex addiction, the Internet becomes another way to engage in sexually compulsive behavior. Recovering addicts who feel overwhelmed, or who experience personal problems or life-changing events such as divorce, relocation, or the death of a loved one, can become absorbed in a virtual world full of fantasy and intrigue; the Internet serves as a new way to engage in sexual behavior (Young, 2008). Sexual compulsives discover a new source for sexual gratification through online pornography and anonymous sex chat. The Internet allows them to continue their sexual behavior without the physical need to visit strip clubs or prostitutes and provides a new and socially acceptable way to cope.

Classic Addicts may also suffer from a history of alcohol or drug dependency, only to find their compulsive use of the Internet a physically safe alternative to their addictive tendency. They believe that being addicted to the Internet is medically safer than being addicted to drugs or alcohol; at the same time, the compulsive behavior avoids the need to confront the unpleasant situation underlying the addiction.

Clients who suffer from multiple addictions (to the Internet as well as to alcohol, cigarettes, drugs, food, sex, etc.) are at the greatest risk of relapsing. This is especially true when it comes to the Internet. Often, addicts need to use the computer for work or school, so the temptation to return to the problematic behavior feels constant because the computer is always available. Multiple addictions also suggest that the client has an addictive personality and compulsive tendencies, making relapse more likely.

New Addicts meet two distinct criteria. First, they become addicted to new forms of Internet use created solely online, such as chat rooms, social networking, instant messaging, online role-playing games, or eBay shopping. For instance, someone who becomes addicted to chat rooms must use the Internet to chat. Someone who becomes addicted to eBay must use the Internet to access it. Granted, these activities have now become portable through mobile devices such as personal digital assistants (PDAs) or cell phones. The key element is that they are all considered Internet-specific activities.

Secondly, New Addicts are individuals with no previous significant addictive or psychiatric history. They develop an addiction to the anonymous, accessible, and interactive nature of online use. New Addicts may include a 50-year-old lawyer using sex chat rooms during work hours and without his wife's knowledge, a 30-year-old business executive compulsively checking his Blackberry, a 20-year-old college student constantly on Facebook, or a 16-year-old boy constantly playing online role-playing games. For New Addicts, compulsive use of the Internet is a new clinical phenomenon.

New Addicts are attracted to the Internet, which is often first seen as a problem by others, usually a spouse, parent, or close friend. Their behavior progressively revolves around Internet use. It involves more and more of a focus on technology that may have initially been required for work use, such as a Blackberry, or may have been an activity pursued for recreation, such as a chat room meeting or game. As the behavior escalates, online use becomes more chronic and ingrained and develops into a compulsive obsession. In this stage, life becomes unmanageable for the addict, as relationships or careers are jeopardized because of the compulsive behavior.

In one such case, I worked with a 34-year-old minister arrested for possession of child pornography obtained from the Internet. He explained his fascination: "I soon discovered the vast array of pornography, including child pornography, available on the Internet. My attraction to online porn was born of sheer amazement at the volume of available material, and this amazement turned to fascination and ultimately to obsession. I knew it was wrong to look at this material. My life became a lonely, isolated mess. I realized that I could lose my job, my marriage, and the respect of everyone I love if I was caught. I have two daughters and would never think about doing anything inappropriate with them, but I could not bring myself to stop despite knowing all the consequences of my actions. What started as curiosity has put my faith and everything important to me in jeopardy."

The New Addict becomes preoccupied with the computer and constantly feels a longing to be online. The person attempts to conceal the nature of his or her online activities and fears that others will discover this secret life. Realizing the impact of this destructive behavior, the person rationalizes it and continues to engage in the activity despite its known potential risks, including possible job loss, divorce, or arrest. The online experience turns into a relief from pain and anxiety, the reward for success, and a way to avoid addressing other emotional issues in the person's life. The addiction is truly an altered state of consciousness in which normal behavior pales by comparison in terms of excitement and relief from troubles that is associated with use of the Internet. In this way, the online world becomes a private refuge. The addict displays a progressive retreat into the computer world as a means of avoiding life's complications and responsibilities.

Underlying Social Problems

Excessive or problematic Internet use often stems from interpersonal difficulties such as introversion or social problems (Ferris, 2001). Many Internet addicts fail to communicate well in face-to-face situations (Leung, 2007). This is part of the reason they use the Internet in the first place. Communicating online seems safer and easier for them. Poor communication skills can also cause low self-esteem and a feeling of isolation and create additional problems in life, such as trouble working in groups, making presentations, or going to social engagements. Assessment and treatment needs to address how Internet addicts communicate offline. Encouraging affect, communication analysis, modeling, and role playing are helpful interventions to apply (Hall & Parsons, 2001).

Researchers have suggested that individuals who suffer from low self-esteem, and who feel lonely, restless, or withdrawn, can use online connections to feel better about themselves and their circumstances (Morahan-Martin & Schumacher, 2003). Socially awkward or emotionally troubled individuals may find it easier to engage in Internet relationships than risk the face-to-face rejection of a real person (Ferris, 2001). The anonymity associated with electronic communication can also increase the online user's feeling of comfort since there is no need to look for, and thus detect, signs of insincerity, disapproval, or judgment in facial expressions, as would be typical in face-to-face interactions (Morahan-Martin & Schumacher, 2003).

Among married couples, this can lead to online affairs (Whitty, 2005). An online affair is a romantic or sexual relationship initiated via online contact and maintained predominantly through electronic conversations and online communication (Atwood & Schwartz, 2002). Electronic communication allows individuals to feel less inhibited, which accelerates perceived intimacy. Online, people are more likely to be open, honest, and forthright, revealing personal truths and the intimacy that might take months or years to establish in an offline relationship may only takes days or weeks online (Cooper & Sportolari, 1997). Online, people can seem more glamorous than they are in day-to-day life (Whitty, 2005). Married Internet users can utilize e-mail and texting to seek out support, comfort, and acceptance from a romantic online partner that gives them a sense of belonging in a nonthreatening manner. Unfortunately, online affairs do more than create a troubling new clinical problem for couples dealing with Internet infidelity (Whitty, 2005). According to the American Academy of Matrimonial Lawyers, 63% of attorneys found that online affairs accounted for a growing number of divorce cases (Dedmon, 2003), creating new legal problems.

As demand has increased for mobile devices, PDAs, and cell phones with Internet access, problem mobile phone use has grown, especially among younger persons (Bianchi & Phillips, 2005). As a result, e-mailing and texting overuse have become growing social and clinical issues. Time spent e-mailing and texting can impair important and purposeful relationships. The more time is spent checking e-mail or chatting online, the less time is spent with one's family and friends. Ultimately, these

new ways of communicating have created a new type of *technostress*.

Due to their Internet addiction, individuals also often damage or lose significant real-life relationships, such as those with a spouse, a parent, or a close friend (Young, 2004). Often, these were individuals who provided the addict with support, love, and acceptance before the Internet, and their absence makes the addict feel worthless and reinforces past notions that they are unlovable. The addict must mend and reestablish these broken relationships to achieve recovery and find the support necessary to fight the addiction. Rebuilding relationships and providing new ways to relate to others allows for amends to be made. Involving loved ones in recovery can be a rich source of nurturing and sponsorship to help a client maintain sobriety and abstinence. Couples or family therapy may be necessary to help educate loved ones about the addiction process and engage them more fully in helping the client maintain boundaries established with the computer.

When evaluating social problems, it is important to investigate how the client has been using the Internet. If it has involved interactive environments such as chat rooms, instant messaging, or social networking sites, then the therapist should evaluate aspects of online use by asking questions such as the following: Does the person make up a persona? What kind of screen name does the person use? Does Internet its use disrupt current social relationships? If so, how? These are important issues to evaluate in order to understand the social dynamics underlying online usage and how relationships formed on the Internet may be substituting or replacing relationships in real life. Possible issues to consider are:

- Have you been honest about your Internet habit with your friends and family?
- Have you ever created an online identity or persona?
- List online activities that you kept secret or thought others would not approve of.
- Have online friends disrupted real-life relationships?
- If so, who was impacted (husband, wife, parent, friend) and how?
- Does Internet use disrupt your social or work relationships?
- If yes, please describe how this happens.
- Please describe other ways in which Internet use has impacted your life.

Questions and requests like these help structure the clinical interview to provide more detailed information on how the Internet has impacted relationships in the client's life. Often clients create online personas, and the answers to these questions provide specific information on the characteristics and nature of these personas. They help therapists understand the client's psychological motives, the ways online personas develop, and how they may be used to fulfill missing or unmet social needs. Once this type of critical examination takes place, the therapist can work with the client to develop new social relationships or reestablish former social connections that will sustain his or her motivation for continued treatment.

Treatment Considerations

Cognitive behavioral therapy (CBT) has been shown to be an effective treatment for compulsive disorders such as intermittent explosive disorder, pathological gambling, and trichotillomania (Hucker, 2004). It has also been effective in treating substance abuse, emotional disorders, and eating disorders as well (e.g., Beck, 1979; Beck et al., 1993).

Cognitive behavioral therapy is a familiar treatment based on the premise that thoughts determine feelings. Patients are taught to monitor their thoughts and identify those that trigger addictive feelings and actions while they learn new coping skills and ways to prevent a relapse. Cognitive behavioral therapy usually requires 3 months of treatment or approximately 12 weekly sessions. The early stage of therapy is behavioral, focusing on specific behaviors and situations in which the impulse control disorder causes the greatest difficulty. As therapy progresses, there is more of a focus on the cognitive assumptions and distortions that have developed and the effects of the compulsive behavior.

In cases of Internet addiction, researchers have suggested that moderated and controlled use of the Internet is the most appropriate treatment (Greenfield, 1999; Orzack, 1999). Behavior therapy is the initial focus of recovery, examining both computer and noncomputer behavior (Hall & Parsons, 2001). Computer behavior deals with actual online usage, with a primary goal of abstinence from problematic applications while retaining controlled use of the computer for legitimate purposes. For example, a lawyer addicted to Internet pornography would need to learn to abstain from visiting adult web sites while still accessing the Internet to conduct legal research and to e-mail clients. Noncomputer behavior focuses on helping clients develop positive lifestyle

changes for life without the Internet. Life activities that do not involve the computer such as offline hobbies, social gatherings, and family activities are encouraged. Similarly to food addiction, in which recovery can be objectively measured through reduced caloric intake and weight loss, online addicts can objectively measure success by maintaining abstinence from problematic online applications and increasing meaningful offline activities.

Young (2007) suggests using a Daily Internet Log to evaluate computer behavior and establish a baseline for clinical treatment. Once a baseline has been established, behavior therapy is used to relearn how to use the Internet to achieve specific outcomes, such as moderated online usage and, more specifically, abstinence from problematic online applications and controlled use for legitimate purposes. Behavior management for both computer usage and adaptive noncomputer behavior focuses on current online behavior. Early outcome studies show that assertion training, behavioral rehearsal, coaching, modeling, and relaxation training are effective methods.

From a cognitive perspective, addictive thinkers, for no logical reason, feel apprehensive when anticipating disaster (Twerski, 1990). While addicts are not the only people who worry and anticipate negative events, they tend to do this more often than other people. Young (1998) first suggested that this catastrophic thinking might contribute to compulsive Internet use by providing a psychological escape mechanism to avoid real or perceived problems. Subsequent studies hypothesized that other maladaptive cognitions such as overgeneralizing or catastrophizing, negative core beliefs, and cognitive distortions also contribute to compulsive use of the Internet (Caplan, 2002; Davis, 2001; LaRose et al., 2001). Those who suffer from negative core beliefs may be the ones drawn most strongly to the anonymous interactive capabilities of the Internet in order to overcome their perceived inadequacies. Early treatment outcome studies show that CBT can be used to address these negative core beliefs, cognitive distortions, and rationalizations such as "Just a few more minutes won't hurt" that sustain problematic or compulsive use of the Internet (Young, 2007).

Future Practice

Over the last decade, the acceptance of Internet addiction has grown in the mental health field, with a new emphasis on assessment and treatment of the disorder. It is difficult to predict the results of these early endeavors. However, it is feasible that with years of collective effort, Internet addiction may be recognized as a legitimate impulse control disorder worthy of its own classification in future revisions of the *Diagnostic and Statistical Manual of Mental Disorders*.

With the growing popularity of the Internet, increased awareness in the mental health field will help clinicians provide knowledgeable care and intervention for the Internet-addicted client. Since this is a new and often seemingly comical addiction, individuals may be reluctant to seek treatment, fearing that clinicians may not take their complaints seriously. Drug and alcohol rehabilitation centers, community mental health clinics, and clinicians in private practice should be aware of the negative ramifications of compulsive Internet use and recognize the signs, which may easily be masked by other comorbid conditions or legitimate use of the Internet.

To pursue effective recovery programs, continued research is needed to better understand the underlying motivations of Internet addiction. Future research should focus on how psychiatric illnesses such as depression or obsessive-compulsive disorder plays a role in the development of compulsive Internet use. Longitudinal studies may reveal how personality traits, family dynamics, or interpersonal skills influence the way people utilize the Internet. Further outcome studies are needed to determine the efficacy of specialized therapeutic approaches to treat Internet addiction and compare their outcomes with those of traditional treatment modalities.

As psychiatric comorbidity is a factor in the development of compulsive Internet use, research should also be considered for medication approaches in treating Internet addiction. No research has currently been conducted in this area. In severe cases, residential inpatient care may be warranted. Inpatient treatment programs provide coordinated and integrated services in hospital settings. Specialized recovery centers are emerging in China, Korea, and Taiwan. In the United States, the new Restart Program in Redmond, Washington, has opened, providing 45-day intensive care for Internet addicts. Generally, the goal of inpatient treatment is to provide a protective environment that includes medical stabilization, support, treatment for psychiatric or addictive disorders, and supervision. Inpatient treatment may combine group and individual treatments with family education and therapy as needed, especially for child and adolescent patients.

References

Aboujaoude, E., Koran, L. M., Gamel, N., Large, M. D., & Serpe, R. T. (2006). Potential markers for problematic Internet use: A telephone survey of 2,513 Adults *CNS* Spectrum, *11*(10), 750–755.

Atwood, J. D., & Schwartz, L. (2002). Cyber-sex: The new affair treatment considerations. *Journal of Couple & Relationship Therapy*, 1(3), 37–56.

BBC News (2005). *China imposes online gaming curbs*. Retrieved August 7, 2007, http://news.bbc.co.uk/1/hi/technology/4183340.stm

Beard, K. W., & Wolf, E. M. (2001). Modification in the proposed diagnostic criteria for Internet addiction. *CyberPsychology & Behavior, 4*, 377–383.

Beck, A. T. (1979). *Cognitive therapy and the emotional disorders*. New York, NY: Plume.

Beck, A. T., Wright, F. D., Newman, C. F., & Liese, B. S. (1993). *Cognitive therapy of substance abuse*. New York, NY: Guilford Press.

Bianchi, A., & Phillips, J. (2005). Psychological predictors of problem mobile phone use. *CyberPsychology & Behavior,* 8(1), 39–51.

Block, J. J. (2007). Pathological computer use in the USA. *International Symposium on the Counseling and Treatment of Youth Internet Addiction* (p. 433). Seoul, Korea, National Youth Commission.

Block, J. J. (2008). Issues for DSM-V: Internet addiction. *American Journal of Psychiatry, 165,* 306–307.

Caplan, S. E. (2002). Problematic Internet use and psychosocial well-being: Development of a theory-based cognitive-behavioral measurement instrument. *Computers in Human* Behavior, *18*, 553–575.

Cooper, A., & Sportolari, L. (1997). Romance in cyberspace: Understanding online attraction. *Journal of Sex Education and Therapy*, *22*(1), 7–14.

Davis, R. A. (2001). A cognitive behavioral model of pathological Internet use. *Computers in Human Behavior*, *17*, 187–195.

Dedmon, J. (2003). *Is the Internet bad for your marriage?* Study by the American Academy of Matrimonial Lawyers, Chicago. Retrieved January 28, 2008, from http://www.expertclick.com/NewsReleaseWire/default.cfm?Action=ReleaseDetail&ID=3051

Dell'Osso, B., Altamura, A. C., Allen, A., Marazziti, D., & Hollander, E. (2006). Epidemiologic and clinical updates on impulse control disorders: A critical review. *Clinical Neuroscience*, *256*, 464–475.

Ferraro, G., Caci, B., D'Amico, A., & Di Blasi, M. (2007). Internet addiction disorder: An Italian study. *CyberPsychology & Behavior*, *10*(2), 170–175.

Ferris, J. (2001). Social ramifications of excessive Internet use among college-age males. *Journal of Technology and Culture, 20*(1), 44–53.

Greenfield, D. (1999). *Virtual addiction: Help for netheads, cyberfreaks, and those who love them*. Oakland, CA: New Harbinger Publications.

Hansen, S. (2002). Excessive Internet usage or "Internet addiction"? Diagnostic categories for student users. *Journal of Computer Assisted Learning, 18*(2), 235–239.

Hollander, E., & Stein, D. J. (Eds.). (2006). *Clinical manual of impulse-control disorders*. Arlington, VA: American Psychiatric Publishing.

Hucker, S. J. (2004). Disorders of impulse control. In W. O'Donohue & E. Levensky (Eds.), *Handbook of Forensic Psychology* (pp. 471–484). New York: Academic Press.

Hur, M. H. (2006). Internet addiction in Korean teenagers. *CyberPsychology & Behavior*, *9*(5), 14–525.

Lee, M. (2007). China to limit teens' online gaming for exercise. Retrieved on August 7, 2007 from http://www.msnbc.msn.com/id/19812989/

Leung, L. (2007). Stressful life events, motives for Internet use, and social support among digital kids. *CyberPsychology & Behavior*, *10*(2), 204–214.

Miller, W. R. (1983). Motivational interviewing with problem drinkers. *Behavioural Psychotherapy*, *11*, 147–172.

Miller, W. R., & Rollnick, S. (1991). *Motivational interviewing: Preparing people to change addictive behavior*. New York: Guilford Press.

Morahan-Martin, J., & Schumacher, P. (2003). Loneliness and social uses of the Internet. *Computers in Human Behavior*, *19*(6), 659–671.

Orzack, M. (1999). Computer addiction: Is it real or is it virtual? *Harvard Mental Health Letter*, *15*(7), 8.

Shapira, N. A., Lessig, M. C., Goldsmith, T. D., Szabo, S. T., Lazoritz, M., Gold, M. S., & Stein, D. J. (2003). Problematic Internet use: Proposed classification and diagnostic criteria. *Depression and Anxiety*, *17*, 207–216.

Simkova, B., & Cincera, J. (2004). Internet addiction disorder and chatting in the Czech Republic. *CyberPsychology & Behavior*, *7*(5), 536–539.

Suhail, K., & Bargees, Z. (2006). Effects of excessive Internet use on undergraduate students in Pakistan. *CyberPsychology & Behavior, 9*(3), 297–307.

Twerski, A. (1990). *Addictive thinking: Understanding self-deception*. New York: HarperCollins.

Whitty, M. (2005). The realness of cybercheating. *Social Science Computer Review*, *23*(1), 57–67.

Young, K. S. (1998). Internet addiction: The emergence of a new clinical disorder. *CyberPsychology and Behavior, 1*, 237–244.

Young, K. S. (2004). Internet addiction: The consequences of a new clinical phenomena. In K. Doyle (Ed.), *Psychology and the new media* (pp. 1–14). Thousand Oaks, CA: American Behavioral Scientist.

Young, K. S. (2007). Cogntive-behavioral therapy with Internet addicts: Treatment outcomes and implications. *CyberPsychology & Behavior*, *10*(5), 671–679.

Young, K. (2008). Internet sex addiction: Stages, risk factors, and treatment. In K. Doyle (Ed.), *Psychology and the new media* (Vol. 2, (1): 21–37). Thousand Oaks, CA: Sage.

CHAPTER

30 Assessment and Treatment of Compulsive Buying

Astrid Mueller, James E. Mitchell, *and* Lisa A. Peterson

Abstract

Compulsive buying disorder (CBD) affects a significant percentage of those in the general population. However, CBD has not received as much attention with regard to research and training providers in its assessment and treatment as other psychiatric disorders. Formal diagnostic criteria have been put forth, and there are a variety of assessment instruments for evaluating buying behavior using both questionnaire and interview formats. Based on the literature to date, it appears that disorder-specific cognitive-behavioral therapy has been most successful in treating those with CBD. Treatment providers should also be aware that mood disorders, anxiety disorders, and compulsive hoarding often accompany CBD and complicate its treatment, and these disorders should be addressed within the treatment paradigm. Further research is needed to define barriers to motivation for change and treatment compliance among individuals with CBD, as well as factors associated with both positive and negative treatment outcomes.

Keywords: compulsive buying, Compulsive Buying Scale, impulse control disorder, compulsive hoarding, cognitive-behavioral therapy

Introduction

Compulsive buying disorder (CBD) is characterized by shopping and buying behavior that results in marked psychological distress as well as financial and interpersonal problems (Faber & O'Guinn, 1992; O'Guinn & Faber, 1989). Although CBD may seem to have emerged only recently, Kraepelin (1909) and Bleuler (1983) originally described *onionmania*, or the urge to buy, a century ago. More recently, research has examined the prevalence of CBD as well as ways to classify its symptoms (Black, 2007). Using the Compulsive Buying Scale (CBS; Faber & O'Guinn, 1992), Koran and colleagues (2006) estimated the prevalence of CBD to be 5.8% within a large general population sample in the United States. Compared with other participants, those with CBD were younger and reported lower incomes but did not differ as to gender.

Compulsive buying disorder is not specifically described in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; APA, 1994) or in the *International Classification of Mental and Behavioral Disorders* (ICD-10; World Health Organization, 1994) but is currently conceptualized as an "impulse control disorder not otherwise specified." In treatment-seeking individuals, the problematic buying behavior is associated with high rates of psychiatric comorbidity, especially mood and anxiety disorders, personality disorders, impulse control disorders (Black et al., 2000; Mitchell et al., 2002; Ninan et al., 2000; Schlosser et al., 1994), and compulsive hoarding (Frost et al., 2009; Mueller et al., 2007). Although research on this disorder has increased over the past 20 years, CBD remains frequently overlooked in clinical practice and many therapists lack specific information on its assessment and treatment.

Assessment

Compulsive buying disorder can be assessed by precise exploration of buying behavior and the

thoughts and feelings associated with buying and shopping. According to McElroy and colleagues (1994), who proposed diagnostic criteria, the disorder is characterized by frequent buying episodes or impulses to buy that are experienced as irresistible or senseless. The spending behaviors and impulses lead to personal distress, social, marital, or occupational dysfunction, and financial or legal problems. The excessive buying behavior does not occur exclusively during episodes of mania or hypomania. Individuals should be asked about these symptoms during the course of a general clinical interview. A variety of other interviews and assessment techniques may then be implemented if indicated.

Several interviews and questionnaires have been developed to assess CBD specifically or within the context of other impulse control disorders.

Questionnaires

COMPULSIVE BUYING SCALE (CBS)

The most widely used screening instrument in empirical research on CBD is the CBS (Faber & O'Guinn, 1992). This screening instrument consists of seven items utilizing a 5-point Likert scale for responses. Two items explore emotional reactions to shopping and five items ask about financial consequences of buying. The unidimensional scale reflects such characteristics as lack of impulse control, distress at the thought of others' knowledge of the person's purchasing habits, irrational use of credit cards, tension when not shopping, and the use of spending to feel better. Lower scores on the scale indicate higher levels of compulsive buying. The authors recommended a cut-off score of two standard deviations below the general population mean (–1.34) to identify those with compulsive buying. In the original study, the CBS correctly classified 89.9% of a general population sample and 85.3% of a compulsive buying group (Faber & O'Guinn, 1992)

CANADIAN COMPULSIVE BUYING MEASUREMENT SCALE

This scale was originally written in French because the study was done with French-speaking consumers (Valence et al., 1988). The items have since been translated from French to English. The Canadian Compulsive Buying Measurement Scale consists of 13 items distributed among three dimensions: (1) tendency to spend (6 items); (2) reactive aspect (4 items); and (3) postpurchase guilt (3 items). Participants are required to express the extent to which they agree with each item on a 5-point Likert scale. The measure demonstrated adequate internal consistency in the original study with 38 respondents with CBD and an equal number of noncompulsive buying individuals, with a Cronbach's alpha of .88 (Valence et al., 1988).

EDWARDS COMPULSIVE BUYING SCALE

Viewing compulsive buying as lying on a continuum from healthy to unhealthy buying behavior, Edwards (1993) developed the Edwards Compulsive Buying Scale to determine how compulsive or addictive subjects are in their spending behavior. The scale includes 13 items that load on five different factors: (1) tendency to spend (5 items); (2) compulsion/drive to spend (2 items); (3) feelings about shopping and spending (2 items); (4) dysfunctional spending (2 items); and (5) postpurchase guilt (2 items). Cronbach's alpha in a sample of 105 individuals with CBD and 101 subjects of a convenience sample for the entire scale was .91 (Edwards, 1993).

EXECUTIVE PERSONAL FINANCE SCALE

Spinella and colleagues (2007) developed the Executive Personal Finance Scale as a specific self-rating measure of executive aspects of personal money management regarding impulse control over spending, conceptualization of finances, and financial planning. The 20-item scale has four factors: (1) impulse control; (2) organization; (3) planning; and (4) motivational drive. Cronbach's alpha for the total score was found to be .86 in a sample of 225 participants (Spinella et al., 2007).

RIDGWAY COMPULSIVE BUYING SCALE

Ridgway and colleagues (2008) developed a six-item scale that includes dimensions of both obsessive-compulsive and impulse control disorders while excluding negative financial consequences and income-related items. The instrument is based on a definition of compulsive buying in terms of proposed underlying consumer behavior tendencies, separating consequence effects of the spending behavior. Cronbach's alpha in a sample of 551 university staff members was .81 (Ridgway et al., 2008).

Interviews

MINNESOTA IMPULSIVE DISORDER INTERVIEW (MIDI)

The MIDI was developed by Christenson and colleagues (1994) for a descriptive study of compulsive buying. This semistructured interview consists of six separate screening modules examining criteria for the DSM-III-R impulse control disorders not elsewhere classified: trichotillomania, pyromania,

intermittent explosive disorder, kleptomania, and pathological gambling, as well as compulsive buying. Grant et al. (2005) reported that the MIDI had sensitivity of 100% and specificity of 96.2% for compulsive buying when comparing the instrument to the proposed diagnostic criteria of McElroy and colleagues (1994).

YALE-BROWN OBSESSIVE-COMPULSIVE SCALE–SHOPPING VERSION (YBOCS-SV)

Monahan and colleagues (2006) modified the YBOCS (Goodman et al., 1989) to measure symptom severity and change in persons with compulsive buying. The Shopping Version (YBOCS-SV) includes 10 questions, 5 exploring cognitions and 5 rating behaviors associated with uncontrolled buying. Consumers are asked about time involved, interference due to the preoccupations or the shopping, resistance to the thoughts or behaviors, and degree of control over the shopping and buying cognitions/behaviors. The authors compared a group of individuals clinically identified as having compulsive buying and control subjects and reported good interrater reliability for those with compulsive buying (r = .81), for control subjects (r = 0.96), and for both groups combined (r = 0.99). Cronbach's alpha was moderately high for treatment-seeking individuals with compulsive buying (.65) and for comparison subjects (.70; Monahan et al., 2006). The YBOCS-SV was sensitive to clinical change and was able to detect improvement during a clinical trial. Thus, this instrument is best used as a severity measure rather than as a screening measure of compulsive buying.

SCID-I IMPULSE CONTROL DISORDERS

The impulse control disorders section of the Structured Clinical Interview (SCID; First et al., 2002) includes questions about DSM-IV impulse control disorders not otherwise specified including intermittent explosive disorder, pathological gambling, pyromania, trichotillomania, compulsive buying, and kleptomania. A more recent version that has not been formally field tested yet includes a number of questions for CBD, as well as for proposed impulse control disorders such as impulsive-compulsive nonparaphilic sexual behavior disorder, impulsive-compulsive Internet use disorder, and impulsive-compulsive skin picking disorder.

It is important to note that self-ratings and semi-structured interviews should not replace the clinical evaluation. Clinicians ought to inquire in detail about shopping and spending, particularly buying attitudes, the extent of preoccupation with buying and shopping, buying behaviors, urges to buy, feelings and thoughts associated with buying, and interference with social, financial, and occupational functioning. Assessment further should include one of the self-ratings presented above. For example, Faber and O'Guinn's CBS (Faber & O'Guinn, 1992) is a useful instrument to screen for clinically significant compulsive buying. To measure the severity and interference caused by buying behaviors and cognitions, the YBOCS-SV (Monahan et al., 2006) should be administered; this instrument also allows for the measurement of clinical change within treatment.

Treatment

Pharmacological Treatment

Guidelines for the pharmacological treatment of CBD are lacking, and treatment research on this topic is limited. With regard to the high comorbidity with anxiety and affective disorders, individuals with CBD may benefit from medications that are successful in treating anxiety and depression. In 1991, McElroy and colleagues (McElroy et al., 1991) described the successful treatment of three individuals with CBD and comorbid mood disorder with antidepressants (buproprion, nortriptyline, and fluoxetine). Lejoyeux and colleagues (1995) reported two cases in which treatment of comorbid depression led to improvement of CBD. Until now, the best-studied class of drugs for CBD have been the selective serotonin reuptake inhibitors.

SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRIS)

The results of open-label trials suggested that SSRIs could reduce compulsive buying (Black et al., 1997; Koran et al., 2002; McElroy et al., 1991). For example, Black and colleagues (1997) treated 10 nondepressed subjects with CBD with fluvoxamine in a dose of up to 300 mg daily for 9 weeks. Nine of the 10 patients improved and were less preoccupied with shopping. Koran and colleagues (2002) enrolled 24 subjects with CBD in an open-label citalopram treatment trial. Exclusion criteria were obsessive-compulsive disorder, bipolar disorder, substance abuse/dependence disorders, or psychotic disorders. Seventeen subjects showed a substantial response and significant improvement in compulsive buying behavior. However, small randomized, controlled trials failed to confirm the optimistic results of open-label trials. Table 30.1 summarizes the results of the controlled psychopharmacological trials.

Table 30.1 Results of Medication Studies

Medication	Sample Size	Dosage/Day	Results
Fluvoxamine (Black et al., 2000)	Medication *n* = 12, Placebo *n* = 11	220 mg	Drug = Placebo
Fluvoxamine (Ninan et al., 2000)	Medication *n* = 20, Placebo *n* = 17	215 mg	Drug = Placebo
Citalopram (Bullock & Koran, 2003)	Open-label trial (*n* = 24) followed by double-blind, placebo-controlled discontinuation trial Responder *n* =15 ↓ Medication *n* = 7, Placebo *n* = 8	20–60 mg	Five of eight randomized to placebo relapsed compared with none of seven randomized to continue taking medication
Escitalopram (Koran et al., 2007)	Open-label trial (*n* = 26) followed by double-blind, placebo-controlled discontinuation trial Responder *n* = 17 ↓ Medication *n* = 8, Placebo *n* = 9	10–20 mg	Drug = Placebo

Two subsequent double-blind, placebo-controlled studies (Black et al., 2000; Ninan et al., 2000) did not find a significant difference in efficacy between fluvoxamine and placebo. In the study of Black and colleagues (2000), primary outcome measures included the YBOCS-SV, the National Institute of Mental Health Obsessive-Compulsive Scale (NIMHOCS), and three Clinical Global Impression (CGI) ratings. The improvement experienced by fluvoxamine recipients was similar to that of the placebo group, suggesting a high placebo response rate. Likewise, Ninan and colleagues (2000) failed to demonstrate significant differences between fluvoxamine-treated and placebo-treated group using the YBOCS-SV, the Hamilton Rating Scale for Depression (HAM-D), and the Global Assessment of Functioning (GAF) as outcome measures. Investigators in both studies assumed that shopping diaries, reviews of shopping behaviors at study visits, and nonspecific support during these visits may have contributed to the positive response.

Two subsequent open-label trials by Koran and colleagues were followed by a double-blind, placebo-controlled discontinuation phase (Koran et al., 2003, 2007). These studies yielded mixed results. In the first study to test the effectiveness of citalopram (Koran et al., 2003), primary outcome measures were the relapse rate in the double-blind portion of the study and the change in YBOCS-SV from randomization baseline to endpoint. Investigators decided to omit shopping diaries to avoid their potential therapeutic effect. Results of the double-blind phase suggested a true drug effect. Subjects randomized to double-blind citalopram did not report relapses and showed significant lower YBOC-SV scores at endpoint. In the second study, Koran and colleagues (2007) used an identical study design to test escitalopram for CBD. The similar relapse rates in the drug and placebo groups during the double-blind phase contradicted the findings of the previous study conducted with citalopram (Koran et al., 2007).

Overall, it seems likely that the improvement in compulsive buying during open-label SSRI treatment was not a true drug effect. Similar medication and placebo rates suggest that the promising results of open-label medication trials may have largely been a placebo response. However, it cannot be excluded that the medication trials failed to show a drug effect due to the lack of power.

OPIATE ANTAGONISTS

Dopaminergic reward pathways may be involved in CBD, and opioid antagonists appear to reduce buying urges (Bullock & Koran, 2003). There have been several treatment studies involving opiate antagonists for the treatment of other impulse control disorders, particularly pathological gambling (Grant et al., 2006, 2008) and kleptomania (Grant et al., 2009). With regard to CBD, only case reports rather than controlled trials have been published. For example, Kim (1998) described the treatment response to the opiate antagonist naltrexone (100 mg/day) in a female patient with CBD and three other cases. Grant (2003) reported improvement of

CBD in three patients treated with naltrexone using higher doses (100–200 mg/day).

In summary, pharmacological studies on compulsive buying are limited by small sample sizes, inadequate numbers of male subjects, and high placebo response rates. Larger controlled pharmacological studies are needed to determine whether or not medication may be effective in the treatment of CBD.

Psychotherapy Treatment

The earliest case reports illustrated psychodynamic considerations in therapeutic interventions for CBD (Krueger, 1988; Lawrence, 1990; Winestine, 1985). Winestine (1985), in his case report, described a woman in her late 30s who sought treatment because of uncontrollable shopping sprees. The psychoanalytical treatment focused on the role of her remembered childhood seduction and demonstrated the interdigitation of childhood abuse with the development of intrapsychic conflicts and adult compulsive buying symptoms. With regard to Krueger (1988), who reported four casuistics, CBD may occur as a reparative effort for a fragmented sense of self and the experience of emptiness. The author recommended as therapeutic interventions emphatic resonance, understanding, and development of a more cohesive sense of self. Lawrence (1990) suggested that castration anxiety could be the main motivation of female compulsive buying.

A letter to the editor by Bernik and colleagues (1996) reported on two women with CBD. Both women suffered from comorbid panic disorder that was successfully treated with clomipramine (150 mg/day), with no effect on compulsive buying. To treat CBD, behavioral therapy was conducted with exposure to external cues and response prevention techniques (i.e., walking around street markets where buying attacks used to occur, touching the objects but not buying anything, first accompanied, then alone). Bernik et al. reported that after 3 to 4 weeks of daily exercises, distress and compulsive buying disappeared.

In recent years, several group cognitive-behavioral therapy (CBT) manuals have been published in a variety of languages (Benson & Gengler, 2004; Damon, 1988; Mitchell, 2010; Müller et al., 2008). Although psychotherapy research on this topic remains limited, CBT has been shown to be helpful in two randomized, controlled studies comparing the efficacy of group CBT to that of a waiting list control (Mitchell et al., 2006; Mueller et al., 2008). Both studies have tested the efficacy of the CBT program described in detail by Mitchell (2010). This group therapy consists of 12 weekly sessions and specifically aims to interrupt and control the compulsive buying habits, to identify and restructure maladaptive thoughts and feelings associated with compulsive buying, and to establish healthy purchasing patterns. Treatment interventions focus on delineating factors that maintain the uncontrolled buying episodes and strategies for controlling shopping and buying. In addition, more general sessions on self-esteem, problem solving, and stress management are included.

Mitchell and colleagues (2006) conducted a pilot trial with female adults with current compulsive buying problems; 28 individuals were assigned to receive active treatment and 11 to the waiting list condition. The findings at the end of treatment showed a substantial reduction in the number of compulsive buying episodes and time spent buying, as well as improved scores on the YBOCS-SV and the CBS in the CBT group. The significant improvement was maintained at 6-month follow-up.

The results of the German replication study (Mueller et al., 2008) supported Mitchell and colleagues' findings. In the German psychotherapy study, 51 women and 9 men with current CBD were enrolled. Thirty-one participants were subsequently randomly assigned to the CBT group and 29 to the waiting list condition. The analyses of the primary outcome variables on the CBS, the YBOCS-SV, and the German Compulsive Buying Measurement Scale found that CBT, compared with the waiting list condition, resulted in a reduction of compulsive buying behavior. Predictor analysis showed that poorer attendance at the group therapy sessions and higher pretreatment compulsive hoarding traits, as measured with the Saving Inventory-Revised (Frost et al., 2004), were significant predictors of a poor outcome (Mueller et al., 2008).

Although the principal disorder was CBD, in the U.S. as well as in the German study, a broad range of comorbid psychiatric disorders were found. The large number of concurrent Axis I disorders was consistent with previous findings (Black et al., 2000; Mitchell et al., 2002; Ninan et al., 2000; Schlosser et al., 1994). Since the typical treatment-seeking patient with CBD suffers from comorbid psychopathology, the inclusion of patients with concurrent psychopathology increased the external validity of these two psychotherapy studies (Rothwell, 2005). Further treatment research is needed to examine specific psychotherapeutic effects and to develop subtyping strategies to account for individual comorbid psychopathology (e.g. compulsive hoarding).

In addition, self-help groups, financial counseling, and couples' counseling may be helpful to individuals with CBD. Several self-help books for CBD are already available (Arenson, 1991; Benson, 2001, 2008; Catalono & Sonenberg, 1993; Wesson, 1991). In general, it should be noted that there is a need to evaluate the efficacy and effectiveness of bibliotherapy.

Cognitive-Behavioral Psychotherapy Strategies

Since there is evidence that CBT may be effective, the following sections describe the main cognitive-behavioral strategies that are helpful in the treatment of CBD.

SELF-MONITORING: PURCHASING RECORDS

Self-monitoring is considered essential in precisely describing the frequency of and situational circumstances involved in shopping episodes. Patients should be asked to complete daily purchasing records. The self-observation requires patients' active cooperation and may represent the first step in changing excessive buying behavior. Purchasing records can help develop a clearer picture of compulsive buying antecedents and consequences and allow the clinician to track progress in terms of severity and frequency of buying/shopping episodes, as well as intensity of urges to buy. Sometimes simply the systematic filling out of purchasing records leads to a reduction of purchasing attacks, at least temporarily. Table 30.2 shows an example of a completed purchasing record.

It is important to note that for individuals with CBD, it can be difficult to differentiate between appropriate (normal) and inappropriate (compulsive/pathological) purchases. Most persons with CBD tend to minimize and justify their impulsive purchases. For example, one patient did not recognize that the monthly purchasing of completely new decorations for her apartment was inappropriate. Another patient justified the purchase of 10 gardening books with overlapping contents as necessary in that she might, in the future, want to begin an education in gardening.

MOTIVATION

Therapeutic progress requires that clients are ready to change. To clarify the motivation to change, individuals with CBD should examine the benefits of normalizing their buying behavior. At the same time, it is important to consider personal resources and weaknesses. The pros and cons of normalizing the buying behavior must be discussed at the beginning of treatment. Often CBD has extremely negative social and financial consequences for the affected persons and their relatives. Treatment-seeking patients with CBD are mostly externally motivated to change (e.g. because of severe conflicts with their spouses and huge debts). Furthermore,

Table 30.2 Purchasing Record

Date	Time	Items	Cost ($)	Thoughts	Feelings
10/05	4:00 p.m.	Shoes for my son	80	Pricey, but he needs new shoes	Pleasure
10/06	6:00 .pm.	Underwear	100	Nice	First proud, then remorseful
10/08	3:00 p.m.	Shirts, jeans	100	I really need the jeans, I liked the T-shirts	Confused
10/09	6:00 p.m.	Makeup, perfume	40	Was on sale	Happy
10/10	4:00 p.m.	Shirts, jeans, gifts	250	Nice jeans, gifts for a potential future occasion	First felt happy, then was confused, felt sad, guilty
10/11	2:00 p.m.	6 pairs of slippers DVDs 2 sets of glasswear Wii game for my son	100 30 25,- 60	Cannot decide, slippers are on sale, want it, don't really need that, but like it so much Son will be happy	First happy, excited, then guilty, ashamed
10/12	10:00 p.m. (online)	Wii controller for my son Wii games for my son	200	Can't really afford them, but want it Want to please my son	Restless, irritable, after ordering very guilty and remorseful, anxious about the debts

compulsive buying can be used as a short-term coping strategy to avoid negative mood states and conflicts. Individuals with CBD should know what to expect to as they modify their buying habits and consider whether they want and are able to invest in this change. According to Miller and Rollnick (2000), the motivation to change can be influenced by the therapist's behavior (e.g., supportive and empathetic styles of intervention combined with an emphasis on the personal responsibility of the client for change, active listening, and open-ended questions enhancing the patient's perceived self-efficacy). Table 30.3 summarizes some reasons for and against changing unhealthy buying habits that were expressed by participants in a therapy group.

FUNCTIONAL ANALYSES

Using a model of short-term positive and negative reinforcement, compulsive buying episodes are used to escape from conflicts, to relieve negative mood states, or simply to reduce boredom. Compulsive buying disorder is maintained by its short-term positive consequences, whereas the negative consequences are often not considered. It is important that patients identify specific individualized cues that trigger their buying episodes and the short- and long-term consequences of their problem buying behavior. Patients should be encouraged to recognize their behavioral patterns, thoughts, and feelings associated with compulsive buying.

STIMULUS CONTROL

Patients can develop specific strategies to deal with environmental buying triggers identified in their functional analysis. Stimulus control strategies are designed to rearrange environmental cues for compulsive buying by encouraging the individual to avoid the buying cue entirely (e.g., discard catalogs/sale advertisements), restrict the stimulus field (e.g., shop only in specific stores), or strengthen cues for desired alternative behavior (e.g., spend more time with friends). It is also important that healthy buying behavior is increased and rewarded during treatment. Furthermore, techniques of internal stimulus control (e.g., internal monologues) are helpful. In addition to the physical avoidance of compulsive buying triggers, the responses to cues can be changed through the delay of response and the implementation of alternative behaviors.

ALTERNATIVE BEHAVIORS

One way to decrease compulsive buying is to develop alternative behaviors in which to engage instead of going shopping. Patients should schedule realistic positive activities to engage in during future high-risk situations identified in their functional analysis (long-term plans) or plan alternative behaviors for moments when they feel shopping urges (short-term plans). It is important to establish an individualized list of alternative behaviors for both types of situations.

Table 30.3 Reasons For and Against Changing Unhealthy Buying Habits

Pros	Cons
Excessive debt, no financial security	Ability to buy whatever and whenever I want
Conflict at home	Provides some security to me, self-esteem builder
No space at home, clutter	Escape from problems and bad moods
Remorse, loss of control	Get pleasure, entertainment if feeling lonely, void filler
Legal problems	Relief from tension, stress, anger, sadness, boredom
Anxious about losing my job	Self-present
Lies, hiding, no openness and honesty	Enjoy the salesperson's attention and compliments
Worries about daughter, who also started to buy compulsively	Gifts are a way to please to others
Depression, guilt, shame	Social reinforcement for appearing attractive, well off
Social withdrawal	Distraction from aversive tasks, release, time-out

COGNITIVE RESTRUCTURING

Cognitive techniques are described in detail in many textbooks (Beck et al., 1987). Strategies for eliciting and testing dysfunctional thoughts that become associated with shopping and buying are an essential element in the psychotherapeutic treatment of individuals with CBD. Patients are encouraged to become aware of the maladaptive styles of thinking in which they engage that may lead to problematic shopping responses and to further question, test, and modify these thoughts. For example, patients are asked to keep records of dysfunctional thoughts in which they record the emotions and automatic thoughts that occur in buying/shopping situations. Patients are further taught to develop more rational responses to their maladaptive thoughts that trigger compulsive buying episodes and to record them in the appropriate column (see Table 30.4).

BEHAVIORAL CHAINS

Most of the time, problem buying behavior consists of a series of components in which each aspect represents a link in a behavioral chain. Figure 30.1 presents an example of such a chain. It is important to obtain a description of the events co-occuring with the onset of the compulsive buying attack. Becoming aware of such events may be difficult. Specifying an initial prompting event and the series of cues, thoughts, and feelings that trigger the problematic buying episode creates an opportunity to break the chain early in the cycle and to avoid compulsive buying behavior.

EXPOSURE

In learning to control urges to buy impulsively, it is important that individuals with CBD learn to expose themselves to high-risk buying situations and to avoid buying in those situations. This is important because they will periodically be in situations where urges and opportunities for compulsive buying arise. Exposure and ritual prevention techniques have been found to be quite effective in the treatment of other disorders and are described in many textbooks (Craske & Barlow, 2008; Foa & Kozak, 1996).

MONEY MANAGEMENT

Materialistic values, maladaptive patterns of beliefs about money, and deficits in money management are typical in individuals with CBD (Dittmar, 2005; Rose, 2007; Mueller et al., 2010). Due to the suggested importance of psychological and social factors, as well as consumerism and attitudes toward money, treatment should focus on the improvement of money management as appropriate. Noting that credit card use has a moderating effect on compulsive buying and can provoke excessive spending behavior (Raghubir, 2008; Roberts & Jones, 2001),

Table 30.4 Cognitive Restructuring

Cues	Responses	Revised Responses	Consequences	Revised Consequences
Invitation to a birthday party	*THOUGHTS* There will be only academics there. At least I should look nice. I need new clothes.	*THOUGHTS-REV* I'm not less intelligent than the other guests. I have enough nice clothes in my closet. Don't need new ones.	*SHORT -TERM* Excited about the chance to buy something new. *LONG -TERM* Again failed. More debts. Angry with myself and desperate.	*SHORT–TERM-REV* My thoughts and worries about invitation and potential shopping decrease. *LONG–TERM-REV* I can manage these situations. Proud.
	FEELINGS I feel self-conscious, a little anxious, ashamed.	*FEELINGS-REV* Still a little irritable, but feel better, calm.		
	BEHAVIORS I buy a completely new outfit: new pants, blouse, shoes, jewelry.	*BEHAVIORS-REV* Wear something from my closet.		

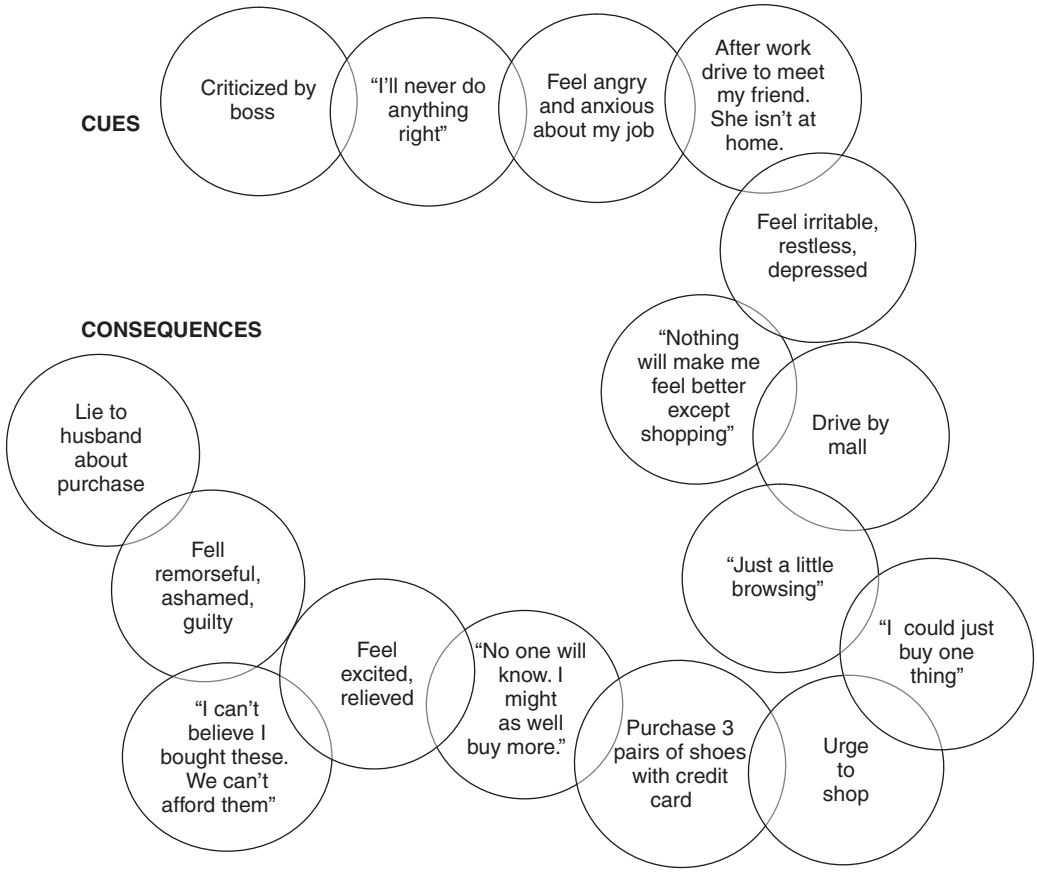

Fig. 30.1 Behavioral chain.

the closing of credit card accounts may be helpful to limit excessive purchases. Patients with CBD should be encouraged to give up their credit cards and to use cash or debit cards instead. In addition, recommendations for appropriate money management should be discussed, including the operationalization of appropriate consumer habits.

COMPULSIVE HOARDING

A strong association between compulsive hoarding and compulsive buying has been reported (Frost et al., 2002). The most visible symptom of compulsive hoarding is clutter. People with compulsive hoarding are unable to discard the purchased items. Acquisition has been defined as a pattern of compulsive hoarding including both the acquisition of free items and compulsive buying (Frost et al., 2009). Individuals with CBD often do not use the things they buy, and many of them do not even unwrap the items or remove the tags; instead, they store or hoard them. In a treatment-seeking sample of individuals with compulsive buying, the severity of hoarding symptoms was associated with the severity of compulsive buying (Mueller et al., 2007). Furthermore, a psychotherapy treatment study with patients with CBD has reported that those with hoarding symptoms were significantly less likely to respond to CBT than nonhoarding patients (Mueller et al., 2008). Because hoarding patients with CBD might be embarrassed to admit their hoarding behavior, especially the amount of clutter in their homes, there is a need for screening of compulsive hoarding and specific treatment interventions to reduce hoarding behavior that focus not only on acquisition but also on clutter and difficulty discarding items.

GROUP VERSUS INDIVIDUAL THERAPY

Based on our clinical experience with patients with CBD, group treatment may be as effective as or more effective than individual therapy. Individuals with compulsive buying are mostly embarrassed about their buying behavior and are used to hiding their spending patterns from friends, family members, and therapists. In the CBD specific group, many of them first have the heart to open up about their

excessive shopping and buying, the extent of their preoccupation, and the consequences. Furthermore, many individuals with CBD are prone to rationalize their buying attacks. The understanding and feedback of other people with the same inappropriate behaviors and thoughts stimulate them to overcome this problem. The strict focus on normalization of spending patterns associated with group cohesion and support by other group members may motivate patients to modify their spending habits.

Summary

Compulsive buying disorder is defined as frequent buying of more than can be afforded and frequent buying of items that are not needed. The buying impulses and behaviors cause marked distress, significantly interfere with social or occupational functioning, and result in interpersonal difficulties and financial problems. Compulsive buying disorder is associated with significant psychiatric comorbidity, particularly mood and anxiety disorders, compulsive hoarding, and personality disorders. It is currently conceptualized as an "impulse control disorder not otherwise specified." Compulsive buying disorder affects a significant percentage of the general population, but it has received much less attention with regard to research and training providers in its assessment and treatment compared to other psychiatric disorders with similar prevalence rates. Formal diagnostic criteria have been put forth, and there are a variety of assessment instruments for evaluating compulsive buying behavior with both questionnaire and interview formats.

Based on the literature to date, it appears that disorder-specific CBT has been most successful in treating CBD. Cognitive-behavioral treatment should include daily purchasing records, functional analysis, stimulus control strategies, increased use of alternative behaviors, identification of behavioral chains, cognitive restructuring, improvement of money management, and exposure practice. Treatment providers should also be aware that mood disorders, anxiety disorders, and compulsive hoarding often accompany CBD and complicate its treatment; these problems should be addressed within the treatment paradigm.

Future Directions

Further research is needed to test the efficacy of other methods of treatment, especially pharmaceutical approaches. With regard to psychotherapeutic treatment, psychiatric comorbidity should be taken into account in tailoring treatment strategies. A subtyping approach might be useful in determining treatment needs.

Further research should also focus on barriers to motivation for change and treatment compliance among individuals with compulsive buying, as well as factors associated with both positive and negative treatment outcomes.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Arenson, G. (1991). Born to Spend: How to Overcome Compulsive Spending. Blue Ridge Summit, PA: Tab Books.

Beck, A., Rush, J., & Shaw, B. (1987). *Cognitive therapy of depression*. New York: Guilford Press.

Benson, A. L. (2000). *I shop, therefore I am: Compulsive buying and the search for self*. New York: Jason Aronson.

Benson, A. L. (2008). *To buy or not to buy: Why we overshop and how to stop*. New York: Trumpeter.

Benson A. L., & Gengler M. (2004). Treating compulsive buying. In R. Coombs (Ed.), *Addictive disorders: A practical handbook* (pp. 451–491). New York: Wiley.

Bernik, M. A., Akerman, D., Amaral, J. A., & Braun, R. C. (1996). Cue exposure in compulsive buying. *Journal of Clinical Psychiatry*, *57*, 90.

Black, D. W. (2007). A review of compulsive buying disorder. *World Psychiatry*, *6*, 14–18.

Black, D. W., Gabel, J., Hansen, J., & Schlosser, S. (2000). A double-blind comparison of fluvoxamine versus placebo in the treatment of compulsive buying disorder. *Annals of Clinical Psychiatry*, *12*, 205–211.

Black, D. W., Monahan, P., & Gabel, J. (1997). Fluvoxamine in the treatment of compulsive buying. *Journal of Clinical Psychiatry*, *58*, 159–163.

Bleuler, E. (1983). *Lehrbuch der Psychiatrie*. Berlin: Springer.

Bullock, K., & Koran, L. (2003). Psychopharmacology of compulsive buying. *Drugs of Today*, *39*, 695–700.

Catalano, E. M., & Sonenberg, N. (1993). *Consuming passions: Help for compulsive shoppers.* Oakland, CA: New Harbinger Publications.

Christenson, G. A., Faber, R. J., de Zwaan, M., Raymond, M. C., Specker, S. M., Ekern, M. D., et al. (1994). Compulsive buying: Descriptive characteristics and psychiatric comorbidity. *Journal of Clinical Psychiatry, 55*, 5–11.

Craske, M. G., & Barlow, D. H. (2008). Panic disorder and agoraphobia. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders: A step-by-step treatment manual* (pp. 1–64). New York: Guilford Press.

Damon, J. E. (1988). *Shopaholics: Serious help for addicted spenders*. Los Angeles: Price Stein Sloan.

Dittmar, H. (2005). Compulsive buying a growing concern? An examination of gender, age, and endorsement of materialistic values as predictors. *British Journal of Psychology*, *96*, 467–491.

Edwards, E. A. (1993). Development of a new scale for measuring compulsive buying behavior. *Financial Counseling and Planning*, *4*, 67–84.

Faber, R. J., & O'Guinn, T. C. (1992). A clinical screener for compulsive buying. *Journal of Consumer Research*, *19*, 459–469.

First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. (2002). *Structured clinical interview for DSM-IV-TR axis I disorders, research version (SCID-I-RV).* New York: Biometrics Research, New York State Psychiatric Institute.

Foa, E. B., & Kozak, M. J. (1996). Psychological treatment for obsessive compulsive disorder. In M. R. Mavissakalian & R. P. Prien (Eds.), *Long-term treatment of anxiety disorders* (pp. 285–309). Washington, DC: American Psychiatric Press.

Frost, R. O., Steketee, G., & Grisham, J. (2004). Measurement of compulsive hoarding: Saving Inventory-Revised. *Behaviour Research and Therapy*, *42*, 1163–1182.

Frost, R. O., Steketee, G., & Williams, L. (2002). Compulsive buying, compulsive hoarding, and obsessive-compulsive disorder. *Behavior Therapy*, *33*, 201–2002.

Frost, R. O., Tolin, D. F., Steketee, G., Fitch, K. E., & Selbo-Bruns, A. (2009). Excessive acquisition in hoarding. *Journal of Anxiety Disorders*, *23*, 632–639.

Goodmann, W. K., Price, L. H., Rasmussen, S. A., Mazure, C., Fleischmann, R. L., Hill C. L., et al. (1989). The Yale-Brown Obsessive-Compulsive Scale I: Development, use, and reliability. *Archives of General Psychiatry*, *46*, 1006–1011.

Grant, J. E. (2003). Three cases of compulsive buying treated with naltrexone. *International Journal of Psychiatry in Clinical Practice*, *7*, 223–225.

Grant, J. E., Kim, S. W., & Hartman, B. K. (2008). A double-blind, placebo-controlled study of the opiate antagonist naltrexone in the treatment of pathological gambling urges. *Journal of Clinical Psychiatry*, *69*, 783–789.

Grant, J. E., Kim, S. W., & Odlaug, B. L. (2009). A double-blind, placebo-controlled study of the opiate antagonist, naltrexone, in the treatment of kleptomania. *Biological Psychiatry*, *65*, 600–606.

Grant, J. E., Levine, L., Kim, D., & Potenza, M. N. (2005). Impulse control disorders in adult psychiatric inpatients. *American Journal of Psychiatry*, *162*, 2184–2188.

Grant, J. E., Potenza, M. N., Hollander, E., Cunningham-Williams, R., Nurminen, T., Smits, G., et al. (2006). Multicenter investigation of the opioid antagonist nalmefene in the treatment of pathological gambling. *American Journal of Psychiatry*, *163*, 303–312.

Kim, S. W. (1998). Opioid antagonists in the treatment of impulse-control disorders. *Journal of Clinical Psychiatry*, *59*, 159–164.

Koran, L. M., Aboujaoude, E. N., Solvason, B., Gamel, N. N., & Smit, E. H. (2007). Escitalopram for compulsive buying disorder: A double-blind discontinuation study. *Journal of Clinical Psychopharmacology*, *27*, 225–227.

Koran, L. M., Bullok, K. D., Hartston, H. J., Elliott, M. A., & D'Andrea, V. (2002). Citalopram treatment of compulsive shopping: An open-label study. *Journal of Clinical Psychiatry*, *63*, 704–708.

Koran, L. M., Chuong, H. W., Bullock, K. D., & Smith, S. C. (2003). Citalopram for compulsive shopping disorder: An open-label study followed by double-blind discontinuation. *Journal of Clinical Psychiatry*, *64*, 793–798.

Koran, L. M., Faber, R. J., Aboujaoude, E., Large, M., & Serpe, R. T. (2006). Estimated prevalence of compulsive buying behavior in the United States. *American Journal of Psychotherapy*, *163*, 1806–1812.

Kraepelin, E. (1909). Ein Lehrbuch für Studierende und Ärzte. I. Band. Allgemeine. Psychiatrie. Leipzig: Johann Ambrosius Barth.

Krueger, D. W. (1988). On compulsive shopping and spending: A psychodynamic inquiry. *American Journal of Psychotherapy*, *42*, 574–584.

Lawrence, L. (1990). The psychodynamics of the compulsive female shopper. *American Journal of Psychoanalysis*, *50*, 67–70.

Lejoyeux, M., Hourtané, M., & Adès, J. (1995). Compulsive buying and depression. *Journal of Clinical Psychiatry*, *56*, 38.

McElroy, S. L., Keck, P. E., Pope, H. G., Smith, J. M. R., & Strakowski, S. M. (1994). Compulsive buying: A report of 20 cases. *Journal of Clinical Psychiatry*, *55*, 242–248.

McElroy, S. L., Satli, A., Pope, H. G., Keck, P. E., & Hudson, J. J. (1991). Treatment of compulsive shopping with antidepressants: A report of three cases. *Annals of Clinical Psychiatry*, *3*, 199–204.

Miller, W. R., & Rollnick, S. (2000). *Motivational interviewing: Preparing people for change*. New York: Guildford Press.

Mitchell, J. E. (2010). Compulsive buying disorder group treatment manual. In A. Müller and J. E. Mitchell (Eds). *Compulsive buying. Clinical foundations and treatment* (pp. 169–278). New York: Routledge, Taylor & Francis Group.

Mitchell, J. E., Burgard, M., Faber, R., Crosby, R. D., & de Zwaan, M. (2006). Cognitive behavioral therapy for compulsive buying disorder. *Behavioral Research Therapy*, *44*, 1859–1865.

Mitchell, J. E., Redlin, J., Wonderlich, S., Crosby, R., Faber, R., Smyth J., et al. (2002). The relationship between compulsive buying and eating disorder. *International Journal of Eating Disorders*, *32*, 107–111.

Monahan, P., Black, D. W., & Gabel, J. (2006). Reliability and validity of a scale to measure change in persons with compulsive buying. *Psychiatry Research*, *64*, 59–67.

Mueller, A., Mueller, U., Albert, P., Mertens, C., Silbermann, A., Mitchell, J. E., et al. (2007). Compulsive hoarding in a compulsive buying sample. *Behaviour Research Therapy*, *45*, 2754–2763.

Mueller, A., Mueller, U., Silbermann, A., Reinecker, H., Bleich, S., Mitchell, J. E., et al. (2008). A randomized, controlled trial of group cognitive behavioral therapy for compulsive buying disorder: Posttreatment and 6-month follow-up results. *Journal of Clinical Psychiatry*, *69*, 1131–1138.

Mueller, A., Mitchell, J. E., Peterson, L. A., Faber, R. J., Steffen et al. (2010). Depression, materialism, and excessive internet use in relation to compulsive buying. *Comprehensive Psychiatry*, Epub ahead of print. doi:10.1016/j.comppsych.2010.09.001

Müller, A., de Zwaan, M., & Mitchell, J. E. (2008). *Pathologisches Kaufen: Ein kognitiv-verhaltenstherapeutisches Therapiemanual.* Cologne: Deutscher Ärzte-Verlag.

Ninan, P. T. N., McElroy, S., Kane, C. P., Knight, B. T., Casuto, L. S., Rose, S. E., et al. (2000). Placebo-controlled study of fluvoxamine in the treatment of patients with compulsive buying. *Journal of Clinical Psychopharmacology*, *20*, 362–366.

O'Guinn, T. C., & Faber, R. J. (1989). Compulsive buying: A phenomenological exploration. *Journal of Consumer Research*, *16*, 147–157.

Raghubir, P. (2008). Monopoly money: The effect of payment coupling and form on spending behavior. *Journal of Experimental Psychology*, *14*, 213–225.

Ridgway, N. M., Kukar-Kinney, M., & Monroe, K. B. (2008). An expended conceptualization and a new measure of compulsive buying. *Journal of Consumer Research*, *35*, 622–639.

Roberts, J. A., & Jones, E. (2001). Money attitudes, credit card use, and compulsive buying among American college students. *Journal of Consumer Affairs, 35*, 213–240.

Rose, P. (2007). Mediators of the association between narcissism and compulsive buying: The roles of materialism and impulse control. *Psychology of Addictive Behavior*, *21*, 576–581.

Rothwell, P. M. (2005). External validity of randomized controlled trials: "To whom do the results of this trial apply?" *Lancet*, *365*, 82–93.

Schlosser, S., Black, D. W., Repertinger, S., & Freet, D. (1994). Compulsive buying: Demography, phenomenology, and comorbidity in 46 subjects. *General Hospital Psychiatry*, *16*, 205–212.

Spinella, M., Yang, B., & Lester, D. (2007). Development of the Executive Personal Finance Scale. *International Journal of Neuroscience*, *117*, 301–313.

Valence, G., d'Àstous, A., & Fortier, L. (1988). Compulsive buying: Concept and measurement. *Journal of Consumer Policy*, *11*, 419–433.

Villarino, R., Otero-Lopez, J. L., & Castro, R. (2001). *Addiction a la compra: Analysis, evaluaction y tratamiento*. Madrid: Ediciones Piramide.

Wesson, C. (1991). *Women who shop too much: Overcoming the urge to splurge*. New York: St. Martin's Press.

Winestine, M. C. (1985). Compulsive shopping as a derivative of a childhood seduction. *Psychoanalytic Quarterly*, *54*, 70–72.

World Health Organization. (1994). The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines. Geneva: Author.

This page intentionally left blank