CHAPTER

9

Stress and Impulsive Behaviors

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This chapter explores the complex relationship between stress, impulsivity, and substance use disorders (SUDs). A representative sample of individual impulse control disorders is reviewed, to include pathological gambling (PG), trichotillomania (TTM), and intermittent explosive disorder (IED) and categorized as problems of reward seeking (PG), habit (TTM), and a lack of premeditation (IED). Factors that mediate the disorders include tension reduction and affective regulation and can be further complicated by, and hold many similarities to, SUDs. Individuals with impulse control disorders can engage in the problem behavior as a means to immediately reduce tension or urge intensity and also seek to regulate affective states, particularly negative states (e.g., depression, boredom, anxiety), which there is low tolerance for. Individuals with impulse control disorders suffer numerous negative psychosocial stressors as a result of their acting on the impulses. Impulsivity, aggression, and sensation seeking are common features of SUD and impulse control disorders (PG, IED) leading to problems in managing either comorbid disorders. Treatment of the differing impulse control problems require multimodal approaches (psychosocial, behavioral, pharmacological) in order to address unique qualities of each problem. Promising psychological treatment approaches include components of cognitive behavioral therapy (CBT) such as cognitive retraining, behavioral modification, cue exposure, and self-control training, requiring further research.

I. INTRODUCTION

Impulsivity has been defined as a predisposition toward rapid, unplanned reactions to either internal or external stimuli without regard for negative consequences (Moeller, Barratt, Dougherty, Schmitz, and Swann, 2001), but this definition may not adequately characterize the complex range of behaviors regarded as impulsive. Although certain disorders are formally classified as impulse control disorders, impulsivity is a key element of many psychiatric disorders (for example, substance use disorders, bipolar disorder, personality disorders, attention deficit hyperactivity disorder). In *DSM-IV-TR*, the category of Impulse Control Disorders Not

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Elsewhere Classified currently includes pathological gambling, trichotillomania, intermittent explosive disorder, kleptomania, and pyromania (American Psychiatric Association, 2000). Although all of these disorders are considered impulsive behaviors, the extent to which these disorders share clinical, genetic, phenomenological, and biological features is incompletely understood.

Research suggests that there is significant heterogeneity within the impulse control disorders, and the concept of "impulsivity" may include distinct subtypes of impulsive behaviors. Because rigorous research is limited on most impulse control disorders, this chapter will focus on pathological gambling, trichotillomania, and intermittent explosive disorder, three impulse control disorders that have received significant research attention and reflect the heterogeneity of these impulsive behaviors. In addition, these disorders represent three distinct, yet not absolute, subtypes of impulsivity: reward-seeking behavior (pathological gambling), habitual behavior (trichotillomania), and behavior characterized by a lack of premeditation (intermittent explosive disorder). We recognized that the subtypes do not completely define the selected or nonselected impulse control problems, with some overlap. This chapter will examine the complex relationship of these disorders to stress, tension reduction, affective regulation, and substance use (drug and alcohol) disorders (SUDs).

II. IMPULSE CONTROL AND SUBSTANCE USE DISORDERS

Individuals with impulse control disorders are found to be at an increased risk for substance use disorders (SUDs). Studies (Lejoyeux, Feuche, Solomon, and Andes, 1999) have reported prevalence rates of 38% for individuals with impulse control disorders receiving in-patient treatment for SUDs. Individuals with early onset alcoholism relative to late onset have reported higher levels of impulsivity and sensation seeking, as well as heightened symptom severity ratings for SUDs (Dom, Hulstijn, and Sabbe, 2006). Of the impulse control disorders reviewed in this chapter, pathological gambling has been reported as having the highest rate of comorbid SUDs (Lejoyeux et al., 1999). The constructs of impulsivity, risk taking, and novelty seeking, which are characteristic of the impulse control disorders, among other psychiatric disorders, can contribute to the initiation of drug and alcohol use, as well as transitions from initial use to regular use to addiction (Kreek, Nielsen, Butelman, and LaForge, 2005). Kreek and colleagues (2005) argued that impulsivity and risk taking contribute most to the initiation of drug use and the progression of regular drug use. Subsequent changes once the addictive process is initiated are attributed to substantial changes in the brain as a result of repeated exposure to drug or alcohol abuse. We will explore in this chapter the commonalities between SUDs and impulse control disorders, as well as abnormal stress response. Table 9-1, on page 194; provides a summary of the complex relationships between the impulse control disorders reviewed and stress.

III. REWARD-SEEKING BEHAVIOR: PATHOLOGICAL GAMBLING (PG)

How does tension reduction and affective regulation relate to gambling? Is stress a driving factor that contributes to or precipitates the impulse to gamble? Individuals who struggle with PG are preoccupied with wagering, and the amount of the wager increases in order for the person to achieve a greater sense of excitement. When the individual attempts to refrain from gambling, or even reduce the amount of gambling, he/she may become irritable. In many individuals, gambling becomes a means of escaping from negative moods

CASE VIGNETTE

B.G., a 40-year-old married Caucasian male, reports the following symptoms. "I come home from work and I feel stressed out and have this need to blow off some steam. When I gamble it gives me an initial rush and I don't really think about anything while gambling except gambling and I don't consider the consequences of my gambling. All I want to do is forget about my other problems but the gambling usually ends in me feeling worse rather than happy regardless of whether I win or lose. I have had several times in my life when I have felt depressed and my family has commented that my depression has worsened lately. My alcohol use and gambling tend to feed off each other, with one worsening the other. My gambling has gotten so out of control that I am currently in serious financial trouble, taking a second mortgage, gambling away my son's college fund, problems at work, and most importantly, my wife threatening divorce. The problems that have resulted from my gambling seem to far out weigh any benefit that I have received, yet I can't stop gambling."

(e.g., anxiety, depression). Whiteside and Lynam (2001) made an effort to broadly define impulse control problems, with a sense of urgency involving individuals experiencing strong impulses under conditions of negative affect. Sensation seeking is one additional aspect of impulse control problems, including PG, which involves the pursuit of activities that are exciting and being open to new experiences that may or may not be dangerous (Whiteside and Lynam, 2001). As seen in the case of BG, individuals with PG may also report co-occurring alcohol and drug use problems, with the problems becoming mutually reinforcing. PG has been found to have many similarities with substance use disorders (e.g., tolerance, telescoping; Taveres, Zilberman, Beites, and Gentil, 2001), as well as high rates of comorbidity with substance use disorders (Cunningham-Williams, Cottler, Compton, and Spitznagel, 1998), prompting Potenza, Fiellin, Heninger, Rounsaville, and Mazurc (2002) to categorize gambling as an addictive disorder.

A. Tension Reduction

An individual's need for tension reduction has been described as a mediating factor in substance addiction and a possible contributing characteristic of problem gambling. A study of New Zealand university students found that individuals who suffered from problem gambling were more depressed and impulsive compared to their nonproblem gambling peers (Clarke, 2004). Motivations reported by problem gamblers in this study were reducing tension, experiencing guilt, and feeling compelled to prove themselves to others. Those students with problem gambling were unable to recognize a connection between their behavior and gambling outcomes, reporting an external versus internal locus of control (Clarke, 2004). The need for tension reduction may be better conceptualized, in some pathological gamblers, as an urge to engage in the problem behavior, which can be reinforcing. Individuals experience a "craving" or an urge to engage in gambling, and as seen in cases of SUD, engaging in the behavior may be considered the only alternative to alleviate that tension. We will review physiological and neurological studies to better understand the changes that occur during gambling and similarities with SUD.

Neuroimaging studies have reported abnormalities in the brain functioning of pathological gamblers (Goldstein and Carlton, 1988; Potenza et al., 2003;

TABLE 9-1 Impulse Control Disorders and Moderating Variables

Moderators	PG (Reward)	TTM (Habit)	IED (Lack of Premeditation)
Tension Reduction	Gambling urge =/> SUD related urge. Tension/stress change pre, during, postgamble. Goal = Reward (e.g., winning, chasing losses and avoidance of distress). Autonomic arousal. Delayed hemispheric activation.	Tension increase and release not characteristic of all TTM cases. Decreased pain sensitivity in hair-pulling site. Poor tolerance for discomfort. Goal = Reduction of distress. Urge unique to location/ type of hair pulled. Autonomic arousal.	Unplanned, uncontrolled aggressive outbursts (tension), followed by remorse + tension reduction. Goal = tension reduction. High autonomic arousal. Reduced central serotonin function.
Affective Regulation	Abnormal reactivity to stressors. Comorbid mood and affective disorders. Gambling negatively reinforced (avoid/escape negative affect). Risk-taking temperament.	Abnormal reactivity to stressors. Boredom significant motivator. Comorbid mood and affective disorders. Affective experience serves as cue for and reinforces hair pulling (reduces unpleasant affective state).	Rapid onset of brief, "manic-like" symptoms followed by depressed mood, fatigue, and sometimes pleasure. Comorbid mood and affective disorders. Poor distress tolerance.
Treatment	Revise erroneous gambling cognitions. Motivational interview. Modify environmental cues. Cue Exposure. Coping Skills training. SSRI Opioid antagonist (Naltrexone)	Behavioral modification (habit reversal). Modify environmental cues. Coping Skills Training. Cognitive Therapy. SSRI (Fluoxetine) Opioid antagonist (Naltraxone)	Limited CBT research. Population reluctant to seek treatment. Cue exposure, relaxation training, and cognitive therapy +. Future research: mindfulness and self-control training. SSRI (Fluoxetine) + Valproate semisodium +.
SUD	Comorbid Alcohol (73%) Comorbid Drug (38%)	Comorbid SUD (22%)	Comorbid SUD (48%)

PG = pathological gambling, TTM = trichotillomania, IED = intermittent explosive disorder, SUD = substance use disorder, CBT = cognitive behavior therapy, (+) = significant research outcomes.

Stojanov et al., 2003). Goldstein and Carlton (1988) through EEG studies found significant delays in hemispheric activation, implying inflexibility in brain shifting ability or perseverative tendencies during gambling tasks regardless of negative consequences. Stojanov and colleagues (2003) found evidence of increased arousal levels or startle response, while Potenza and colleagues (2003) reported increased activation in the right middle frontal gyrus during a gambling scenario. Potenza and colleagues concluded their findings were evidence that pathological gamblers have similar brain pathways as those experiencing drug cravings, or the need for tension reduction. The experience of stress before, during, and after problem gambling has been considered a "moderating factor." Studies have investigated "stress related" changes in problem gamblers, finding autonomic arousal and

immune system changes before, during, and after gambling (Brown, Rodda, and Phillips, 2004; Shinohara, Yanagisawa, and Kagota, 1999); higher levels of noradrenergic metabolites in males with PG (Roy, Adinoff, and Roehrich, 1988); and higher epinephrine and cortisol levels and blood pressure differences on days focused on gambling (Schmitt, Harrison, and Spargo, 1998). Additional physiological studies of pathological gamblers have found higher skin conductance levels during gambling (Sharpe, Tarrier, Schotte, and Spence, 1995); lower diastolic blood pressure throughout a gambling task (Carrol and Huxley, 1994); and higher heart rates (Blanchard, Wulfert, Freidenberg, and Malta, 2000). Another conclusion proposed by Goudriaan, Oosterlaan, de Beurs, and Van den Brink (2004) for differences in brain functioning was an abnormal reaction to stressors, with pathological gamblers presenting with different response patterns compared to nonpathological gamblers.

Individuals who develop PG appear to have deficits in shifting attention and have a diminished capacity to consider the negative consequences of their actions. Their focus is solely on the short-term urge to gamble, focusing only on the immediate consequences—that is, the need for tension reduction. The need for tension reduction or urge to gamble is experienced once the decision has been made to gamble, which may have been cued by any number of internal or external stimuli. PGs have been shown to have a greater "craving" to gamble or need for tension reduction, and are more impulsive in comparison to individuals with SUD (Castellani and Rugle, 1995; Tavares, Zilberman, Hodgins, and el-Guebaly, 2005). As noted by Goudrian and colleagues (2004), individuals who develop PG problems appear to have abnormal reactions to stress, which is also characteristic of individuals with comorbid mood, affective disorders, and SUDs. We will review problematic affective states resulting from an inability to cope with stressors, which can potentially initiate the urge to gamble.

B. Affective Regulation

Pathological gamblers have demonstrated high rates of co-occurring mood disorders, and this may suggest that the gambling behavior is associated with impairment in affective regulation. An individual's inability to cope with significant life stressors or a lack of the requisite coping skills can lead to the development of affective disorders. Individuals receiving inpatient psychiatric care (McCormick, Russo, Ramirez, et al., 1984) with PG have exhibited elevated rates of co-occurring major depressive disorder (76%) and hypomanic episodes, while outpatient populations have also reported comorbid major depressive disorder (28%) as well as elevated rates of anxiety (28%) and bipolar (24%) disorders (Linden, Pope, and Jonas, 1986). There has been limited longitudinal study as to the order of onset of these comorbid disorders. As noted by Petry, Stinson, and Grant (2005), anxiety and mood disorders may predispose individuals to develop gambling problems, or PG may lead to the development of anxiety disorders. The national epidemiological survey on alcohol and related conditions found that individuals with PG had elevated rates of comorbid alcohol use disorders (73%), any drug use disorder (38.10%), any mood disorder (49.62%), any anxiety disorder (41.30%), and any personality disorder (60.82%). The high comorbid rates with the aforementioned psychiatric and SUDs may imply diagnostic overlap. PG can be considered an escape or avoidance from problems or a means of relieving an aversive affective state, which is characteristic of mood, anxiety, and SUD. Petry and colleagues reported one gender difference, with women reporting higher rates of generalized anxiety and major depressive disorder, which could imply that women have an increased likelihood to develop PG in an effort to alleviate anxious and depressed

mood than men (p. 571). Additional studies have reported on negative affective states in pathological gamblers.

Brown et al. (2004) reported greater "negative pre-gambling valence" for problem gamblers, which dramatically decreased if the gambler lost and did not significantly improve after winning. The gambling behavior is thereby negatively reinforced, in avoidance of negative affect or distress (Clarke, 2004). A number of internally mediated factors contribute to problem gambling, while external factors can also contribute substantially. Common risk factors identified for adolescent pathological gamblers that are predictive of this problem behavior include family problems, having conduct problems, addiction to drug or alcohol, and being male (Hardoon, Gupta, and Derevensky, 2004). Individuals experiencing stress from problematic and unsupportive home lives can subsequently lead to maladaptive strategies to reduce negative mood states and manage stress. Impulsivity and emotional distress have been found to influence risk taking in gamblers, especially young gamblers (Martins, Taveres, de Sliva Lobo, Galetti, and Gentil, 2004).

According to the St. Louis Personality, Health, and Lifestyle Study (SLPHL; Cunningham-Williams et al., 2005), factors found to be most predictive of PG included personality traits (high novelty-seeking temperament) and character styles (e.g., low cooperativeness, low self-directedness) indicating immature development, which are characteristics often associated with alcohol and substance use disorders. These findings reflect the fact that, while stress plays a significant role in PG, there is also the influence of having a risk-taking temperament.

SUD and PG both share similar traits, including compulsivity, impaired control, tolerance, and interpersonal problems (Grant, Kushner, and Kim, 2002; Petry et al., 2005). Additional commonalities between PG and alcohol and substance use disorders include novelty-seeking tendencies and stress-related changes before, during, and after engaging in the problem behavior. One potential difference between PG and SUD is the finding that alcoholics use alcohol to deal with negative affect, particularly anxiety, whereas individuals with PG turn to gambling as a way to cope with depressive feelings and a lack of positive experiences in their life (Tavares et al., 2005). This "externalizing problem" appears to be motivated by several internal- and external-mediated variables (e.g., tension reduction, depression, poor family life) that are negatively reinforced. Individuals categorized as pathological gamblers also lack the requisite skills to contend with daily stressors and may tend to actually overreact. As identified in neuroimaging and neuropsychological studies, problem-solving ability can be limited and worsened by perseverative tendencies, with individuals unable to immediately recognize the negative consequences of their behavior.

C. Treatment of Pathological Gambling

We have categorized PG as a reward-seeking behavior, with individuals also exhibiting deficits in stress management. In targeting these two problems, individuals with PG require guidance in the identification of healthy alternatives to reward seeking and stress management. A majority of psychosocial treatment programs for individuals with PG are modeled after addiction programs and include self-help groups, inpatient treatment, and rehabilitation programs. However, programs such as Gamblers Anonymous (GA) have not demonstrated significant efficacy in the treatment of this problem, in isolation. Dropout rates from GA have ranged from 75–90% (Moody, 1990), with 8% reporting gambling abstinence at 1-year follow-up (Brown, 1985).

Cognitive behavioral methods, such as cognitive restructuring, have demonstrated

some benefit in decreasing the frequency of gambling and irrational verbalizations associated with gambling (Hodgins and el-Guebaly, 2004). Others (Russo, Taber, McCormick et al., 1984; Taber, McCormick, Russo, et al., 1987) have suggested that professionally guided, multimodal treatment programs, provided alone or in combination with GA, may be more effective than GA alone. Self-help programs have reported some benefit (Hodgins, Currie, el-Gurbaly, and Peden, 2004). Hodgins and colleagues (2004) randomly assigned subjects (N = 67) to self-help workbook only or workbook and two motivational telephone interventions. At 1 year follow-up, 60% of the subjects in the motivational phone intervention group did not meet criteria for PG. The motivational interviewing appears to have had an effect on individuals inhibiting the urge to gamble and in developing healthy alternatives to gambling.

Pharmacological treatment may also offer significant promise in treating PG. Although the use of SSRIs, such as paroxetine, fluvoxamine, and sertraline for PG, has produced mixed findings (Grant and Potenza, 2004), they may be particularly beneficial for stress reduction in gamblers. Other randomized, placebo-controlled studies have demonstrated possible benefit from opioid antagonists (Grant et al., 2006; Kim et al., 2002) and lithium (Hollander, Pallanti, Allen, Sood, and Rossi, 2005). These medications may be better targeted to reduce reward-seeking behavior in gamblers. Studies of medication treatment, while providing short-term reduction of PG symptoms, have not demonstrated long-term benefits. The majority of the pharmacological studies, like the psychological studies, have failed to include co-occurring disorders and therefore the results may not generalize to the larger population of individuals with PG.

Future controlled-treatment studies for PG are needed in order to explore strategies (pharmacological and psychosocial) that effect long-term change. We have discussed individuals with this impulse control problem experiencing an urge to engage in the problem behaviors, as seen in SUD, which can be mediated by internal and external stressors and/or triggers. Of concern, as reported by Tavares and colleagues (2005), is the fact that individuals with PG report significantly greater cravings compared to individuals with SUD. One possible treatment approach to be explored is cue exposure, which has been used successfully to treat other anxiety disorders (Barlow, 1993). The core idea in CE is that prolonged presentations of the conditioned cues (e.g., sights, sounds, and smells of gambling) in the absence of the naturally evocative experience (e.g., gambling behavior) will eliminate or substantially weaken (i.e., "extinguish" or "habituate") the pathological conditioned reactions (e.g., urge to gamble) that these cues produce (Foa and Kozak, 1986). In terms of CE treatment for PG, Symes and Nicki (1997) reported two cases of PG successfully treated with CE. McConaghy, Blaszczynski, and Frankova, (1991) (also see McConaghy, Armstong, Blaszczynski, and Allock, 1983) reported one of the only randomized controlled trials of CE for PG. They found that CE procedures resulted in a greater percentage of PG cases that demonstrated "controlled" or "ceased" gambling at follow-up than did a control group undergoing relaxation training only. However, the control group actually had a higher proportion of participants that were actually abstinent from gambling at the follow-up. Future controlled studies are needed to determine the efficacy of CE for PG.

Additional risks for relapse in individuals with PG include unstructured time, boredom, and a lack of positive experiences in their lives (Hodgins and el-Guelby, 2004; Tavares et al., 2005). A multimodal approach to this problem would therefore consider training in healthy alternative coping skills that would include immediate and

more enduring rewards, as experienced in gambling.

IV. HABIT BEHAVIOR: TRICHOTILLOMANIA (TTM)

Some impulse control disorders may be better conceptualized as habitual behaviors (for example, trichotillomania, skin picking, and nail biting) given that many individuals with trichotillomania (TTM) often report performing the behaviors without a clear driving force or a lack of premeditation (Whiteside and Lynam, 2001). Habits by definition are considered automatic processes and in the case of TTM, a maladaptive coping strategy to manage negative affect (depression) and serving to reduce tension.

CASE VIGNETTE

S.A. is a 16-year-old female who sought outpatient therapy for treatment of her hair pulling. She reported a depressed mood and significant hair loss. Her problem developed at age 10 when she would pull hair when upset about her parents fighting and when feeling sad, lonely, or bored. She reported that she usually pulled her hair "without thinking" or when under stress related to school, family, and relationship problems. Sometimes, however, she reported a significant urge to pull, with some relief (tension reduction) when she engaged in the behavior. At those times she found the hair pulling enjoyable.

As noted in this case vignette, individuals with TTM can experience a significant urge to engage in the hair-pulling behavior, and the behavior can become conditioned in the sense that it becomes an automatic response to stressors.

A. Tension Reduction

As defined in the *DSM-IV* (American Psychiatric Association, 2000), individuals with TTM experience an increased state of tension prior to pulling hair and a sense of relief when pulling. Azrin and Nunn (1973) characterize TTM as a nervous habit, starting as a normal reaction and becoming a strongly established habit. Rothbaum (1992) described hair pulling as occurring, increasing, and reappearing in conjunction with stress, leading to the conclusion that individuals with this problem would benefit from stress reduction training. Additional research has challenged the *DSM-IV* criteria requiring tension increase and reduction as overly restrictive (Christenson, Makenzie, and Mitchell, 1991). Christenson and colleagues (1991) reported that 17–23% of a sample of individuals diagnosed with TTM did not experience a sense of tension immediately before or while trying to resist hair pulling, or feelings of pleasure, gratification, or relief following hair pulling. Of interest in this study was the finding that each individual's experience of tension increase and subsequent relief was unique to the location of the hair pulled and type of hair pulled (e.g., scalp hair, gray hairs).

There have also been differences noted in the experience of tension and relief, with children and adolescents at times reporting none (Hanna, 1997) in comparison to adults. Hanna (1997) reported the differences in symptom endorsement could be attributed to the cognitive development of the different age groups, with younger children having less awareness of internal states.

The impact of heightened levels of stress on the psychosocial functioning of individuals struggling with TTM has also been examined. Diefenbach, Tolin, Hannan, Crocetto, and Workhunsky (2005) compared a psychiatric control group to individuals diagnosed with TTM and found the TTM group reported lower life

satisfaction, higher levels of distress, and lower levels of self-esteem. The lower levels of self-esteem were related to concerns about appearance, feelings of embarrassment, and frustration with the inability to control the impulse to pull hair. In addition, the majority of the sample (96.4%) reported current problems with negative affect/negative self-evaluations. In terms of life functioning, the TTM group reported lifetime and current problems with grooming, physical health, social interaction, recreational activities, and work productivity (Diefenbach et al., 2005). In this study, the severity of hair loss was the most significant predictor of self-esteem.

B. TTM and SUD

Studies have reported upwards of 80% of individuals with TTM have comorbid *DSM-IV-TR* axis I disorders, with 20% reporting current or past SUDs and/or eating disorders (Christenson et al., 1991). Among alcohol-dependent patients (N = 79), 38% were diagnosed with impulse control disorders, with only one patient being diagnosed with TTM (Lejoyeux et al., 1999). Individuals with TTM do share similar characteristics with other impulse control disorders, but there appears to be less prevalence in having comorbid SUD. Nonetheless, individuals with TTM commonly experience mood and affective disorders, which are commonly reported as highly comorbid in SUD populations. The literature does not, however, reflect high comorbid SUD rates with TTM when TTM is studied independent of mood and affective disorders. As a result, there will not be as many comparisons between individuals with SUD and TTM in the following section.

C. Affective Regulation

Individuals with TTM frequently endorse comorbid *DSM-IV* Axis I disorders, with as much as 82% of an adult clinical sample with TTM (N = 60) meeting criteria (Christenson et al., 1991). Lifetime prevalence rates of this adult sample included 65% for mood disorders, 57% for anxiety disorders, 22% for substance abuse disorders, 20% for eating disorders, and 42% for personality disorders. The presence of co-occurring mood disorders may suggest an association between TTM and impaired affective regulation. Mansueto, Stemberger, Thomas, and Golomb (1997) in construction of a behavioral model of TTM reported the affective experience as an important motivator for hair pulling, serving as both a stimulus cue and a reinforcer of the behavior. An additional variable in studying this problem is the behavioral sequence of the problem and associated emotional states. Stanley, Borden, Mouton, and Breckenridge (1995) found that hair pulling was associated with decreases in tension, boredom, sadness, and anger. Diefenbach, Mouton-Odum, and Stanley (2002) further investigated changes in emotional states across the hair-pulling cycle (pre to during to post), finding significant decreases in boredom across the entire cycle. The identified emotional states were found to act as both stimulus cues and reinforcers for hair pulling (Diefenbach et al., 2002). Additional emotional state changes included significant increases in sadness, guilt, and anger after hair pulling was completed.

Townsley-Stemberger, Mansueto, and Gardner-Carter (2000) found in a group of treatment seeking (N = 67) patients with TTM, marked, day-to-day distress and social impairment due to hair pulling. This treatment-seeking group reported avoidance of interpersonal activities, significant depressed mood, irritability, and relationship problems (Townsley-Stemberger et al., 2000). Simeon and colleagues (1997) surveyed 71 female hair-pullers for self-injurious behaviors. Two TTM groups with and without comorbid self-injurious behaviors were surveyed, with the former reporting more significant history of depression,

suicide attempts, and thoughts of death during self-injurious acts. Of the comorbid self-injurious behaviors, nail biting and skin picking were most related to TTM. In regard to affective cues for hair pulling, the TTM-only group reported more anxiety, diminished motivation, and depression. These findings, however, are from a survey of a national magazine predominantly for women, and the diagnosis of TTM could not be verified using a structured clinical interview. Despite the limitations, Simeon and colleagues' (1997) findings were consistent with previous studies' (Christenson, Ristvedt, and MacKenzie, 1993) finding that subgroups of TTM patient behaviors were cued by negative affect. Christenson and colleagues (1993) found that negative affect and sedentary/contemplative states served as hair-pulling cues. Of interest to this discussion, the negative affective cues included feeling terms (e.g., feeling angry, hurt) and situations associated with negative self-evaluation (e.g., weighing oneself, interpersonal conflicts) (Christenson et al., 1993).

One hypothesis is that hair pulling develops as a habit to cope with stress, and then ironically the behavior results in negative intra- and interpersonal distress. Distress results from having to avoid certain activities due to hair loss, such as public activities, sexual intimacy, and athletic endeavors (Stemberger, Thomas, Mansueto, et al., 2000). Distress also results from the individual's inability to control the hair pulling resulting in lowered self-esteem (Casti, Toner, and Yu, 2000; Stemberger et al., 2000). Once this impulse control habit has developed into TTM, the negative self-evaluative thoughts and negative affect can serve to perpetuate this problem by prompting additional pulling episodes (Franklin, Tolin, and Diefenbach, In press).

D. Treatment of Trichotillomania

The experience of stress or distress in association with TTM has been identified at different stages of the hair-pulling process and is also associated with comorbid mood and affective disorders. We categorized TTM as a problem of habit, or maladaptive habit developed to deal with affective states and stress. Treatment therefore would need to target healthy stress reduction strategies with more enduring tension reduction effects. While tension reduction appears to be a motivating factor in TTM, negative affective states and the experience of "boredom" serve to perpetuate this problem. Behavioral strategies utilized with this problem have been found to be most successful with symptom reduction. Manseuto and colleagues (1997) proposed that functional analysis of the hair-pulling process and accompanying affective states could assist in identification of the most appropriate treatment options. Individuals who experience reductions in boredom as a result of hair pulling may benefit from activity planning or "competing behavioral response" training. In contrast, relaxation skills may be more relevant for individuals who experience tension and anxiety reduction while pulling hair. Individuals who experience negative affective states (e.g., guilt) as a result of hair pulling could benefit from cognitive restructuring and relapse prevention in order to reframe negative cognitions associated with the behavior. In a review of behavioral treatment strategies, Friman, Finney, and Christophersen (1984) reported additional components of successful treatment programs for individuals with TTM, to include hair collection and self-monitoring with therapist, self-imposed consequences, token reinforcement, and self-denial of privileges.

Habit reversal has been identified as the most successful strategy (Azrin, Nunn, and Frantz, 1980) with symptom improvements of 90% in the short term. This strategy includes practicing motor responses that compete with the habit, habit inconvenience review, solicitation of social support, and self-monitoring. Considering the tension reduction component of TTM, Rothbaum

(1992) added the use of deep muscle relaxation to habit-reversal training, as well as thought stopping, cognitive restructuring, and stimulus control (e.g., identification of high-risk situations). Controlled studies to date have reported short-term benefits of cognitive behavioral therapy (CBT; Ninan, Rothbaum, Marsteller, Knight, and Eccard, 2000; van Minnen, Hoogduin, Keijsers, Hellenbrand, and Jan Hendriks, 2003) compared to medication (clomipramine and fluoxetine). Unfortunately, the short-term CBT benefits for TTM have not been maintained in follow-up periods. Lerner, Franklin, Meadows et al. (1998) reported that only 4 of 13 subjects maintained their initial gains at 3.9 years post-treatment from CBT. Similarly, Mouton and Stanley (1996) found that two patients out of six maintained clinically significant gains at 6-month follow-up after participation in habit-reversal training.

Additional study is needed in the use of medications combined with CBT to further evaluate the efficacy of this combined approach. There are few studies examining medications other than serotonin reuptake inhibitors. Naltrexone, an opioid-blocking compound thought to decrease positive reinforcement by preventing the binding of endogenous opiates to relevant receptor sites in the brain, may offer some benefit beyond the antidepressants (Carrion, 1995; Christenson, Crow, and MacKenzie, 1994). Christenson and colleagues (1994) reported significant effects on decreasing TTM symptoms when comparing naltrexone to placebo. In an uncontrolled case study, Carrion (1995) reported the combination of fluoxetine and naltrexone decreased the duration and intensity of hair-pulling episodes, with the individual reporting "little pleasure" from the hair-pulling episodes. The early gains in this case study were maintained during 8 subsequent weeks of treatment and at a 4-month follow-up. The use of naltrexone necessitates further evaluation with larger controlled studies, combined with and compared to the behavioral treatments discussed. The benefit observed from these preliminary naltrexone studies may be related, in part, to individuals with TTM experiencing altered pain sensitivity at the site of pulling (Christenson et al., 1994). For those individuals with TTM that do experience pain, the pain itself could be reinforcing, because of the distraction from negative emotional or physiological states (Christenson and Mansueto, 1999). Ultimately, the goal for individuals with TTM is to learn healthy strategies for regulating negative affect and thereby inhibit the urge to pull.

Future directions in the treatment of TTM may include a combination of pharmacological and psychosocial treatments, as well as the components of CBT proven to be most effective in symptom reduction. Although there is a short-term benefit to CBT approaches, such as habit reversal, there is limited evidence of the long-term benefits. This problem of habit appears to require a multimodal approach to address the different components of TTM, including the pleasure-seeking aspects, need for tension reduction and affective regulation, and the psychosocial deficits (e.g., avoidance of social activities due to hair loss). Future treatment studies for TTM may also focus on the addition of mindfulness/acceptance strategies to traditional CBT approaches (Franklin, Tolin, and Diefenbach, In press) and in comparison to use of hypnosis (Robiner, Edwards, and Christensen, 1999).

V. LACK OF PREMEDITATION: INTERMITTENT EXPLOSIVE DISORDER (IED)

The final subtype of impulse control disorders is behavior characterized by its lack of premeditation. Intermittent explosive disorder (IED) is defined by recurrent, significant outbursts of aggression, often leading to assaultive acts against people or property, which are disproportionate to outside stressors and no better explained

by another psychiatric diagnosis (American Psychiatric Association, 2000). Individuals with IED are prone to aggressive outbursts when under stress. They may perceive the stress as threat, frustration, insult, vulnerability, or any combination of the above. The person is often upset, guilt-laden, and remorseful after the rage-filled episode even though there may be a sense of relief after the aggressive outburst. A defining characteristic of IED is the complete lack of premeditation preceding the behavior, with individuals requiring only a subtle slight to feel provoked (Coccaro and Danehy, 2005).

CASE VIGNETTE

Jake is a 27-year-old male reporting for outpatient treatment for the first time due to his inability to manage his anger and outbursts. He reports a long-standing history of easily losing his temper in spite of illogical provocation. For example, Jake describes how, after getting cut off in traffic, he chased the car and crashed into it. He then approached the vehicle, was verbally aggressive to the driver, and pounded the driver's car window until it cracked. The police came and he was given a citation. After he calmed down, he was extremely embarrassed and remorseful of his actions. He describes anger episodes as "uncontrollable rages" which are never planned but instead "just happen." He reports a type of amnesic dissociative state during the outbursts with only partial memory of the particular details. Due to these outbursts, Jake has had numerous legal problems, and his finances and relationships are quite strained. He is on probation currently and is required to receive treatment.

Coccaro, Schmidt, Samuals, and Nestadt (2004) reported on prevalence rates of individuals with IED in a community-based sample. In addition to *DSM-IV* criteria, Coccaro et al. added the following research criteria: verbal or physical aggression toward people, animals, or property; aggressive acts occurring twice weekly, on average, for 1 month or three episodes involving physical assault over a year; and the aggressive act was not premeditated and was not committed in order to achieve some tangible objective. Coccaro and colleagues (2004) reported 28 (11.07%) of 253 subjects met inclusion criteria for lifetime IED by either *DSM-IV* or research criteria. Of the individuals meeting criteria for IED, 81.3% reported significant psychosocial impairment, and 50% reported personal distress associated with their aggressive behavior. In addition, impairment was reported in association with aggression-related problems in relationships in 62.5% of subjects or psychosocial impairment with the law (50%). Despite the external consequences of their aggressive acts, individuals with IED perceive a limited capacity to resist the urge to engage in the behavior, as they tend to experience a tension build-up and are compelled to follow through with the aggressive act.

A. IED and SUD

Lifetime prevalence studies (McElroy, Soutullo, Beckman, Taylor, and Keck, 1998) of individuals with comorbid IED and SUD include 44% for alcohol, 33% for drug, and 48% for any substance use disorder. Subjects with IED have reported alcohol worsening or reducing symptoms, and cannabis use reducing symptoms (McElroy et al., 1998). Approximately 57% of a community sample of aggressive drivers (N = 30) reported current or past alcohol abuse or dependence (Galovski, Blanchard, and Veaszey, 2002). Of the individuals that were diagnosed with IED (N = 10), 70% reported current or past SUD. Individuals with IED are also likely to have comorbid

personality disorders, including borderline personality disorder and antisocial personality disorder, which place individuals at a greater risk for SUD (Galovski et al., 2002). We will integrate discussion of SUD in the following section of the mediating factors contributing to IED.

B. Tension Reduction

As we have discussed with other impulse control problems, individuals can experience an urge to engage in the behavior, and it may be rewarding as in the case of PG, or develop out of habit, as seen with TTM. Most individuals with IED, however, do not appear to obtain similar benefits (e.g., reward) from acting on their impulsive tendencies, but rather, experience tension reduction alone (McElroy et al., 1998). McElroy and colleagues (1998) reported that all subjects (N = 27) with IED in a case series experienced an irresistible impulse to be aggressive prior to the aggressive acts. For subjects in this small case study, aggressive impulses were associated with a build-up in tension (88%), the aggressive acts were associated with a relief of tension (75%), and in a smaller percentage of cases (48%), individuals experienced pleasure following the aggressive acts. Impulsiveness and aggression, which are prominent symptoms among individuals with IED, are also strong predictors of craving substances in SUD (Zilberman, Taveres, and el-Guebaly, 2003). In addition, individuals with SUD reporting high levels of aggression have been found to endorse more situations that trigger use of substances (McCormick and Smith, 1995). Individuals with IED misinterpret benign environmental cues to be aggressive and are more likely to act on the aggressive impulse when disinhibited following substance use. Alcohol expectancies, or what benefit an individual expects from using drugs or alcohol, are strongly influenced by levels of impulsivity. Alcohol use and impulsivity were studied in a sample of alcohol-dependent subjects with comorbid conduct disorder (Finn, Bobova, Wehner, Fargo, and Rickert, 2005). Finn and colleagues (2005) found that individuals considered high in impulsivity were more likely than nonimpulsive subjects to be accurate in their proximal (immediate) alcohol expectancies (e.g., expect negative outcomes), and despite their accuracy, they continue to engage in abusive substance use. However, negative distal (next day) alcohol expectancies in impulsive subjects could potentially act as a deterrent from successive heavy drinking episodes. This concept will be further discussed in treatment section for IED.

Biological models may offer a greater understanding of changes in tension in IED. Impulsive aggression has been correlated with reduced central serotonin function (Linnoila, Virkkunen, Scheinin, et al. 1983; Virkkunen, Rawlings, Tokola, et al. 1994). Human (Coccaro, Kavoussi, Hauger, Cooper, and Ferris, 1998) and animal (Ferris and Deville, 1994) studies suggest that central vasopressin and aggression are inversely related to serotonin. However, the inverse relationship between serotonin and aggression is not present when, for example, norepinephrine system function is diminished (Wetzler, Kahn, Asnis, et al., 1991). Brain imaging studies (Soloff, Meltzer, Greer, et al., 2000) of impulsive aggression in other populations such as borderline personality disorder have found an association between aggression and decreased serotonin uptake in the medial and orbital regions of the right, prefrontal cortex. Gerald and Higley (2002) proposed that one of the mediating factors that place subjects with low CNS serotonin functioning at an increased risk for alcohol dependence is impulsivity. Serotonin dysfunction, however, is not specific to IED and has been found in many psychiatric disorders (alcohol abuse and dependence) known to have impulse control deficits (Cloninger, 1987), as well as deficits in regulating negative affect.

C. Affective Regulation

Individuals with IED commonly have comorbid *DSM-IV* Axis I and II disorders. In their study, McElroy and colleagues (1998) reported 89% and 93% of IED subjects met criteria for a current and lifetime mood disorder, respectively. Additionally, IED subjects suffer from high rates of lifetime anxiety (48%) and substance use (48–57%) disorders (Coccaro et al., 2004; Galovski et al., 2002). These high rates of co-occurring mood, anxiety, and SUD suggest possible impairment in stress management skills, particularly the ability to tolerate negative emotion. McElroy and colleagues (1998) found that IED subjects commonly reported affective or manic-like symptoms, such as irritability and rage during their aggressive acts, and a rapid onset of depressed mood and fatigue after the acts. Subjects were excluded from this study if their aggressive acts were better accounted for by bipolar disorder, alcohol abuse, or antisocial personality disorders. The subjects with IED and comorbid bipolar disorder that were retained in the study (52%) reported that the mood and energy changes during aggressive acts were qualitatively similar, but much briefer than those associated with hypomanic or manic episodes (McElroy et al., 1998). Despite the limitations of this study, the findings are consistent with other research (McElroy, Pope, Keck, et al., 1996) reporting subjects with IED commonly having mood-related problems, and that IED is in fact comorbid, rather than secondary to mood, affective, or personality disorders (Coccaro, Posternak, and Zimmerman, In press). Treatment of individuals with IED should therefore consider addressing deficits in affective regulation as well as the maladaptive response to daily stressors.

D. Treatment of IED

IED was defined as a problem related to a lack of premeditation. Treatment efforts may therefore be aimed at increasing the person's ability to consider the consequences of his/her behavior, inhibiting aggressive urges, and developing alternative tension reduction strategies using a combination or individualized psychosocial and pharmacological interventions. A maladaptive coping strategy in which individuals with IED are likely to engage in is alcohol and substance use, which can lead to the development of SUD. As discussed throughout this section, individuals with IED share the common traits of impulsivity and aggression with individuals with SUD. Aggression is also considered a strong predictor for a strong urge to use substances and therefore is an added challenge for individuals with high levels of impulsivity. One strategy suggested by Finn and colleagues (2005) is to address negative alcohol expectancies in individuals high in impulsivity with comorbid SUD. A parallel that may be drawn between the comorbid disorders relates to delaying or inhibiting the impulse to act. Instructing individuals with comorbid IED and SUD to focus on the negative consequences of successive substance use episodes as well as aggressive acts in the context of the triggering situation may act to facilitate inhibition. Unfortunately, there is limited research in the area of psychotherapy specifically developed for individuals with IED, and this may reflect the reluctance of individuals with IED to acknowledge a need for or seek treatment. Coccaro and colleagues (2004) reported that only 2 of 28 subjects diagnosed with IED pursued help for their aggressive behaviors, and only 50% reported distress from their aggressive behaviors. Of those with IED that do seek treatment, the motivation is typically some externally related consequence, such as aggressive driving or other legal problems stemming from impulsive behaviors.

Although there are no published controlled psychological studies for IED, Grodnitsky and Tafrate (2000) found in a nonrandomized pilot study of adult outpatients that participated in imaginal exposure

therapy for anger habituated to anger-provoking scenarios, while Deffenbacher et al. (2002) reported diminished anger for self-identified angry drivers following participation in relaxation training and cognitive therapy. In this study, individuals benefited equally from relaxation only and relaxation plus cognitive therapy. Anger and emotional regulation can also be addressed by dialectical behavior therapy (DBT; Linehan, Tutek, Heard, et al., 1994). Studies have demonstrated improvements in impulsivity and anger in individuals with borderline personality disorder following participation in DBT (Linehan et al., 1994).

Data concerning pharmacological treatment for IED is equally limited. The use of fluoxetine (Coccaro and Kavoussi, 1997) has resulted in diminished impulsive aggression in a double-blind, placebo-controlled study of individuals (N = 40) with personality disorders and current histories of impulsive aggression and irritability, and no current history of major depression, bipolar disorder, or schizophrenia. One randomized, placebo-controlled study of divalproex in impulsivity and aggression included subjects with IED as well as subjects with and without a Cluster B personality disorder (Hollander, Tracy, Swann, et al., 2003; Kavoussi and Coccaro, 1998). In that study divalproex was effective in reducing the aggression of subjects with borderline personality disorder, but no similar benefit was reported for subjects with IED. As noted by McElroy (1999) in an open-label study of patients with IED and comorbid manic or mixed bipolar symptoms, aggressive outbursts have been shown to decrease by greater than 50% using valproate (6 of 8 subjects) and lithium (1 of 2 subjects).

The preliminary treatment findings for individuals with IED require further validation with well-controlled studies to assess the use of various treatment combinations (e.g., pharmacological and psychosocial interventions). Cognitive, relaxation, and exposure therapies have demonstrated some benefit in treating individuals with aggressive behaviors. For example, components of programs such as DBT for individuals with borderline personality disorder may prove efficacious for individuals with IED. Individuals with IED have deficits in the ability to recognize and be mindful of behavioral consequences, necessitating training in self-control in order to inhibit stress responses to benign environmental cues. One additional component of treating anger and the lack of premeditation characteristic of individuals with IED is mindfulness training. A case study (Singh, Wahler, Adkins, and Myers, 2003) of an individual with comorbid psychiatric diagnosis and mental retardation demonstrated self-control over anger using a specific mindfulness strategy. A mindfulness strategy may involve learning a meditation technique that requires an individual to shift awareness and attention from the anger-producing situation to a neutral situation or a neutral part of the body (soles of the feet), as used in Singh and colleagues' pilot investigation. Other areas of research to be explored may include the accuracy of individuals with IED perception of emotion (Silver, Goodman, Knoll, Isakov, and Modai, 2005) as studied in individuals with schizophrenia, and more specifically, misperception of facial expressions (Eastwood and Smilek, 2004). As noted by Eastwood and Smilek, a rapid physiological response to an "unconsciously perceived" facial expression prepares us to react in an adaptive manner to the presence of a threatening individual. However, this adaptive function as mediated by the amygdala (Davidson and Irwin, 1999) may not function as well in individuals with IED in terms of who and what they perceive as threatening.

VI. CONCLUSIONS

In our review of the three categories of impulse control problems, reward, habit, and lack of premeditation, we came to understand the influence and impact of stress or

stressors. Individuals with impulse control problems are understood to have deficits in the ability to regulate emotion, reduce tension, and engage in maladaptive sensation or reward-seeking behaviors. There is a characteristic experience of tension or stress throughout the process of engaging in the various impulsive behaviors, with some experiencing a complete lack of tension, as in TTM. There are many similarities between the impulse control problems and SUD. Individuals with PG problems were found to most closely resemble alcohol and substance dependence, with similar reward pathways and urge intensities, for example. Aggression and impulsivity were found to be mediating factors in IED as well as SUD and personality disorders such as antisocial personality disorder and borderline personality disorder. Individuals found to have higher levels of impulsivity and aggression are considered a higher risk for developing SUD, which is an argument for developing integrated treatment approaches to address inhibitory control for the comorbid problems. A review of the current literature on impulse control problems is limited in the areas of controlled treatment studies, especially with IED. Each of the impulse control problems would appear to benefit from a multimodal treatment approach including psychosocial and pharmacological interventions. CBT programs and specific medications have been utilized with moderate success in treating these problems, having short-term but not long-term benefits with TTM, for example. Future research can evaluate the most effective components of CBT and compare to other treatment approaches such as mindfulness training and self-hypnosis. In dealing with various stressors, individuals with different impulse control problems are not necessarily aware of the problem behavior while they are engaging in it, and the consequences, no matter how severe, don't necessarily deter them from engaging in the behavior. It is important for us to understand the motivations (e.g., reward, sensation seeking, tension release) behind these behaviors and to recognize the strength of the craving or urge (as in PG) to engage in the behavior. Finally, there are numerous negative consequences as a result of engaging in the problem behaviors. Individuals with TTM avoid relationships and certain social activities, individuals with PG can experience severe financial consequences, and individuals with IED can endanger themselves and others by their behavior. Treating these complex impulse control problems requires increasing the individuals' awareness of the problem and positive/negative consequences, diminishing the urge to engage in the behaviors, implementing healthy alternative coping skills (e.g., affective regulation, tension reduction), and providing long-term support and maintenance plans.

REFERENCES

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.) Washington, D.C.: Author.

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

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

Barlow, D. H. (Ed.). (1993). *Clinical handbook of psychological disorders: A step-by-step treatment manual* (2nd ed.). New York: Guilford Press.

Blanchard, E. B., Wulfert, E., Freidenberg, B. M., and Malta, L. S. (2000). Psychophysiological assessment of compulsive gamblers' arousal to gambling cues: A pilot study. *Applied Psychophysiological Biofeedback, 25*, 155–165.

Brown, R. I. (1985). The effectiveness of gamblers anonymous. In W. R. Eadington (Ed.), *The gambling studies: proceedings of the 6th National Conference on Gambling and Risk Taking*. Reno, NV: University of Nevada.

Brown, S. L., Rodda, S., and Phillips, J. G. (2004). Differences between problem and non-problem gamblers in subjective arousal and affective valence amongst electronic gaming machine players. *Addictive Behaviors, 29*, 1863–1867.

Carrion, V. G. (1995). Naltrexone for the treatment of trichotillomania: A case report. Journal of Clinical *Psychopharmacology, 15*(6), 444–446.

Carrol, D., and Huxley, J. A. A. (1994). Cognitive, dispositional, and psychophysiological correlates of dependent slot machine gambling in young people. *Journal of Applied Social Psychology, 24,* 1070–1083.

Castellani, B., and Rugle, L. (1995). A comparison of pathological gamblers to alcoholics and cocaine misusers on impulsivity, sensation seeking, and craving. *International Journal of Addiction, 30,* 275–289.

Casti, J., Toner, B. B., and Yu, B. (2000). Psychosocial issues for women with trichotillomania. *Comprehensive Psychiatry, 41,* 344–351.

Christenson, G. A., Crow, S. J., and MacKenzie, T. B. (1994, May). A placebo controlled double blind study of naltrexone for trichotillomania. Paper presented at the 147th annual meeting of the American Psychiatric Association, Philadelphia, PA.

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

Christenson, G. A., and Manseuto, C. S. (1999). Trichotillomania: Descriptive characteristics and phenomenology. In D. G. Stein, G. A. Christenson, E. Hollander (Eds.), *Trichotillomania* (pp. 1–41). Washington, DC: American Psychiatric Press.

Christenson, G. A., Ristvedt, S. L., MacKenzie, T. B. (1993). Identification of trichotillomania cue profiles. Behavioral Research and Therapy, *31,* 315–320.

Clarke, D. (2004). Impulsiveness, locus of control, motivation, and problem gambling. *Journal of Gambling Studies, 20*(4), 319–345.

Cloninger, C. R. (1987). Neurogenetic adaptive mechanisms in alcoholism. *Science, 236,* 410–416.

Coccaro, E. F., and Danehy, M. (2005). Intermittent explosive disorder. In E. Hollander and D. J. Stein (Eds.), *Clinical manual of impulse-control disorders* (pp. 19–37). Arlington, VA: American Psychiatric Publishing Inc.

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

Coccaro, E. F., Kavoussi, R. J., Hauger, R. L., Cooper T. B., and Ferris, C. F. (1998). Cerebrospinal fluid vasopressin: Correlates with aggression and serotonin function in personality disordered subjects. *Archives of General Psychiatry, 55,* 708–714.

Coccaro, E. F., Posternak, M. A., and Zimmerman, M. (In press). Prevalence and features of intermittent explosive disorder in a clinical setting. *Journal of Clinical Psychiatry.*

Coccaro, E. F., Schmidt, C. A., Samuals, J. F., and 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.

Cunningham-Williams, R. M., Cottler, L. B., Compton, W. M., and Spitznagel, E. L. (1998). Taking chances: Problem gamblers and mental health disorders. Results from the St. Louis Epidemiological Catchment Area study. *American Journal of Public Health, 88,* 1093–1096.

Cunningham-Williams, R. M., Gruzca, R. A., Cottler, L. B., Womack, W. B., Books, S. J., Rrzybeck, T. R., Spitznagel, E. L., and Cloninger, C. R. (2005). Prevalence and predictors of pathological gambling: Results from the St. Louis Personality, Health, and Lifestyle study (SLPHL). *Journal of Psychiatric Research, 39,* 377–390.

Davidson, R. J., and Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in Cognitive Sciences, 3*(1), 11–21.

Deffenbacher, J. L., Filetti, L. B., Lynch, R. S. Lynch, R. S., Dahlen, E. R., and Oetting, E. R. (2002). Cognitive behavioral treatment of high anger drivers. *Behavioral Research and Therapy, 40,* 895–910.

Diefenbach, G. J., Mouton-Odum, S., and Stanley, M. A. (2002). Affective correlates of trichotillomania. *Behavioral Research and Therapy, 40,* 1305–1315.

Diefenbach, G. J., Tolin, D. F., Hannan, S., Crocetto, J., and Worhunsky, P. (2005). Trichotillomania: Impact on psychological functioning and quality of life. *Behaviour Research and Therapy, 43,* 869–884.

Dom, G., Hulstijn, W., and Sabbe, B. (2006). Differences in impulsivity and sensation seeking between early and late-onset alcoholics. *Addictive Behaviors, 31,* 298–308.

Eastwood, J. D., and Smilek, D. (2005). Functional consequences of perceiving facial expressions of emotion without awareness. *Consciousness and Cognition, 14,* 565–584.

Ferris, C. F., and Delville, Y. (1994). Vasopressin and serotonin interactions in the control of agonistic behavior. *Psychoendocrinology, 19,* 593–601.

Finn, P. R., Bobova, B., Wehner, E., Fargo, S., and Rickert, M. E. (2005). Alcohol expectancies, conduct disorder and early-onset alcoholism: Negative alcohol expectancies are associated with less drinking in non-impulsive versus impulsive subjects. *Addiction, 100,* 953–962.

Foa, E. B., and Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin, 99,* 20–35.

Franklin, M. E., Tolin, D. F., and Diefenbach, G. J. (In press). Trichotillomania. In E. Hollander and D. J. Stein (Eds.), *Clinical manual of impulse-control disorders* (pp. 149 173). Arlington, VA: American Psychiatric Publishing Inc.

Friman, P. C., Finney, J. W., and Christopherson, E. R. (1984). Behavioral treatment of trichotillomania: An evaluative review. *Behavior Therapy, 15,* 249–265.

Galovski, T., Blanchard, E. B., and Veazey, C. (2002). Intermittent explosive disorder and other psychiatric comorbidity among court-referred and self-referred aggressive drivers. *Behaviour Research and Therapy, 40,* 641–651.

Gerald, M. S., and Higley, J. D. (2002). Evolutionary underpinnings of excessive alcohol consumption. *Addiction, 97,* 415–425.

Goldstein, L., and Carlton, P. L. (1988). Hemispheric EEG correlates of compulsive behavior: The case of pathological gamblers. *Research in Community Psychology and Psychological Behaviors, 13*, 103–111.

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

Grant, J. E., Kushner, M. G., and Kim, S. W. (2002). Pathological gambling and alcohol use disorder. *Alcohol Research and Health, 26*(2), 143–150.

Grant, J. E., and 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., Hollander, E., Cunningham-Williams, R., Nurminen, T., Smits, G., and Kallio, A. (2006). A multicenter investigation of the opioid antagonist nalmefene in the treatment of pathological gambling. *American Journal of Psychiatry, 163*, 303–312.

Grodnitzky, G. R., and Tafrate, R. C. (2000). Imaginal exposure for anger reduction in adult outpatients: A pilot study. *Journal of Behavioral Therapy and Experimental Psychiatry, 31*, 259–279.

Hanna, G. L. (1997). Trichotillomania and related disorders in children and adolescents. *Child Psychiatry and Human Development, 27*, 255–268.

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

Hodgins, D. C., Currie, S., el-Guebaly, N., and Peden, N. (2004). Brief motivational treatment for problem gambling: A 24-month follow-up. *Psychology of Addictive Behaviors, 18*(3), 293–296.

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

Hollander, E., Pallanti, S., Allen, A., Sood, E., and Rossi, N. B. (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*, 137–145.

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

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

Kim, S. W., Grant, J. E., and Adson, D. E. (2002). Double-blind placebo-controlled study in the treatment of pathological gambling. *Biological Psychiatry, 63*, 501–507.

Kreek, M. J., Nielsen, D. A., Butelman, E. R., and LaForge, K. S. (2005). Genetic influences on impulsivity and vulnerability to drug abuse and addiction. *Nature Neuroscience, 8*(11)1450–1457.

Lejoyeux, M., Feuche, N., Loi, S., Solomon, J., and Ades, J. (1999). Study of impulse control disorders among alcohol dependent patients. *Journal of Clinical Psychiatry, 60(5)*, 302–305.

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

Linden, R. D., Pope, H. G., and Jonas, J. M. (1986). Pathological gambling and major affective disorders: Preliminary findings. *Journal of Clinical Psychiatry, 47*, 201–203.

Linehan, M. M., Tutek, D. A., Heard, H. L., et al. (1994). Interpersonal outcome of cognitive behavioral treatment for chronically suicidal borderline patients. *American Journal of Psychiatry, 151*, 1771–1776.

Linnoila, M., Virkkunen, M., Scheinin, M., et al. (1983). Low cerebral spinal fluid 5-hydroxyindoleacetic acid concentration differentiates impulsive from nonimpulsive violent behavior. *Life Science, 33*, 2609–2614.

Mansueto, C. S., Stemberger, R. M. T., Thomas, A. M., and Golomb, R. G. (1997). Trichotillomania: A comprehensive behavioral model. *Clinical Psychology Review, 17*, 567–577.

Martins, S. S., Taveres, H., de Sliva Lobo, D.S., Galetti, A. M., and Gentil, V. (2004). Pathological gambling, gender, and risk-taking behaviors. *Addictive Behaviors, 29*, 1231–1235.

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

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

McCormick, R. A., Russo, A. M., Ramirez, L. F., et al. (1984). Affective disorders among pathological gamblers seeking treatment. *American Journal of Psychiatry, 141*, 215–218.

McCormick, R. A., and Smith, M. (1995). Aggression and hostility in substance abusers: The relationship to abuse patterns, coping styles, and relapse triggers. *Addictive Behaviors, 20*(5), 555–562.

McElroy, S. L. (1999). Recognition and treatment of DSM-IV intermittent explosive disorder. *Journal of Clinical Psychiatry*, 60(Suppl 15), 12–16.

McElroy, S. L., Pope, H. G., Keck, P. E., et al. (1996). Are impulse control disorders related to bipolar disorder. *Comprehensive Psychiatry, 37*, 229–240.

McElroy, S. L., Soutullo, C. A., Beckman, D. A., Taylor, P., and Keck, P. E. (1998). DSM-IV intermittent explo-

sive disorder: A report of 27 cases. *Journal of Clinical Psychiatry, 59*(4), 203–210.

Moeller, F. G., Barratt, E. S., Dougherty, D. M., Schmitz, J. M., and Swann, A. C. (2001). Psychiatric aspects of impulsivity. *American Journal of Psychiatry, 158,* 1783–1793.

Moody, G. (1990). Quit compulsive gambling. London: Thorsons.

Mouton, S. G., and Stanley, M. A. (1996). Habit reversal training for trichotillomania: A group approach. *Cognitive and Behavioral Practice, 3,* 159–182.

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

Petry, N. M., Stinson, F. S., and Grant, B. F. (2005). Comorbidity of DSM-IV pathological gambling and other psychiatric disorders: Results from the national epidemiological survey on alcohol and related conditions. *Journal of Clinical Psychiatry, 66,* 564–574.

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

Potenza, M. N., Steinberg, M. A., Skudlarski, P., Fulbright, R. K., Lacadie, C. M., Wiber, M. K., Rounsaville, B. J., Gore, J. C., and Wexler, B. E. (2003). Gambling urges in pathological gambling: A functional magnetic resonance imaging study. *Archives of General Psychiatry, 60,* 828–836.

Robiner, W. N., Edwards, P. E., and Chistenson, G. A. (1999). Hypnosis in the treatment of trichotillomania. In D. J. Stein, G. A. Christenson, and E. Hollander (Eds.), *Trichotillomania* (pp. 176–199). Washington, DC: American Psychiatric Association.

Rothbaum, B. O. (1992). The behavioral treatment of trichotillomania. *Behavioral Psychotherapy, 20,* 85–90.

Roy, A., Adinoff, B., Roehrich, L., et al. (1988). Pathological gambling: A psychobiological study. *Archives of General Psychiatry, 45,* 369–373.

Russo, A. M., Taber, J. I., McCormick, R. A., et al. (1984). An outcome study of an inpatient treatment program for pathological gambling. *Hospital and Community Psychiatry, 35,* 823–827.

Schmitt, L. H., Harrison, G. A., and Spargo, R. M. (1998). Variation in epinephrine and cortisol excretion rates associated with behavior in Australian Aboriginal community. *American Journal of Physiological Anthropology, 106,* 249–253.

Sharpe, L., Tarrier, N., Schotte, D., and Spence, S. H. (1995). The role of autonomic arousal in problem gambling. *Addiction, 90,* 1529–1540.

Shinohara, K., Yanagisawa, A., Kagota, Y., et al. (1999). Physiological changes in pachinko players: Beta-endorphin, catecholamines, immune system substances and heart rate. *Journal of Physiological Anthropology and Applied Human Science, 18,* 37–42.

Silver, H., Goodman, C., Knoll, G., Isakov, V., and Modai, I. (2005). Schizophrenia patients with history of severe violence differ from nonviolent schizophrenia patients in perception of emotions but not cognitive function. *Journal of Clinical Psychiatry, 66*(3), 300–308.

Simeon, D., Cohen, L. J., Stein, D. J., Schmeidler, J., Spadaccinni, E., and Hollander, E. (1997). Comorbid self-injurious behaviors in 71 female hair-pullers: A survey study [Brief reports]. *The Journal of Nervous and Mental Disease, 185*(2), 117–119.

Singh, N. N., Wahler, R. G., Adkins, A., and Myers, R. E. (2003). Soles of the feet: A mindfulness-based self-control intervention for aggression by an individual with mild mental retardation and mental illness. *Research in Developmental Disabilities, 24,* 158–169.

Soloff, P. H., Meltzer, C. C., Greer, P. J., et al. (2000). A fenfluramine-activated FDG-PET study of borderline personality disorder. *Biological Psychiatry, 47,* 540–547.

Stanley, M. A., Borden, J. W., Mouton, S. G., and Breckenridge, J. K. (1995). Non-clinical hair-pulling: Affective correlates and comparison with clinical samples. *Behavior Research and Therapy, 33,* 179–186.

Stemberger, R. M. T., Thomas, A. M., Mansueto, C. S., et al. (2000). Personal toll of trichotillomania: Behavioral and interpersonal sequalae. *Journal of Anxiety Disorders, 14,* 97–104.

Stojanov, W., Karayanidis, F., Johnston, P., Bailey, A., Carr, V., and Schall, U. (2003). Disrupted sensory gating in pathological gambling. *Biological Psychiatry, 54,* 474–484.

Symes, B. A., and Nicki, R. M. (1997). A preliminary consideration of cue-exposure, response prevention treatment for pathological gambling behavior: Two case studies. *Journal of Gambling Studies, 13*(2), 145–157.

Taber, J. I., McCormick, R. A., Russo, A. M., et al. (1987). Follow-up of pathological gamblers after treatment. *American Journal of Psychiatry, 144,* 757–761.

Taveres, H., Zilberman, M. L., Beites, F. J., and Gentil, V. (2001). Gender differences in gambling progression. *Journal of Gambling Studies, 17,* 151–160.

Taveres, H., Zilberman, M. L., Hodgins, D. C., and el-Guebaly, N. (2005). Comparison of craving between pathological gamblers and alcoholics. *Alcoholism: Clinical and Experimental Research, 29*(8), 1427–1431.

Townsley-Stemberger, R. M., Mansueto, C. S., and Gardner-Carter, J. (2000). Personal toll of trichotillomania: Behavioral and interpersonal sequelae. *Journal of Anxiety Disorders, 14*(1), 97–104.

van Minnen, A., Hoogduin, K. A. L., Keijsers, G. P. J., Hellebrand, I., and Jan Hendricks, G. (2003). Treatment of trichotillomania with behavioral

therapy or fluoxetine. *Archives of General Psychiatry, 60*, 517–522.

Virkkunen, M., Rawlings, R., Tokola, R., et al. (1994). CSF: Biochemistries, glucose metabolism, and diurnal activity rhythms in alcoholics, violent offenders, fire setters, and healthy volunteers. *Archives of General Psychiatry, 51*, 20–27.

Wetzler, S., Kahn, R. S., Asnis, G. M., et al. (1991). Serotonin receptor sensitivity and aggression. *Psychiatry Research, 37*, 271–279.

Whiteside, S. P., and Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality of Individual Differences, 30*, 669–689.

Zilberman, M. L., Tavares, H., and el-Guebaly, N. (2003). Relationship between craving and personality in treatment-seeking women with substance-related disorders. *BMC Psychiatry, 3*(1).