

IMPULSE-CONTROL DISORDERS NOT ELSEWHERE CLASSIFIED

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Whereas impulse-control disorders (ICDs) were once conceptualized as either addictive or compulsive behaviors, they are now classified within the DSM-IV-TR (American Psychiatric Association 2000) ICD category. These include intermittent explosive disorder (IED; failure to resist aggressive impulses), kleptomania (failure to resist urges to steal items), pyromania (failure to resist urges to set fires), pathological gambling (failure to resist urges to gamble), and trichotillomania (failure to resist urges to pull one's hair) (Table 19–1). However, behaviors characteristic of these disorders may be notable in individuals as symptoms of another mental disorder. If the symptoms progress to such a point that they occur in distinct, frequent episodes and begin to interfere with the person's normal functioning, they may then be classified as a distinct ICD.

There are also a number of other disorders that are not included as a distinct category but are categorized as ICDs not otherwise specified in DSM-IV-TR. These include sexual compulsions (impulsive-compulsive sexual behavior), compulsive shopping (impulsive-compulsive buying disorder), skin picking (impulsive-compulsive psychogenic excoriation), and Internet addiction (impulsive-compulsive computer usage

disorder). One proposal for the research agenda leading up to DSM-V is to include these emerging disorders as new and unique ICDs rather than lumping them together as ICDs not otherwise specified. These disorders are unique in that they share features of both impulsivity and compulsivity and might be labeled as ICDs. Patients afflicted with these disorders engage in the behavior to increase arousal. However, there is a compulsive component in which the patient continues to engage in the behavior to decrease dysphoria. An area of discussion for DSM-V may include whether these disorders should be recognized as distinct ICDs.

In DSM-IV-TR, ICDs are characterized by five stages of symptomatic behavior (Table 19–2). First is the increased sense of tension or arousal, followed by the failure to resist the urge to act. Third, there is a heightened sense of arousal. Once the act has been completed, there is a sense of relief from the urge. Finally, the patient experiences guilt and remorse at having committed the act.

To properly conceptualize ICDs, it is helpful to understand the role of impulsivity within them. Impulsivity is a defining characteristic of many psychiatric illnesses, even those not classified as ICDs, including

TABLE 19-1. DSM-IV-TR impulse-control disorders

Impulse-control disorders not elsewhere classified
Intermittent explosive disorder
Kleptomania
Pyromania
Pathological gambling
Trichotillomania
Impulse-control disorders not otherwise specified
Impulsive-compulsive sexual disorder
Impulsive-compulsive self-injurious disorder
Impulsive-compulsive Internet usage disorder
Impulsive-compulsive buying disorder
Other disorders with impulsivity
Childhood conduct disorders
Binge-eating disorder
Bulimia nervosa
Paraphilias
Exhibitionism
Fetishism
Frotteurism
Pedophilia
Sexual masochism
Sexual sadism
Transvestic fetishism
Voyeurism
Paraphilia not otherwise specified
Bipolar disorder
Attention-deficit/hyperactivity disorder
Substance use disorders
Cluster B personality disorders
Neurological disorder with disinhibition

Source. American Psychiatric Association 2000.

Cluster B personality disorders such as borderline personality disorder (BPD) and antisocial personality disorder, neurological disorders characterized by disinhibited behavior, attention-deficit/hyperactivity disorder (ADHD), substance and alcohol abuse, conduct

TABLE 19-2. Core features of impulse-control disorders

Essential features	Failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person or to others
Before the act	The individual feels an increasing sense of tension or arousal
At the time of committing the act	The individual experiences pleasure, gratification, or relief
After the act	The individual experiences a sense of relief from the urge The individual may or may not feel regret, self-reproach, or guilt

Source. American Psychiatric Association 2000.

disorder, binge eating, bulimia, and paraphilias. It is important for clinicians to recognize that individuals who are prone to impulsivity and ICDs are often afflicted with a cluster of related conditions including sexual compulsions, substance use disorders, and post-traumatic stress disorder and to screen for comorbid conditions, such as bipolar spectrum disorders and ADHD, that contribute to impulsivity (Figure 19-1).

Impulsivity research has been conducted both in disorders characterized by impulsivity, such as BPD, antisocial personality disorder, and conduct disorder, and in traditional ICDs, such as IED. As such, the basic tenets of impulsivity can be applied both to the ICDs and to other related psychiatric conditions.

Impulsivity—the failure to resist an impulse, drive, or temptation that is potentially harmful to oneself or others—is both a common clinical problem and a core feature of human behavior. An impulse is rash and lacks deliberation. It may be sudden and ephemeral, or a steady rise in tension may reach a climax in an explosive expression of the impulse, which may result in careless actions without regard for self or others. Impulsivity is evidenced behaviorally as carelessness; an underestimated sense of harm; extraversion; impatience, including the inability to delay gratification; and a tendency toward risk taking, pleasure, and sensation seeking (Hollander 2002). What makes an impulse pathological is an inability to resist it and its expression. The nature of impulsivity as a core symptom domain within the ICDs allows it to be distinguished as either a symptom or a distinct disorder, much in the same way as anxiety or depression.

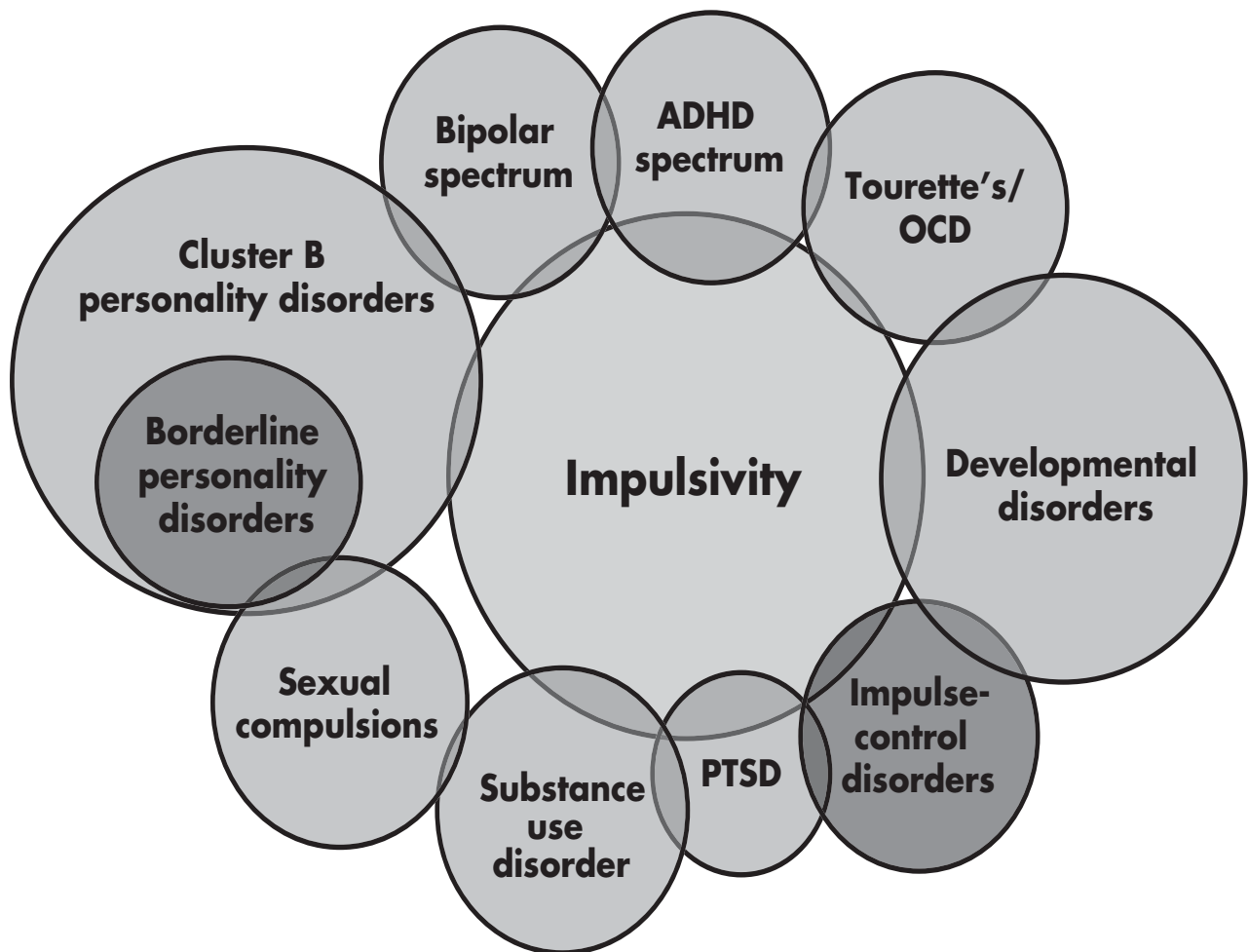


FIGURE 19–1. Impulsive disorder spectrum.

ADHD=attention-deficit/hyperactivity disorder; OCD=obsessive-compulsive disorder; PTSD=posttraumatic stress disorder.

Source. Reprinted from Hollander E, Baker BR, Kahn J, et al.: "Conceptualizing and Assessing," in *Clinical Manual of Impulse-Control Disorders*. Edited by Hollander E, Stein DJ. Washington, DC, American Psychiatric Publishing, 2006, pp. 1–18. Copyright 2006, American Psychiatric Publishing. Used with permission.

Intermittent Explosive Disorder

Definition and Diagnostic Criteria

IED is a DSM diagnosis used to describe people with pathological impulsive aggression. Many clinicians and researchers rarely consider this diagnosis, although impulsive aggressive behavior is relatively common. In community surveys, 12%–25% of men and women in the United States reported engaging in physical fights as adults, a frequent manifestation of impulsive aggression (Robins and Regier 1991). Impulsive aggressive behavior usually is pathological

and causes substantial psychosocial distress or dysfunction (McElroy et al. 1998). Being on the receiving end of impulsive aggressive behavior can lead to similar behavior in a child who grows up in this environment (Huesmann et al. 1984).

Research Criteria for Intermittent Explosive Disorder—Revised

Due to difficulties with the DSM criteria, until recently little research was done using categorical expressions of impulsive aggression. To use an IED diagnosis in research studies, research criteria were created. The Research Criteria for Intermittent Explosive Disorder—Revised (IED-R) described five criteria for IED, em-

phasizing the severity, impulsive nature, frequency, and pathology of the impulsive aggressive behavior. Less severe impulsive aggressive behavior (i.e., verbal aggression or aggression toward property) was included because these forms of aggression had been shown to respond to treatment (Coccaro and Kavoussi 1997). The criteria also specified that *impulsive*, not *premeditated*, aggression would be required for this diagnosis. Prior research had shown psychosocial, biological, and treatment response findings specific to only impulsive and not premeditated aggression. A minimal frequency of aggressive acts was required to increase the reliability of the IED diagnosis and exclude those without severe symptoms. Finally, to distinguish the IED diagnosis as pathological, the criteria required the presence of subjective distress and/or social or occupational dysfunction.

IED-R and DSM-IV Criteria: Defining Integrated Research Criteria for IED

Although DSM-IV (American Psychiatric Association 1994) made some changes to the IED criteria (Table 19–3), it still did not provide criteria useful for research. The “aggressive impulses” of criterion A are not specific in terms of the type or number of acts or the time frame during which the acts must occur. Apparently, no official guidelines for these items had been determined or considered by the DSM-IV subcommittee.

When the subjects from the original IED-R series were reassessed with Research Criteria for IED-R and DSM-IV IED criteria, 69% met both IED-R and DSM-IV IED diagnoses, 20% met criteria for only DSM-IV IED, and 11% met criteria for only IED-R (Coccaro 2003). Because the two criteria sets did not differentiate groups with different aggression and impulsivity levels, and each alone leaves a number of subjects undiagnosed, Integrated Research Criteria for Intermittent Explosive Disorder (IED-IR) were created to allow subjects from any or both of these groups to be identified.

Epidemiology

DSM-IV-TR describes IED as “apparently rare.” However, clinical interview or survey data give a different picture. A number of studies have looked at clinical populations, and one community survey has been done to determine the prevalence of IED. Numbers range between 1.1% and 6.3%. The evaluation of studies is complicated by the variety of defining criteria used, from DSM-III (American Psychiatric Association 1980) to current research criteria and IED-IR. More recently, Zimmerman et al. (1998) used the Structured

TABLE 19–3. DSM-IV-TR diagnostic criteria for intermittent explosive disorder

- 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.
- 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, Alzheimer’s disease).

Clinical Interview for DSM-IV to study current or lifetime IED in 411 outpatient psychiatric subjects. They reported a rate of 3.8% for current IED and 6.2% for lifetime IED using DSM-IV criteria. A recent reanalysis of a much larger sample from the same population revealed similar rates of IED (Coccaro et al. 2005). Further, data from a pilot community sample study revealed a community rate of lifetime IED by DSM-IV-TR criteria at 4% and by IED-IR criteria at 5.1% (Coccaro et al. 2004). Considering the rates found in these more recent studies, IED could be as common as other major psychiatric disorders such as schizophrenia or bipolar illness. Most of the limited published data on gender differences suggest that males outnumber females with IED. However, more recent data suggest that the male-to-female ratio is approximately 1 to 1 (Coccaro et al. 2005).

Comorbidity

Subjects with IED most frequently have other Axis I and II disorders. The most frequent Axis I diagnoses comorbid with IED lifetime include mood, anxiety, substance, eating, and other ICDs ranging in frequency from 7% to 89% (Coccaro et al. 1998a; McElroy et al. 1998). Such Axis I comorbidity rates raise the question of whether IED constitutes a separate disorder. However, recent data finding earlier onset of IED compared with all disorders, except for phobic-type anxiety disorders, suggest that IED is not secondary to these other disorders (Coccaro et al. 2005).

Bipolar Disorder

McElroy et al. (1998) reported that the aggressive episodes observed in their subjects resembled “microdysphoric” manic episodes. Symptoms in common with both manic and IED episodes included irritability (79%–92%), increased energy (83%–96%), racing thoughts (62%–67%), anxiety (21%–42%), and depressed (dysphoric) mood (17%–33%). However, this finding may not be surprising, because 56% of the subjects in question had a comorbid bipolar diagnosis of some type (bipolar I, 33%; bipolar II, 11%; bipolar not otherwise specified or cyclothymia, 11%). The Rhode Island Hospital Study (Coccaro et al. 2005) suggests a much lower rate of comorbid bipolar illness, with a rate of 11% (bipolar I, 5%; bipolar II, 5%; bipolar not otherwise specified, 1%). Regardless, clinicians should fully evaluate for bipolar disorder prior to determining treatment for IED because mood stabilizers, rather than selective serotonin reuptake inhibitors (SSRIs), would be the first-line treatment for IED comorbid with bipolar disorder.

Other Impulse-Control Disorders

McElroy et al. (1998) reported that up to 44% of their IED subjects had another ICD, such as compulsive buying (37%) or kleptomania (19%). However, in the Coccaro et al. (1998a) study, few IED subjects had a comorbid ICD, and in the Rhode Island Hospital Study, only 5% of IED subjects had another ICD (Coccaro et al. 2005).

Borderline and Antisocial Personality Disorders

Coccaro et al. (1998a) reported the rate of BPD and/or antisocial personality disorder in IED subjects to be 38%. However, rates of IED in subjects with BPD have been noted at 78% and in subjects with antisocial personality disorder at 58% (Coccaro et al. 1998a). A review of unpublished data from the author’s (Hollander 2005) research program suggests that these rates are lower among subjects not seeking treatment and are lowest in the community (23% for BPD and/or antisocial personality disorder; see also Coccaro et al. 2004). Regardless, BPD and antisocial personality disorder subjects with a comorbid diagnosis of IED do appear to have higher scores for aggression and lower scores for general psychosocial function than do BPD/antisocial personality disorder subjects without IED (Coccaro et al. 2005).

Pathogenesis

Family and Twin Studies

Clinical observation and family history data suggest that IED is familial. Familial aggregation of temper outbursts and IED has been reported in psychiatric patients with “temper problems” (Mattes and Fink 1987), and McElroy et al. (1998) reported that nearly a third of first-degree relatives of IED probands had IED. A recent blinded, controlled family history study using IED-IR criteria (Coccaro 1999) found a morbid risk of IED of 26% in relatives of IED-IR probands compared with 8% among the relatives of control probands, a significant difference. Although twin studies have confirmed the hypothesis that both impulsivity (Serozynski et al. 1999) and aggression (Coccaro et al. 1997a) are under substantial genetic influence, there are no twin studies of IED itself. Genetic influence for these two traits ranges from 28% to 47%, with non-shared environmental influences making up the lion’s share of the remaining variance.

Molecular Genetic Studies

Studies of particular genes in aggressive populations have used the candidate gene approach. *Candidate genes* are the genes for proteins with a suspected, or proven, biological association to a disorder (e.g., serotonin [5-HT] receptors in aggression). The polymorphism HTR1B/G861C and short tandem repeat locus D6S284 are part of the gene for the 5-HT_{1B} receptor for serotonin. These genetic sites were examined in 350 Finnish sibling pairs and 305 Southwestern American Indian sibling pairs, both with a high rate of alcoholism. The diagnoses of antisocial personality disorder and IED were used to examine the traits of impulsivity and aggression. The rate of IED in relatives of antisocial personality disorder probands was 15%, and the relatives of healthy control subjects had neither IED nor antisocial personality disorder. Lappalainen et al. (1998) were able to discover that the gene predisposing to antisocial personality disorder and alcoholism resides close to the HTR1B version of the coding sequence. They concluded that impulsivity and aggression might be influenced, in part, by 5-HT_{1B} receptors. Other candidate genes include the genes for tryptophan hydroxylase and monoamine oxidase-A. Manuck et al. (1999, 2000) revealed an association of the traits of aggression, impulsivity, and serotonin activity (tested by *d,l-Fen* challenge) with variations in both the tryptophan hydroxylase and monoamine oxidase-A genes in community samples.

Biological Correlates

Serotonin and other centrally acting neurotransmitters are the most studied biological factors in aggression. Measures examining central (as well as peripheral) serotonin function correlate inversely with life history, questionnaire, and laboratory measures of aggression. This relationship has been demonstrated by cerebrospinal fluid 5-hydroxyindoleacetic acid (CSF 5-HIAA; Linnoila et al. 1983; Virkkunen et al. 1994), physiological responses to serotonin agonist probes (Coccaro et al. 1989, 1997b; Dolan et al. 2001; Manuck et al. 1998), and platelet measures of serotonin activity (Birmaher et al. 1990; Coccaro et al. 1996). The type of aggression associated with reduced central serotonin function appears to be *impulsive*, as opposed to *nonimpulsive*, aggression (Linnoila et al. 1983; Virkkunen et al. 1994). These findings suggest that impulsive aggressive behavior can be distinguished biologically from nonimpulsive aggression. Interestingly, the inverse relationship between aggression and serotonin is not observed when catecholamine system function is impaired (Coccaro et al. 1989; Wetzler et al. 1991).

There is also evidence to support the role of other nonserotonergic brain systems and modulators in impulsive aggression. These findings suggest a facilitating role for dopamine (DePue et al. 1994), norepinephrine (Coccaro et al. 1991), vasopressin (Coccaro et al. 1998b), brain-derived neurotrophic factor (Lyons et al. 1991), opiates (Post et al. 1984), and testosterone (Giammanco et al. 2005; Virkkunen et al. 1994) and an inhibitory interaction between neuronal nitric oxide synthase and testosterone in rodents (Kriegsfeld et al. 1997).

Imaging and Brain Localization

Few localization and functional studies have looked at impulsive aggression or IED. Using fluorodeoxyglucose positron emission tomography (PET), Siever et al. (1999) found blunted glucose utilization responses to serotonin stimulation in the orbitofrontal cortex (an area associated with impulsive aggression) of IED subjects with BPD. A similar finding was reported in the anterior cingulate and anteromedial orbital cortex of impulsive aggressive subjects after stimulation with the direct serotonin agonist *m*-chlorophenylpiperazine (New et al. 2002). Using PET with a 5-HT_{1A} antagonist in healthy volunteers, Parsey et al. (2002) found a significant inverse correlation between lifetime aggression and serotonin receptor binding in the dorsal raphe, anterior cingulate cortex, amygdala, medial prefrontal cortex (PFC), and orbital PFC. Using neuropsychological testing in impulsive aggressive subjects, Best et al.'s (2002) data supported a possible dys-

functional frontal circuit. More work is needed to reveal the specific functional brain abnormalities in impulsive aggressive individuals.

Course

Limited research is available concerning the age at onset and natural course of IED. However, according to DSM-IV-TR, the onset appears to be from childhood to the early 20s. The age at onset and course of IED distinguish it as separate from its comorbid diagnoses. The course of IED is variable, with an episodic course in some and a more chronic course in others. A mean age at onset of 16 years and an average duration of about 20 years have been described (McElroy et al. 1998). Preliminary data (Coccaro et al. 2005) confirm these findings and indicate that onset of DSM-IV-TR IED occurs by the end of the first decade in 31%, by the end of the second decade in 44%, by the end of the third decade in 19%, and by the end of the fourth decade in only 6%.

The mode of onset of IED is abrupt and without a prodromal period. Episodes typically last less than 30 minutes and involve physical assault, verbal assault, and/or destruction of property. If provocation is involved, it is usually from a known person and is seemingly minor in nature (McElroy et al. 1998). Many individuals frequently have minor aggressive episodes in the interim between severely aggressive/destructive episodes. Considerable distress and social, financial, occupational, or legal consequences typically result from these episodes.

Treatment

There are few studies in which subjects with IED have been the focus of treatment. There are, however, a number of studies concerning the treatment of impulsive aggression in related subjects (Table 19-4).

Pharmacotherapy

A number of medications have been used to treat impulsive aggression, such as tricyclic antidepressants, benzodiazepines, mood stabilizers, and neuroleptics. Recently, pharmacotherapy studies of aggression have turned to SSRIs and mood stabilizers as first-line treatments. Fluoxetine and other SSRIs have been studied in impulsive aggressive subjects and IED patients. In a treatment trial of subjects meeting IED-IR criteria, impulsive aggressive behavior did respond to fluoxetine (Coccaro and Kavoussi 1997), but non-serotonin-specific antidepressants had little benefit for impulsive aggression and many side effects in treatment studies.

TABLE 19-4. Intermittent explosive disorder: treatment summary

Authors	Treatment	Description
Pharmacotherapy		
Coccaro and Kavoussi 1997	Fluoxetine	Double-blind, placebo-controlled; subjects meeting IED-IR criteria; reduced impulsive aggression
Soloff et al. 1986a	Amitriptyline	Double-blind, placebo-controlled; BPD and SPD inpatients; affective symptoms improved; impulsivity and aggression worsened
Cornelius et al. 1993 Soloff et al. 1993	Phenelzine vs. haloperidol	Double-blind, placebo-controlled; BPD inpatients; phenelzine produced moderate reduction in anger and hostility; only minor benefits in depression and irritability after 16 weeks
Cowdry and Gardner 1988	Alprazolam, tranylcypromine, carbamazepine, trifluoperazine	Double-blind, placebo-controlled, crossover; treatment-resistant BPD outpatients with history of impulsive aggression; improved with tranylcypromine, carbamazepine (decreased behavioral dyscontrol severity and frequency of impulsive aggression episodes; 18% had worsening of mood), and trifluoperazine (improved depression and anxiety objective, not subjective, ratings); increased severity and frequency of episodes of serious dyscontrol with alprazolam
Links et al. 1990	Lithium vs. desipramine	Placebo-controlled; BPD outpatients; objective ratings of anger and suicidality improved with lithium; no improvement in mood
Sheard et al. 1976	Lithium	Double-blind, placebo-controlled; chronically impulsive aggressive prisoners; significant reduction in objective (not subjective) aggressive behavior
Barratt et al. 1997	Phenytoin	Double-blind, placebo-controlled, crossover; impulsive aggressive prisoners; reduced impulsive aggressive acts, but not premeditated aggressive acts
Kavoussi and Coccaro 1998	Divalproex	Open-label trial; variety of personality disorders resistant to SSRIs; decreased irritability and impulsive aggression
Hollander et al. 2003	Divalproex	Double-blind, placebo-controlled; Cluster B personality disorder; decreased impulsive aggression, irritability, and global severity
Soloff et al. 1986b, 1989	Haloperidol vs. amitriptyline	Double-blind, placebo-controlled; BPD inpatients; with haloperidol, decreased symptom severity; depression, hostile depression, anxiety, hostility, impulsivity, schizotypal symptoms, paranoid ideation, psychoticism; improved global functioning

TABLE 19-4. Intermittent explosive disorder: treatment summary (continued)

Authors	Treatment	Description
Montgomery and Montgomery 1982	Flupenthixol	Placebo-controlled; people with history of suicidal and parasuicidal behavior; decreased suicidal and parasuicidal behavior
Zanarini and Frankenburg 2001	Olanzapine	Double-blind, placebo-controlled; BPD patients; improved anger, hostility, impulsivity, and interpersonal relationships, but not depression
Psychotherapy		
Groditzky and Tafrate 2000	Imaginal exposure therapy	Adult outpatients referred for anger management; decreased anger and habituated to anger-provoking scenarios
Deffenbacher et al. 2000, 2002	Relaxation training; relaxation training plus cognitive therapy	Controlled trial; college students with high driving anger; decreased trait anger and driving anger
Linehan et al. 1994	Dialectical behavior therapy vs. treatment as usual	Chronically suicidal BPD patients; improvement in anger, social adjustment, and global functioning

Note. BPD=borderline personality disorder; IED-IR=Integrated Research Criteria for Intermittent Explosive Disorder; SPD=schizotypal personality disorder; SSRI=selective serotonin reuptake inhibitor.

Soloff et al. (1986a) found that affective symptoms improved with amitriptyline in some BPD and schizotypal personality disorder inpatients, but impulsivity and aggression worsened in a set of patients, perhaps due to the noradrenergic effects of tricyclic antidepressants (Links et al. 1990). Thus, clinicians should be cautious when using the new dual-action antidepressants in these patients.

Monoamine oxidase inhibitors such as tranylcypromine and phenelzine have also been studied in impulsively aggressive subjects. In a double-blind study, Soloff et al. (1993) found that compared with placebo and haloperidol, phenelzine produced a moderate reduction in anger and hostility in BPD patients. Yet a 16-week continuation phase revealed that the subjects had experienced only minor benefits in depression and irritability and remained substantially impaired after the treatment phase (Cornelius et al. 1993; Soloff et al. 1993). In a double-blind crossover trial (Cowdry and Gardner 1988), treatment-resistant BPD patients with a history of impulsive aggression showed improvement with tranylcypromine, carbamazepine (decreased severity of behavioral dyscontrol), and trifluoperazine but had an increase in the severity and frequency of the episodes of serious dyscontrol with alprazolam. Benzodiazepine treatment might have released the subjects' control or inhibition of these episodes.

Mood stabilizers have also been used to treat aggression. Links et al. (1990) found objective ratings of anger and suicidality in BPD outpatients improved the most on lithium compared with desipramine and placebo, but subjects and their clinicians did not report any improvement in mood. Sheard et al. (1976) found an improvement using lithium versus placebo in chronically aggressive prisoners. Again, however, only objective findings supported this; no improvement was reported subjectively. Barratt et al. (1997) also reported a reduction in aggression with phenytoin in impulsive aggressive prisoners.

The other mood stabilizers studied for impulsive aggression are carbamazepine and divalproex. In the Cowdry and Gardner (1988) study, carbamazepine lessened episodes of impulsive aggression in BPD subjects, but 18% of subjects had a worsening of mood that improved once carbamazepine was stopped. Kavoussi and Coccaro (1998) and Hollander et al. (2003) reported an anti-aggressive effect of divalproex sodium in IED subjects with a Cluster B personality disorder. Given the relative adverse event profiles for SSRIs versus mood stabilizers, it is likely that clinical treatment of IED patients should start with SSRIs unless the subject is extremely aggressive or has a history of a bipolar disorder, in which case treatment with a mood stabilizer would be more appropriate.

The neuroleptics haloperidol, trifluoperazine, and depot flupenthixol have all been studied in BPD patients. Cowdry and Gardner's (1988) subjects showed significant improvement in depression and anxiety objective ratings with trifluoperazine, but subjective ratings did not support this. Trifluoperazine was seen as less useful than tranylcypromine (a monoamine oxidase inhibitor) and carbamazepine in improving behavior and affect among subjects. Soloff et al. (1986b, 1989) found that BPD inpatients improved on hostility and global function measurements with haloperidol, but considerable depression remained. Montgomery and Montgomery (1982) found that suicidal and parasuicidal behavior, in subjects with a history of such behaviors, decreased in a depot flupenthixol treatment group versus a placebo group. Zanarini and Frankenburg (2001) compared the atypical antipsychotic olanzapine with placebo in outpatients with BPD. The treatment improved anger, hostility, and other symptoms but did not improve depression, and patients remained quite ill.

Psychotherapy

Anger treatment studies focus on treatment of anger as a component of other psychiatric illnesses such as substance abuse, posttraumatic stress disorder, depression, and domestic violence and in forensic and mentally impaired populations. Therapy for anger and aggression focuses on cognitive-behavioral group therapy. In a few rare cases, anger is addressed as the primary or only problem, and a limited number of treatments have been described. Imaginal exposure therapy, used frequently in anxiety disorders, was studied in a noncontrolled pilot study of anger treatment (Grodnitzky and Tafrate 2000). Subjects habituated to anger-provoking scenarios, and the treatment was felt to be useful.

In a controlled trial of college students with high levels of driving anger, Deffenbacher et al. (2000) compared pure relaxation training with relaxation training combined with cognitive therapy and an assessment-only control. Neither treatment condition improved general trait anger, but both treatments improved driving anger. When repeated in a new population of drivers with higher anger levels, both treatments lowered trait anger (Deffenbacher et al. 2002). Because relaxation training with cognitive therapy provided little gain over pure relaxation training, relaxation training in itself may be adequate treatment for driving anger.

Other versions of cognitive-behavioral therapy (CBT), such as dialectical behavior therapy, have been studied in BPD patients. One study showed improvement in anger, social adjustment, and global function-

ing compared with a treatment-as-usual condition (Linehan et al. 1994). Improvement in anger and impulsivity has been shown with dialectical behavior therapy across many disorders. There are no published double-blind, placebo-controlled studies on IED subjects in therapy, but studies of therapy for IED subjects are ongoing.

Kleptomania

Definition and Diagnostic Criteria

Kleptomania was officially designated a psychiatric disorder in 1980 in DSM-III, and in DSM-III-R (American Psychiatric Association 1987) it was grouped under the category "disorders of impulse control not elsewhere classified." Kleptomania is currently classified in DSM-IV-TR as an ICD, but it is still poorly understood and has received very little empirical study. The DSM-IV-TR criteria for kleptomania are listed in Table 19-5.

Criterion A, which focuses on the senselessness of the items stolen, has often been considered the criterion that distinguishes kleptomania patients from ordinary shoplifters (Goldman 1991), but interpretation of this criterion is controversial. The archetype of the middle-aged female kleptomania patient who steals peculiar items may not adequately account for all people with kleptomania (Goldman 1991; McElroy et al. 1991a). Patients with kleptomania may in fact desire the items they steal and be able to use them, but they do not need them. This may be particularly the case with kleptomania patients who hoard items (Goldman 1991), for which multiple versions of the same item are usually not needed, but the item itself may be desired and may be of practical use to the patient.

Patients with kleptomania often report amnesia surrounding the act of shoplifting (Goldman 1991; Grant 2004) and deny feelings of tension or arousal prior to shoplifting and feelings of pleasure or relief after the thefts. They often recall entering and leaving a store but have no memory of events in the store, including the theft (Grant 2004). Other patients, who are not amnesic for the thefts, describe shoplifting as "automatic" or "a habit" and may also deny feelings of tension prior to a theft or pleasure after the act (DSM-IV-TR criterion B or C), although they report an inability to control their shoplifting (criterion A). Some patients report that they felt tension and pleasure when they started stealing, but it became a "habit" over time. Some speculate that patients who are amnesic for shoplifting or who shoplift "out of habit" represent two subtypes of kleptomania.

TABLE 19-5. DSM-IV-TR diagnostic criteria for kleptomania

- 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 relief 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.
- E. The stealing is not better accounted for by conduct disorder, a manic episode, or antisocial personality disorder.

Epidemiology

Although preliminary evidence suggests that the lifetime prevalence of kleptomania may be approximately 0.6% (Goldman 1991), this figure may be an underestimate. The shame and embarrassment associated with stealing prevent most people from voluntarily reporting kleptomania symptoms (Grant and Kim 2002c). No national epidemiological studies of kleptomania have been performed, but studies of kleptomania in various clinical samples suggest a higher prevalence. A recent study in the United States of 204 adult psychiatric inpatients with multiple disorders revealed that kleptomania may in fact be fairly common. The study found that 7.8% ($n=16$) endorsed current symptoms consistent with a diagnosis of kleptomania and 9.3% ($n=19$) had a lifetime diagnosis of kleptomania (Grant et al. 2005). Kleptomania appeared equally common in patients with mood, anxiety, substance use, or psychotic disorders. These findings are further supported by two French studies. One study of 107 inpatients with depression found that 4 (3.7%) had kleptomania (Lejoyeux et al. 2002); in another study of 79 inpatients with alcohol dependence, 3 patients (3.8%) reported symptoms consistent with kleptomania (Lejoyeux et al. 1999). In two studies examining comorbidity in pathological gamblers, rates of comorbid kleptomania were found to range from 2.1% to 5% (Grant and Kim 2003; Specker et al. 1995). A study of bulimia patients found that 24% met DSM-III criteria for kleptomania (Hudson et al. 1983).

The literature clearly suggests that the majority of patients with kleptomania are women (e.g., Grant and Kim 2002b; McElroy et al. 1991b; Presta et al. 2002). One

explanation for this is that kleptomania occurs more frequently in women, but another reason may be that women are more likely to present for psychiatric evaluation. The courts may send male shoplifters to prison while sending female shoplifters for psychiatric evaluation (Goldman 1991). The severity of kleptomania symptoms and the clinical presentation of symptoms do not appear to differ based on gender (Grant and Kim 2002b).

Comorbidity

High rates of other psychiatric disorders have been found in patients with kleptomania and have sparked debate over the proper characterization of this disorder. Rates of lifetime comorbid affective disorders range from 59% (Grant and Kim 2002b) to 100% (McElroy et al. 1991b). The rate of lifetime comorbid bipolar disorder has been reported as ranging from 9% (Grant and Kim 2002b) to 27% (Bayle et al. 2003) to 60% (McElroy et al. 1991b). Studies have also found high lifetime rates of comorbid anxiety disorders (60%–80%; McElroy et al. 1991b, 1992), ICDs (20%–46%; Grant and Kim 2003), substance use disorders (23%–50%; Grant and Kim 2002b; McElroy et al. 1991b), and eating disorders (60%; McElroy et al. 1991b). Personality disorders have been found in 43%–55% of patients with kleptomania, the most common being paranoid personality disorder and histrionic personality disorder (Bayle et al. 2003; Grant 2004).

Pathogenesis

Biological Theories

Serotonin and inhibition. Patients with kleptomania report significant elevations of impulsivity and risk taking compared with control subjects (Bayle et al. 2003; Grant and Kim 2002d), and diminished inhibitory mechanisms may underlie the risk-taking behavior of kleptomania. The most well-studied inhibitory pathways involve serotonin and the PFC (Chambers et al. 2003). Decreased measures of serotonin have long been associated with a variety of adult risk-taking behaviors including alcoholism, fire setting, and pathological gambling (Moreno et al. 1991; Virkkunen et al. 1994). Blunted serotonergic responses in the ventromedial PFC have been seen in people with impulsive aggression (New et al. 2002), and this region has also been implicated in poor decision making (Bechara 2003), as seen in those with kleptomania.

Although there are few biological studies of kleptomania, early evidence may support a theory of serotonergic involvement in the disorder. One study found

a lower number of the platelet serotonin transporter in kleptomania patients versus healthy control subjects (Marazziti et al. 2000). Pharmacological case studies suggest that serotonin reuptake inhibitors such as clomipramine and the SSRIs (Lepkifker et al. 1999; McElroy et al. 1991b) may reduce the impulsive behavior associated with kleptomania.

Dopamine and reward deficiency. Dopaminergic systems influencing rewarding and reinforcing behaviors have also been implicated in ICDs and may play a role in the pathogenesis of kleptomania. One proposed mechanism is “reward deficiency syndrome,” a hypothesized hypodopaminergic state involving multiple genes and environmental stimuli that puts an individual at high risk for multiple addictive impulsive and compulsive behaviors (Blum et al. 2000). Alterations in dopaminergic pathways have been proposed as underlying the seeking of rewards (e.g., shoplifting) that trigger the release of dopamine and produce feelings of pleasure (Blum et al. 2000). Furthermore, dopamine release into the nucleus accumbens has been implicated in the translation of motivated drive into action, serving as a “go” signal (Chambers et al. 2003). Dopamine release into the nucleus accumbens seems maximal when reward probability is most uncertain, suggesting it plays a central role in guiding behavior during risk-taking situations (Fiorillo et al. 2003). The structure and function of dopamine neurons within the nucleus accumbens, in conjunction with glutamatergic afferent and intrinsic γ -aminobutyric acid (GABA)-ergic activities, appear to change in response to experiences that influence the function of the nucleus accumbens. Thus, future behavior may be determined in part by prior rewarding experiences via neuroplastic changes in the nucleus accumbens. This may explain why, over time, many kleptomania patients report shoplifting “out of habit” even without a pronounced urge or craving.

Opioid system, cravings, and pleasure. Preclinical and clinical studies demonstrate that 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 (VTA-NA-OFC) circuit (Hyman 1993; Koob and Bloom 1988), which modulates animal and human motivation (e.g., urges, cravings). Dopamine may play a major role in the regulation of this region (Koob 1992).

Kleptomaniacs report frequent urges to steal that result in theft two times per week, on average (Grant and Kim 2002b). Thus, urges linked to the experienc-

ing of reward and pleasure may represent an important clinical target in treating kleptomania. 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 and Kim 2002b). The μ -opioid system is thought to underlie urge regulation by processing reward, pleasure, and pain at least in part via modulation of dopamine neurons in mesolimbic pathway through GABA interneurons (Potenza and Hollander 2002). Studies of naltrexone, a μ -opioid antagonist, have demonstrated its efficacy in reducing urges in those with kleptomania and other ICDs (Dannon et al. 1999; Grant and Kim 2002c; Kim et al. 2001). Naltrexone may be effective by modulating dopamine function within the VTA-NA-OFC circuit via the antagonism of opioid receptors in the ventral tegmental area (Broekkamp and Phillips 1979).

In summary, repeated kleptomaniac behavior may be a result of an imbalance between a pathologically increased urge and a pathologically decreased inhibition. The repeated shoplifting may therefore be due to increased activity of the mesocorticolimbic dopamine circuitry, indirectly enhanced through the opioid system, and decreased activity in the cortical inhibitor processes, largely influenced via serotonin.

Psychological Theories

Kleptomania may result from an attempt to relieve feelings of depression through stimulation (Goldman 1991; McElroy et al. 1991a). Risk-taking behavior may produce an antidepressant effect for some patients (Fishbain 1987; Goldman 1991). Shoplifting may distract depressed patients from stressors and unpleasant cognitions. Ironically, problems resulting directly from shoplifting (e.g., embarrassment and shame from getting caught) may in turn lead to even more shoplifting as a misguided means of symptom management (Goldman 1991). The self-medication hypothesis of shoplifting is supported by reports from patients with kleptomania of high lifetime rates of depression (45%–100%; Bayle et al. 2003; McElroy et al. 1991b), which usually (60% of cases) precedes the kleptomaniac behavior (McElroy et al. 1991b). Furthermore, several case studies report patients who described shoplifting as relief for their depressed moods (Fishbain 1987) and suggest that kleptomania symptoms improve with antidepressants (Lepkifker et al. 1999; McElroy et al. 1991b).

Behavioral models also provide clues as to the pathogenesis of kleptomania. From an operant viewpoint, the positive reinforcer in kleptomania is the ac-

quisition of items for nothing, and the intermittent reinforcement (e.g., not always being able to shoplift because of store security) of kleptomaniac behavior may therefore be particularly resistant to extinction. Physiological arousal related to shoplifting (Goldman 1991) may be another reinforcer that initiates and perpetuates the behavior. Negative reinforcement (i.e., involving the removal of a punishing stimulus) hypothesizes that shoplifting is performed to experience relief from the aversive arousal of urges. The self-medication theory of kleptomania may represent a negative reinforcement. This could explain why kleptomaniac behavior continues despite the offender being frequently apprehended.

There may also be specific cognitive errors that are directly linked to kleptomaniac behavior: 1) belief that only shoplifting will reduce the urge or the depressive state, 2) selective memory (e.g., remembering the thrill of shoplifting and ignoring the shame and embarrassment from being apprehended), and 3) erroneous self-assessment (e.g., that one deserves to be caught stealing because one is not intrinsically worth anything). A biopsychological perspective will most likely provide the most useful understanding for the treatment and prevention of kleptomania.

Course

Kleptomania may begin in childhood, adolescence, or adulthood and sometimes in late adulthood. However, most patients have an onset of symptoms before age 21 years (i.e., by late adolescence; Goldman 1991; Grant and Kim 2002b; McElroy et al. 1991a, 1991b; Presta et al. 2002). Onset beyond 50 years of age is unusual, and in some of these cases, remote histories of past kleptomania can be elicited (Goldman 1991). Most clinical samples of kleptomania patients report shoplifting for more than 10 years prior to entering treatment (Goldman 1991; Grant and Kim 2002c; McElroy et al. 1991b).

Due to the sparse data on the course of kleptomania and the unavailability of longitudinal studies, the prognosis is not clearly known. However, without treatment the behavior may persist for decades, despite multiple convictions for shoplifting (arrest or imprisonment), with transient periods of remission. Three typical courses have been described: sporadic with brief episodes and long periods of remission; episodic with protracted periods of stealing and periods of remission; and chronic with varying intensity (American Psychiatric Association 2000). Fifteen or 16 years may pass before an individual seeks treatment (Goldman 1991; McElroy et al. 1991a). At peak fre-

quency, McElroy et al. (1991a) found a mean of 27 episodes of theft per month, with one patient reporting as many as 4 thefts per day.

Treatment

Studies of treatment approaches for kleptomania are summarized in Table 19–6.

Pharmacotherapy

No medication is currently approved by the U.S. Food and Drug Administration for treating kleptomania, so it is important to inform patients of “off-label” uses of medications for this disorder and the empirical basis for considering medication treatment.

Only case reports, two small case series, and one open-label study of pharmacotherapy have been conducted for kleptomania. Various medications—tricyclic antidepressants, SSRIs (Lepkifker et al. 1999), mood stabilizers, and opioid antagonists—have been examined for the treatment of kleptomania (Grant and Kim 2002c; McElroy et al. 1989). McElroy et al. (1991b) reported 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 and 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 lithium, fluoxetine plus imipramine (McElroy et al. 1991b), and fluvoxamine plus valproate (Kmetz et al. 1997).

The findings from case reports have not been consistent. Seven cases of fluoxetine, three of imipramine, two of lithium as monotherapy, two of lithium augmentation, four of tranylcypromine, and one of carbamazepine combined with clomipramine all failed to reduce kleptomania symptoms (McElroy et al. 1991b). Some evidence suggests that SSRIs may even induce kleptomania symptoms (Kindler et al. 1997). A case series found that kleptomania symptoms respond to topiramate (Dannon 2003). In another case series, the two subjects treated with naltrexone responded to medication (Dannon et al. 1999).

In the only open-label medication trial for kleptomania, naltrexone (mean effective dosage, 145 mg/day) resulted in a significant decline in the intensity of urges to steal, stealing thoughts, and stealing behavior (Grant and Kim 2002c). A lower dosage, possibly 50 mg/day, may be effective in younger people with kleptomania (Grant and Kim 2002a). Opioid antagonists

TABLE 19-6. Kleptomania: treatment summary

Authors	Treatment	Description
Pharmacotherapy		
Lepkifker et al. 1999	Fluoxetine or paroxetine plus psychotherapy	Kleptomania patients ($n=5$); successfully treated
McElroy et al. 1989	Fluoxetine, trazodone, or tranlycypromine	Kleptomania and bulimia nervosa patients ($n=3$); partial or complete response of both bulimic and kleptomaniac
McElroy et al. 1991b	Fluoxetine, nortriptyline, trazodone, clonazepam, valproate, lithium	Kleptomania patients ($n=20$); 10 of 20 responded to treatment
Chong and Low 1996	Fluvoxamine	Kleptomania patient ($n=1$); successfully treated with fluvoxamine; failed to respond to psychotherapy, behavioral therapy, and pharmacotherapy with clomipramine, imipramine, and lithium
Kraus 1999	Paroxetine	Kleptomania patient ($n=1$); successfully treated
Burstein 1992	Lithium plus fluoxetine	Kleptomania patient ($n=1$); successfully treated
Durst et al. 1997	Fluvoxamine plus buspirone	Kleptomania patient ($n=1$); successfully treated
McElroy et al. 1991b	Fluoxetine plus imipramine Fluoxetine plus lithium	Kleptomania patient ($n=1$); remission with fluoxetine plus imipramine Kleptomania patient ($n=1$); partial response with fluoxetine plus lithium
Kmetz et al. 1997	Fluvoxamine plus valproate	Kleptomania patient ($n=1$); successfully treated
Kindler et al. 1997	Selective serotonin reuptake inhibitors (SSRIs)	Depressed patients ($n=3$); induced kleptomaniac behavior
Dannon 2003	Topiramate (alone or plus SSRIs)	Kleptomania patients ($n=3$); responded well
Dannon et al. 1999	Naltrexone	Kleptomania patients ($n=2$); responded well
Grant and Kim 2002a	Naltrexone	Adolescent with kleptomania ($n=1$); responded to treatment
Grant and Kim 2002c	Naltrexone	Only open-label medication trial for kleptomania ($n=10$); reduced urges to steal, stealing thoughts, and stealing behavior; improved social and occupational functioning
Psychotherapy		
Fishbain 1988 Schwartz 1992	Psychoanalysis	Limited success for kleptomania symptoms, but usually with the addition of medication

TABLE 19–6. Kleptomania: treatment summary (continued)

Authors	Treatment	Description
McElroy et al. 1991b	Insight-oriented psychotherapy	Kleptomania patients ($n=11$); unsuccessful
Gauthier and Pellerin 1982 Glover 1985 Keutzer 1972	Behavioral therapy (i.e., covert sensitization, exposure and response prevention, conditioning)	Successfully treated some cases of kleptomania
McConaghy and Blaszczyński 1988	Imaginal desensitization	Compulsive shoplifting ($n=2$); complete remission
Gudjonsson 1987	Substitution of urges to steal with alternative sources of satisfaction/excitement	Kleptomania patient ($n=1$); successfully treated for 5 months; reported 2 years of symptom remission

such as naltrexone may be effective in reducing both the urges to shoplift and the shoplifting behavior by reducing the “thrill” associated with shoplifting and thus preventing the positive reinforcement of the behavior. Antidepressants, particularly those that influence serotonergic systems (e.g., serotonin reuptake inhibitors), may also be effective in reducing the symptoms of kleptomania by targeting serotonergic systems implicated in impaired impulse regulation. If kleptomania represents both impaired urge regulation and inhibition of behavior, both opioid antagonists and antidepressants may play a pivotal role in controlling this behavior.

Psychotherapy

Many different kinds of psychotherapy have been tried in the treatment of kleptomania. The success of these therapies exists only in case reports, with no published controlled trials of therapy. Psychoanalysis has resulted in some limited success for kleptomania symptoms, but usually with the addition of medication (Fishbain 1988; Schwartz 1992). Insight-oriented psychotherapy, however, has been unsuccessful in treating this disorder in 11 published cases (McElroy et al. 1991b). Behavioral therapies such as covert sensitization, exposure and response prevention, and conditioning have successfully treated some cases of kleptomania (Gauthier and Pellerin 1982; Glover 1985; Keutzer 1972).

Imaginal desensitization uses the idea of imagining the steps of stealing while maintaining a relaxed state. The patient then images the potential scene of stealing but also imagines his or her ability to not steal in that context. Undergoing fourteen 15-minute sessions over 5 days, two patients reported complete remission of symptoms for a 2-year period (McConaghy and Blaszczyński 1988). Learning to substitute alternative sources of satisfaction and excitement when urges

to steal occur was successful in a woman treated weekly for 5 months who later reported 2 years of remitted symptoms (Gudjonsson 1987).

Because few empirical studies are available, research is needed to guide the selection of which psychotherapy to utilize and to investigate the combination of medication and psychotherapy in treating patients with kleptomania.

Pyromania

Definition and Diagnostic Criteria

The essential feature of pyromania is multiple deliberate and purposeful (rather than accidental) fire setting (Table 19–7). The fire-setting behavior is primary, unrelated to another psychiatric state or to ideology, vengeance, or criminality, and does not result from impaired judgment (e.g., in dementia or mental retardation). DSM-IV-TR also excludes “communicative arson,” by which some individuals with mental disorders or personality disorders use fire to communicate a desire or need.

Another important clinical feature of pyromania is the fascination of subjects with fire. People with pyromania like watching fire. They are often recognized as regular “watchers” at fires in their neighborhoods. They may like setting off false fire alarms. Their fascination with fire leads some to seek employment or to volunteer as a firefighter. Patients may be indifferent to the consequences of the fire for life or property or may get satisfaction from the resulting destruction. Their behaviors may lead to property damage, legal consequences, or injury or loss of life to themselves or others.

Recent diagnostic classifications include pyromania among the ICDs. Although the fire setting results

TABLE 19-7. DSM-IV-TR diagnostic criteria for pyromania

- A. Deliberate and purposeful fire setting on more than one occasion.
- B. Tension or affective arousal before the act.
- C. Fascination with, interest in, curiosity about, or attraction to fire and its situational contexts (e.g., paraphernalia, uses, consequences).
- D. Pleasure, gratification, or relief when setting fires, or when witnessing or participating in their aftermath.
- E. The fire setting is not done for monetary gain, as an expression of sociopolitical ideology, to conceal criminal activity, to express anger or vengeance, to improve one's living circumstances, in response to a delusion or hallucination, or as a result of impaired judgment (e.g., in dementia, mental retardation, substance intoxication).
- F. The fire setting is not better accounted for by conduct disorder, a manic episode, or antisocial personality disorder.

from a failure to resist an impulse, there may be important preparation of the fire (Wise and Tierney 1999). The person may leave obvious clues of his or her fire preparation. Pyromania, however, is considered an uncontrolled and most often impulsive behavior.

Epidemiology

Most epidemiological studies have not directly focused on pyromania. These studies include various populations of arsonists or fire setters. Most reveal a preponderance of males with a history of fire fascination (Barker 1994). They also suggest that true pyromania is rare. Fire setting for profit or revenge or secondary to delusions or hallucinations is more frequent than "authentic" ICD. Fire setting is frequent in children and in adolescents. "True" pyromania in childhood rarely appears. Juvenile fire setting is most often associated with conduct disorder, ADHD, or adjustment disorder.

The classic study *Pathological Fire-Setting (Pyromania)* by Lewis and Yarnell (1951) is one of the largest epidemiological studies of this topic and includes approximately 2,000 records from the National Board of Fire Underwriters and cases provided through fire departments, psychiatric clinics and institutions, and police departments near New York City. Thirty-nine per-

cent of the fire setters from the study received the diagnosis of pyromania. Twenty-two percent had borderline to dull normal intelligence, and 13% had between dull and low average intelligence. The authors also described the fire setter as a "pale and yellow, insignificant creature" driven by an irresistible impulse to set fires. The peak incidence of fire setting was between the ages of 16 and 18 years, but this observation has not been confirmed by more recent studies. Pyromania is found in adolescents and is also present at any age. Among females, the diversity of ages is particularly apparent (Barker 1994).

The high prevalence rates of pyromania have not been confirmed by more recent studies. Koson and Dvoskin (1982) found no cases of pyromania in a population of 26 arsonists. Ritchie and Huff (1999) identified only 3 cases of pyromania in 283 cases of arson. According to DSM-IV-TR, pyromania occurs more often in males, especially those with poorer social skills and learning difficulties. This notation confirms the Lewis and Yarnell (1951) data that only 14.8% of those with pyromania are female.

Comorbidity

Pyromania and Depression

Lejoyeux et al. (2002) assessed ICDs, using the Minnesota Impulsive Disorders Interview, in 107 depressed inpatients who met DSM-IV-TR criteria for major depressive episodes (Tables 19-8 and 19-9). Thirty-one depressed patients met criteria for ICDs: 18 had IED, 3 had pathological gambling, 4 had kleptomania, 3 had pyromania, and 3 had trichotillomania. Patients with comorbid ICDs were significantly younger (mean age, 37.7 vs. 42.8 years). Patients with pyromania had a higher number of previous depressions (3.3 vs. 1.3, $P=0.01$). Bipolar disorders were more frequent in the ICD group than in the group without ICDs (19% vs. 1.3%, $P=0.002$). Bulimia (42% vs. 10.5%, $P=0.005$) and compulsive buying (51% vs. 22%, $P=0.006$) were significantly more frequent in the ICD group. The findings of this study may suggest higher prevalence rates of ICDs than are found in a less severely ill population. There were no significant gender differences among patients presenting with ICDs, and in all cases the ICD appeared when patients no longer had mania or hypomania.

Pyromania and Alcohol Dependence

Laubichler et al. (1996) compared the files ($n=103$) of criminal fire setters and subjects with pyromania. Subjects with pyromania were younger (average age, 20 years) than criminal fire setters (average age, 30 years).

TABLE 19–8. Sociological and demographic characteristics of depressed patients without and with impulse-control disorders (ICDs)

	No ICD	Pyromania	Pathological gambling	Intermittent explosive disorder	Kleptomania	Trichotillomania	All ICDs
Patients, <i>n</i> (%)	76 (71.0)	3 (2.8)	3 (2.8)	18 (16.8)	4 (3.7)	3 (2.8)	31 (29.0)
Age, <i>y</i> (mean ± SD)	42.8 (13.9)	49.3 (6.6)	46.6 (13)	35.1 (10) ^a	39.7 (2.5)	30 (5)	37.7 (11)
Gender ratio, men:women	17:59	1:2	2:1	3:15	0:4	0:3	6:25
Married, <i>n</i> (%)	34 (44.7)	3 (100)	3 (100)	9 (50)	3 (75)	3 (100)	21 (67) ^b

^aDifference statistically significant between ICD and non-ICD groups, $t=2.19$, $df=92$, $P=0.03$.

^bDifference statistically significant between ICD and non-ICD groups, $\chi^2=4.66$, $df=1$, $P=0.03$.

Source. Adapted from Lejoyeux et al. 2002.

TABLE 19–9. Clinical characteristics of depressed patients with and without impulse-control disorders (ICDs)

	No ICD	Pyromania	Pathological gambling	Intermittent explosive disorder	Kleptomania	Trichotillomania	All ICDs
Patients, <i>n</i>	76	3	3	18	4	3	31
Previous depressive episodes, <i>n</i> (mean ± SD)	1.3 (1.3)	3.3 (1) ^a	1.3 (1.1)	1.3 (1.5)	5.7 (4) ^b	0	1.9 (2.4)
History of manic episodes (bipolar disorder), <i>n</i> (%)	1 (1.3)	0	0	3 (16)	3 (75)	0	6 (19) ^c
Suicide attempts, <i>n</i> (mean ± SD)	1.1 (2.1)	1 (1)	1 (1.7)	0.5 (0.6)	6 (4.2) ^d	0.6 (0.5)	1.3 (2.3)
Antisocial personality, <i>n</i> (%)	1 (1.3)	0	0	1 (5.5)	0	0	1 (3)
Borderline personality, <i>n</i> (%)	8 (10)	1 (33)	1 (33)	5 (27)	1 (25)	0	8 (26)
Bulimia, <i>n</i> (%)	8 (10.5)	0	1 (33)	5 (27)	4 (100)	3 (100)	13 (42) ^e
Compulsive buying, <i>n</i> (%)	17 (22)	0	3 (100)	9 (50)	1 (25)	3 (100)	16 (51) ^f

Note. Student's *t* test was used.

^aDifference between non-ICD and pyromania groups, $t=2.46$, $df=77$, $P=0.01$.

^bDifference between non-ICD and kleptomania groups, $t=5.33$, $df=78$, $P<0.0001$.

^cDifference between non-ICD and ICD groups, $\chi^2=8.95$, $df=1$, $P=0.002$.

^dDifference between non-ICD and kleptomania groups, $t=4.17$, $df=78$, $P<0.001$.

^eDifference between non-ICD and ICD groups, $\chi^2=11.8$, $df=1$, $P=0.0005$.

^fDifference between non-ICD and ICD groups, $\chi^2=7.5$, $df=1$, $P=0.006$.

Source. Adapted from Lejoyeux et al. 2002.

Seventy of the 103 subjects had consumed alcohol before setting a fire. Fifty-four presented with alcohol dependence. The authors suggested a correlation between the amount of alcohol consumed and the frequency of fire setting. Rasanen et al. (1995) found that young arsonists have frequent alcohol problems: 82% had alcoholism and 82% were intoxicated at the time of committing the crime. The excessive consumption of alcohol had a close connection with the arson committed.

Lejoyeux et al. (1999) searched for ICDs among consecutive admissions for detoxification of alcohol-dependent patients in a French department of psychiatry. They found 30 alcohol-dependent persons presenting with at least one ICD (19 with IED, 7 with pathological gambling, 3 with kleptomania, and 1 case of trichotillomania), but none of the patients presented with two or more ICDs, and no patient presented with pyromania. However, it cannot be concluded from such a limited population that pyromania is not associated with alcohol dependence. Further studies are clearly needed to corroborate or refute this preliminary result.

Fire Setting and Psychiatric Disorders

In most cases, fire-setting behavior is not directly related to pyromania. On the other hand, fire setting in subjects who do not have pyromania appears frequent and often underrecognized. Among psychiatric patients, some research found that 26% of the subjects had a history of fire-setting behavior. Sixteen percent of these patients had actually set fires (Geller and Bertsch 1985). Ritchie and Huff (1999) reviewed mental health records and prison files from 283 arsonists, 90% of whom had a recorded history of mental health problems. Thirty-six percent had schizophrenia or bipolar disorder, and 64% were misusing alcohol or drugs at the time of their fire setting.

Pathogenesis

Biological Markers

Virkkunen et al. (1987, 1994) suggested that pyromania may be associated with reactive hypoglycemia and/or lower concentrations of 3-methoxy-4-hydroxyphenylglycol (MHPG) and CSF 5-HIAA. Their results supported the hypothesis that poor impulse control in criminal offenders is associated with low levels of certain CSF monoamine metabolites and with a hypoglycemic trend.

Impulse fire setters who are violent offenders are often dependent on alcohol and have a father who is also alcohol dependent (Linnoila et al. 1989). Virkkunen et al. (1996) investigated biochemical and fam-

ily variables and predictors of recidivism among forensic psychiatric patients who had set fires. Male alcoholic patients and fire setters ($n=114$) were followed for an average of 4.5 years after their release from prison. Low CSF 5-HIAA and homovanillic acid concentrations were associated with a family history of paternal alcoholism with violence. A low plasma cholesterol concentration was associated with a family history positive for paternal alcoholism without violence. Compared with nonrecidivists, the recidivists who set fires during the follow-up period had low CSF 5-HIAA and MHPG concentrations and early family environments characterized by paternal absence and the presence of brothers at home.

Psychodynamic Models

Psychodynamic models refer to the symbolism of fire, which is complemented by “normal” human interest in fire. Fire interest starts between 2 and 3 years of age and was almost universal in a study of healthy schoolboys at ages 6, 8, and 10 years (Kafry 1980). Among children, the distinction between normal interest in fire and excessive interest leading to pyromania is not always clear. Playing with matches is not a symptom of pyromania. Kolko and Kazdin (1989) showed that “future” pyromaniacs had more curiosity about fire and liked to be in contact with peers or family members involved with fire. According to Geller and Bertsch (1985), children at risk of pyromania were more often involved in fire setting, threatening to set a fire, sounding a false fire alarm, or calling the fire department with a false report of fire than were control subjects. Thus, there may be a continuum between excessive interest in fire and “pure” pyromania.

Since the first description of pyromania by M. Marc, a French psychiatrist, in 1833, the symbolic sexual dimension of pyromania has been noted. Pyromaniacs were later described as fire fetishists. Many pyromaniacs have fire fetishes. A “fire experience” may become a “fire fetish” (McGuire et al. 1965). For example, a fire fantasy, whether imagined or recalled, just before orgasm is conditioned by the positive feedback of orgasm to become more and more exciting.

Lewis and Yarnell (1951) suggested three main groups of fire setters: the accidental, the occasional, and the habitual. Motivations might include sexual gratification derived from converting a sexual impulse into substitutive excitement, or accidental or unintentional, delusional, erotic, revenge, or group effect in children and adolescents. The diverse symbolism of fire is represented in the psychoanalytic interpretations of pyromania.

Females with pyromania frequently have a history of self-harm, sexual abuse, and psychosocial traumas (Noblett and Nelson 2001). The authors suggested that pyromania could be a displacement of aggression, which is observed in people with a history of sexual trauma. Patients may be unable to directly confront people. The channeling of aggression through fire setting may be seen as an attempt to influence their environment and improve their self-esteem where other means have failed. Fire setting may be an attempt at communication by individuals with few social skills (Geller and Bertsch 1985).

The most frequent motives for arson by juveniles (Rasanen et al. 1995) are revenge on parents or other authorities, the search for heroism or excitement, self-destructiveness, the craving for sensation, and an expression of outrage. There is also a lot of self-destructive behavior by juveniles before committing arson: 74% have suicidal thoughts and 44% have tried to commit suicide before committing their crimes.

Course

According to DSM-IV-TR, there are insufficient data to establish a typical age at onset of pyromania and to predict the longitudinal course. In individuals with pyromania, fire-setting incidents are episodic and may wax and wane in frequency. Studies indicate that the recidivism rate for fire setters ranges from 4.5% (Mavromatis and Lion 1977) to 28% (Lewis and Yarnell 1951). Barnett et al. (1997, 1999) compared mentally ill and mentally “healthy” fire setters from trial records

in Germany in a cross-sectional and 10-year follow-up study. Mentally disordered arsonists were more likely than those with no disorder to have a history of arson before their trial. They also were more often convicted of arson again (11% relapse compared with 4%), had fewer registrations of common offenses such as theft as well as traffic violations and alcohol-related offenses, had a higher rate of recurrence, and committed fewer common offenses other than fire setting. Among all arsonists who committed crimes other than arson, those who were found to be partly responsible for their arson committed the highest number of offenses, followed by those who were deemed not responsible for their actions and those who were fully responsible.

Treatment

Treatment for fire setters is problematic because they frequently refuse to take responsibility for their acts, are in denial, have alcoholism, and lack insight (Mavromatis and Lion 1977). Behavioral treatments such as aversive therapy have helped fire setters (Koles and Jenson 1985; McGrath and Marshall 1979). Other methods of treatment rely on positive reinforcement with threats of punishment, stimulus satiation, and operant structured fantasies (Bumpass et al. 1983). Bumpass et al. (1983) treated 29 child fire setters and used a graphing technique that sequentially correlated external stress, behavior, and feelings on graph paper. After treatment (average follow-up, 2.5 years), only 2 of the 29 children continued to set fires. Studies of treatment approaches for pyromania are summarized in Table 19–10.

TABLE 19–10. Pyromania: treatment summary

Authors	Treatment	Description
Psychotherapy		
McGrath and Marshall 1979	Behavioral therapy	Child fire setter ($n=1$); successful
Koles and Jenson 1985	Behavioral therapy	Child fire setter ($n=1$); successful
Bumpass et al. 1983	Technique that sequentially correlates external stress, behavior, and feelings on graph paper to help patients become aware of the cause–effect relationship between feelings and behavior so as to substitute an acceptable behavior	Child fire setters ($n=29$); after treatment (average follow-up, 2.5 years), only 2 of the 29 children continued to set fires
G.A. Franklin et al. 2002	Trauma Burn Outreach Prevention Program (TBOPP), 1-day, interactive program focusing on the medical, financial, legal, and societal impact of fire setting, emphasizing individual accountability and responsibility	132 juveniles (66 arsonists, 66 fire setters) in the TBOPP group; 102 juveniles (33 arsonists and 66 fire setters) in the no-TBOPP group; TBOPP participants had essentially no recidivism compared with the no-TBOPP group

G. A. Franklin et al. (2002) confirmed the positive effect of a prevention program for pyromania. In 1999, they developed the Trauma Burn Outreach Prevention Program. All subjects arrested and convicted after setting a fire received 1 day of information. The program's interactive content focused on the medical, financial, legal, and societal impacts of fire-setting behavior. The rate of recidivism was less than 1% in the group who attended the program compared with 36% in the control group.

Pathological Gambling

Definition and Diagnostic Criteria

Pathological gambling has been considered a distinct diagnostic entity since 1980, when it was first included in DSM-III and similarly in ICD-9-CM (World Health Organization 1978). DSM-IV-TR currently classifies pathological gambling as an ICD not elsewhere classified. The essential feature of pathological gambling is recurrent gambling behavior that is maladaptive (e.g., loss of judgment, excessive gambling) and in which personal, family, or vocational endeavors are disrupted (Table 19–11).

Epidemiology

Prevalence estimates for pathological gambling range from 1% to 3% of the U.S. population (American Psychiatric Association 1994) and show increasing prevalence among females (33% of pathological gambling patients are women; Lesieur 1988) and high school students (1.7%–5.7%; Ladouceur and Mireault 1988; Lesieur and Klein 1987). A U.S. national survey suggested that 68% of the general population participated in some form of gambling and that 0.77% of American adults are considered probable pathological gamblers (Commission on the Review of the National Policy Toward Gambling 1976). Prevalence estimates of probable pathological gambling from state surveys range from 1.2% to 3.4%, with increased rates in states that provide greater opportunity for legal gambling (Commission on the Review of the National Policy Toward Gambling 1976; Volberg 1990; Volberg and Steadman 1988, 1989).

A meta-analysis of 120 published studies indicated that the lifetime prevalence of serious gambling (meeting DSM criteria for pathological gambling) among adults is 1.6% (Shaffer et al. 1999). Among those younger than 18 years, the prevalence is 3.9%, with past-year rates for adults and adolescents being 1.1%

TABLE 19–11. DSM-IV-TR diagnostic criteria for pathological gambling

- A. Persistent and recurrent maladaptive gambling behavior as indicated by five (or more) of the following:
- (1) is preoccupied with gambling (e.g., preoccupied with reliving past gambling experiences, handicapping or planning the next venture, or thinking of ways to get money with which to gamble)
 - (2) needs to gamble with increasing amounts of money in order to achieve the desired excitement
 - (3) has repeated unsuccessful efforts to control, cut back, or stop gambling
 - (4) is restless or irritable when attempting to cut down or stop gambling
 - (5) gambles as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)
 - (6) after losing money gambling, often returns another day to get even (“chasing” one’s losses)
 - (7) lies to family members, therapist, or others to conceal the extent of involvement with gambling
 - (8) has committed illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling
 - (9) has jeopardized or lost a significant relationship, job, or educational or career opportunity because of gambling
 - (10) relies on others to provide money to relieve a desperate financial situation caused by gambling
- B. The gambling behavior is not better accounted for by a manic episode.

and 5.8%, respectively (Shaffer and Hall 1996).

Prevalence estimates of pathological gambling in the general population differ from estimates in a treatment-seeking population. In a New York State epidemiological survey, relative to gamblers identified in treatment programs, there were higher rates of pathological or probable pathological gamblers who were female (36% vs. 7%, respectively), younger (less than 30 years; 38% vs. 18%, respectively), and nonwhite (43% vs. 9%, respectively) (Volberg and Steadman 1988). Female pathological gamblers clearly represent an understudied and underserved group, because they account for approximately one-third of pathological gamblers (Lesieur 1988). Prevalence estimates of

pathological gambling among high school students range from 1.7% to 3.6% (Ladouceur and Mireault 1988) to 5.7% (Lesieur and Klein 1987).

Comorbidity

The literature to date strongly suggests that three Axis I disorders frequently co-occur with pathological gambling: substance abuse or dependence, affective disorders (i.e., bipolar spectrum disorders), and ADHD (Figure 19–2).

There appears to be a strong relationship between pathological gambling and substance abuse as evidenced by the high rates of comorbid substance abuse and dependency with pathological gambling (Lesieur 1988; Lesieur et al. 1986; Linden et al. 1986; McCormick et al. 1984). Failure to treat comorbid substance use disorders in gamblers may lead to higher relapse rates (Maccallum and Blaszczyński 2002). Pathological gambling is also highly comorbid with affective disorders among inpatient (McCormick et al. 1984) and outpatient (Linden et al. 1986) samples.

Pathological gambling has been associated with ADHD (Carlton and Goldstein 1987). Interestingly, because an association between alcoholism and childhood ADHD has been found (Wood et al. 1983), as well as high co-occurrence between pathological gambling and alcohol abuse (Linden et al. 1986; McCormick et al. 1984), inadequate impulse control may be a key factor that links these three disorders dimensionally (Carlton and Manowitz 1988).

Pathological gambling has been described as being part of the obsessive-compulsive spectrum and sharing features with both obsessive-compulsive disorder (OCD) and the impulsive cluster of obsessive-compulsive spectrum disorders (Bienvenu et al. 2000; Dell’Osso et al. 2005).

Compulsive sexual behavior, compulsive buying disorder, and IED are relatively frequent, as are personality disorders. Murray (1993) found that pathological gamblers fit no particular personality profile, but several investigators have reported abnormal personality traits in pathological gamblers based on dimensional assessments (e.g., Roy et al. 1989). Taber et al. (1987) reported that 20% of 66 pathological gambling inpatients had personality disorders.

Pathogenesis

Neurobiology

There is evidence of serotonergic, noradrenergic, and dopaminergic dysfunction in pathological gambling,

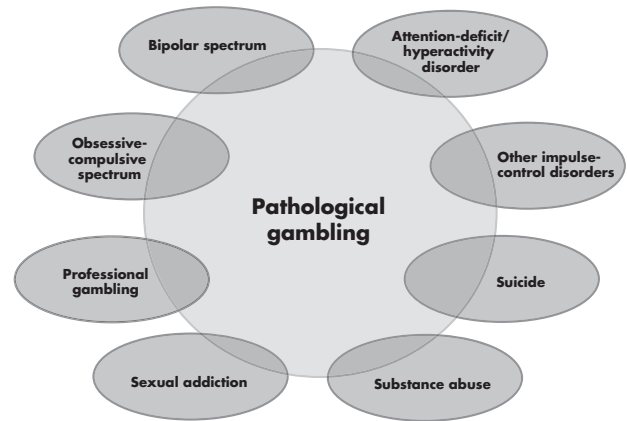


FIGURE 19–2. Pathological gambling: comorbidity and issues in classification.

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and each of these neurotransmitter systems may play a unique role in the mechanisms that underlie the arousal, behavioral initiation, behavioral disinhibition, and reward or reinforcement evident in pathological gambling and other addictive disorders (Table 19–12).

Serotonergic function is linked to behavioral initiation, inhibition, and aggression. Noradrenergic function mediates arousal and detects novel or aversive stimuli. Dopaminergic function is associated with reward and reinforcement mechanisms. Thus, decreased serotonin, increased norepinephrine, and increased dopamine function facilitate addictive or impulsive behavior (Table 19–13).

Evidence of serotonergic dysfunction in pathological gamblers comes from neurobiological studies (Carrasco et al. 1994; DeCaria et al. 1998; Moreno et al. 1991). There is evidence of serotonergic dysfunction in depression (Coccaro et al. 1989), impulsivity (Linnoila et al. 1983), suicidality (Mann et al. 1992), and alcoholism (Tollefson 1991). This is of interest because pathological gambling is strongly associated with depression (Roy et al. 1988a, 1988b), impulsivity (Moreno et al. 1991), suicidality (Ciarrochi and Richardson 1989), and alcohol or drug abuse (Linden et al. 1986; McCormick et al. 1984). Thus, pathological gambling may also be associated with serotonergic dysfunction as it relates to these comorbid features.

In addition to being considered an ICD, pathological gambling has also been linked phenomenologically

TABLE 19-12. Developmental and neurobiological model of pathological gambling

Vulnerable state
Primed genetically/neurobiologically
Repeated environmental exposure
Gambling cycle: behavioral mechanisms
Stimulation readiness → norepinephrine
Behavioral initiation → serotonin
Reward/reinforcement → dopamine
Behavioral disinhibition → serotonin

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TABLE 19-13. Evidence of neurobiological dysfunction in pathological gambling

Norepinephrine dysfunction
Increased urinary norepinephrine levels
Increased cerebrospinal fluid MHPG levels
Enhanced growth hormone response to clonidine
Serotonin dysfunction
Low platelet monoamine oxidase activity
Blunted prolactin response to intravenous clomipramine and increased response to <i>m</i> -chlorophenylpiperazine
Response to serotonin reuptake inhibitors
Dopamine dysfunction
Increased prevalence of altered alleles of the genes for dopamine receptors D ₁ , D ₂ , D ₃ , and D ₄

Note. MHPG = methoxyhydroxyphenylglycol.

Source. Reprinted from Pallanti S, Baldini Rossi N, Hollander E: "Pathological Gambling," in *Clinical Manual of Impulse-Control Disorders*. Edited by Hollander E, Stein DJ. Washington, DC, American Psychiatric Publishing, 2006, p. 263. Used with permission.

to OCD and obsessive-compulsive related/spectrum disorders (DeCaria and Hollander 1993; DeCaria et al. 1992; Hollander and Wong 1995a, 1995b), which have also shown evidence for serotonergic dysfunction (Hollander et al. 1992a). Furthermore, patients with OCD, obsessive-compulsive spectrum disorders, and pathological gambling respond well to serotonin re-

uptake blockers such as clomipramine or the SSRIs (Clomipramine Collaborative Study Group 1991; Hollander 1993; Hollander et al. 1992b).

The noradrenergic system also seems to play a role in the pathophysiology of pathological gambling. Pathological gamblers have shown significantly higher CSF MHPG levels, a metabolite of norepinephrine, and greater urinary output of norepinephrine than control subjects (Roy et al. 1988a). Measures of extraversion in pathological gamblers significantly correlate with indices of noradrenergic function (Roy et al. 1989). Furthermore, increased noradrenergic function has been associated with arousal, irritability, and risk-taking behavior (Coccaro et al. 1991), and pathological gambling has been associated with increased arousal and tonic activity of the central noradrenergic system (Brown 1986; Commission on the Review of the National Policy Toward Gambling 1976; Dickerson et al. 1987; Roy et al. 1988a). Noradrenergic mechanisms have also been associated with impulsive and compulsive behavior in other related disorders (Glassman et al. 1993; Hollander et al. 1991).

Genetics

Serotonergic, noradrenergic, and dopaminergic genes have been investigated because of the putative role of these neurotransmitters in pathological gambling, and a number of molecular genetic studies performed to date have reported findings consistent with the involvement of these neurotransmitter systems in pathological gambling (Comings et al. 1996, 1997; Ibanez et al. 2000, 2001; Perez de Castro et al. 1997, 2002). However, some of the studies performed to date have not been adequately controlled for potential differences in racial and ethnic compositions, factors that could account for differences in allelic variant distributions. As such, these studies, although promising, should be regarded as preliminary.

Studies with clinical samples of pathological gamblers suggest an incidence of about 20% of pathological gambling in first-degree relatives (Ibanez et al. 2003; Lesieur 1988), and this led to the consideration of the possible role of a genetic component in the development of pathological gambling. Gambino et al. (1993) found that patients who perceived that their parents had gambling problems were three times more likely to score as probable pathological gamblers on the South Oaks Gambling Screen (Lesieur and Blume 1987). Those who also perceived that their grandparents had gambling problems had a 12-fold increased risk compared with patients who did not perceive gambling problems in their parents and grandparents.

At present, the main source of evidence for the genetic influence in the etiology of pathological gambling derives from a study of 3,359 male twin pairs from the Vietnam Era Twin Registry cohort (Eisen et al. 1998, 2001; Slutske et al. 2000). These data suggest that gambling problems of increasing severity represent a single continuum of vulnerability rather than distinct entities (Eisen et al. 1998, 2001), a genetic susceptibility model in the pathogenesis of pathological gambling (Eisen et al. 1998), and indicate a common genetic vulnerability for pathological gambling and alcohol dependence in men (Slutske et al. 2000). In a smaller twin study, Winters and Rich (1999) found a significant heritability explaining “high action” gambling, like casinos and gambling slot machines, in 92 monozygotic and dizygotic male twin pairs, but no significant differences in heritability were found among males for “low action” games and among 63 female monozygotic and dizygotic twin pairs for either “high action” or “low action” gambling.

Neuropsychology

Clinical comorbidities, along with observations that pathological gambling involves strong motivations to engage in gambling and subjective feelings of reward, withdrawal, and craving for gambling, support the categorization of pathological gambling as “a non-pharmacological addiction” (Blanco et al. 2001; Holden 2001). This view is corroborated by neuroimaging findings that gambling-associated cognitive and motivational events, or responses of pathological gamblers to gambling-related stimuli, are associated with metabolic changes in brain regions implicated in studies of substance use disorders (Breiter et al. 2001; Holden 2001; Potenza et al. 2003). Using fluorodeoxyglucose PET in unmedicated pathological gamblers without comorbid substance use disorders ($n=7$), Hollander et al. (2001) found heightened limbic and sensory activation in a gambling-for-money condition, with increased emotional valence and greater risk and reward, which confirms the salience of monetary reward in the development of pathological gambling.

Data exist to support the notion that individuals with impaired impulse control exhibit abnormalities in risk-benefit decision making in both gambling and nongambling activities and that their cognitive or emotional sense of what distinguishes gambling from other decisions of daily living may be compromised (Bechara 2001; Bechara et al. 2000, 2001; Crean et al. 2000; Petry 2001a, 2001b, 2001c; Petry and Casarella 1999; Potenza 2001; Rogers and Robbins 2001). These deficits could produce an inability to inhibit motivated

drives to gamble, leading to persistent gambling. Myopia for the future and insensitivity to punishment have also been shown in patients with orbitofrontal and ventromedial PFC lesions (Bechara et al. 1994; Berlin et al. 2004), using gambling tasks. Cavedini et al.’s (2002) data suggest a link between pathological gambling and other disorders (i.e., OCD and drug addiction) associated with diminished ability to evaluate future consequences, which may be explained at least in part by an abnormal functioning of the orbitofrontal cortex. Attention problems and impulsivity in pathological gamblers could reflect deficits in executive functioning that are often a consequence of minimal brain damage with orbitofrontal cortex impairment (Berlin et al. 2004; Rugle and Melamed 1993; Specker et al. 1995).

Course

The course of pathological gambling tends to be chronic, but the pattern of gambling may be regular or episodic. Chronicity is usually associated with increases in the frequency of gambling and the amount gambled. Gambling may increase during periods of increased stress. Gambling behavior frequently leads to severe personal, familial, financial, social, and occupational impairment.

Psychiatric disorders such as major depression and alcohol or substance abuse and dependence may develop from or be exacerbated by pathological gambling. There is also a mortality risk associated with pathological gambling. Estimates of suicide attempts in pathological gamblers range from 17% to 24% (Ciarrochi and Richardson 1989; Hollander et al. 2000a). One study found that the suicide rate in cities where gambling is legalized is four times higher than the rate in cities without legal gambling (Phillips et al. 1997). Younger patients are more likely to have suicidal tendencies and major depressive disorders (McCormick et al. 1984). Because most pathological gamblers begin gambling during adolescence (Hollander et al. 2000a), early identification and intervention are critical.

Gender differences have been described in the course of pathological gambling. In males, the disorder usually begins in adolescence (Hollander et al. 2000a) and may remain undiagnosed for years; male pathological gamblers often present with a 20- to 30-year gambling history, with gradual development of dependence. In contrast, onset of pathological gambling in females is more likely to occur later in life. Prior to their seeking treatment, the duration of pathological gambling in women is approximately 3 years. Thus, as a result of the differences in onset and dura-

tion, female pathological gamblers generally have a better prognosis than male pathological gamblers (Rosenthal 1992). Female pathological gamblers also tend to be depressed and may use gambling as an anesthetic, accompanied by excitement, to escape from life's problems (i.e., as in a dissociative state [Jacobs 1988]).

Treatment

There is a relative lack of effective treatments for pathological gambling reported in the literature. The uncontrolled and few controlled treatment studies in the literature, although helpful in providing preliminary direction, are frequently methodologically flawed. Studies of treatment approaches for pathological gambling are summarized in Table 19–14.

TABLE 19–14. Pathological gambling: treatment summary

Authors	Treatment	Description
Pharmacotherapy		
de la Gandara 1999 Hollander et al. 1992b, 1998, 2000a Kim et al. 2002 Zimmerman et al. 2002	Serotonin reuptake inhibitors	Positive results
Pallanti et al. 2002b	Serotonin antagonist (nefazodone)	Prospective 8-week open-label; pathological gambling outpatients ($N=14$); improvements in all gambling outcome measures and depression and anxiety ratings
Haller and Hinterhuber 1994 Hollander et al. 2002 Pallanti et al. 2002a	Mood stabilizers	Positive results
Kim et al. 2001	Opiate antagonist (naltrexone)	1-week single-blind placebo lead-in followed by an 11-week double-blind naltrexone or placebo trial; subjects with pathological gambling disorder ($N=45$); significant improvement on all three gambling symptom measures
Potenza and Chambers 2001	Atypical antipsychotics	Positive results
Psychotherapy		
Petry and Armentano 1999, review	Gamblers Anonymous (GA); cognitive-behavioral therapy	Only 8% of GA attendees achieve a year of abstinence; combining professional therapy and GA participation may improve retention and abstinence; the few studies of cognitive-behavioral treatments are promising
Russo et al. 1984 Taber 1981 Taber et al. 1987	Inpatient programs for pathological gambling with various combinations of individual and group psychotherapy and substance use treatment	Total abstinence was reported by 55%–56% of the patients 1 year later; improved interpersonal relationships, better financial status, decreased depression, and participation in professional aftercare and GA
Dickerson et al. 1990	Self-help manuals	Useful for some
Ladouceur 1990	Cognitive restructuring	Decrease in the frequency of gambling and irrational verbalizations associated with gambling

Pharmacotherapy

Currently there are only a few controlled pharmacological treatment studies of pathological gambling, although this is a recently developing area of research. Pharmacological treatment studies of pathological gambling have demonstrated some promising results with the use of serotonin reuptake inhibitors (de la Gandara 1999; Hollander et al. 1992b, 1998, 2000b; Kim et al. 2002; Zimmerman et al. 2002), serotonin antagonists (Pallanti et al. 2002a), mood stabilizers (Haller and Hinterhuber 1994; Hollander et al. 2002; Pallanti et al. 2002b), opiate antagonists (Kim et al. 2001), and atypical antipsychotics (Potenza and Chambers 2001). However, some studies have not reported significant findings, primarily due to small samples, high placebo response rates, or high rates of discontinuation (Blanco et al. 2002; Grant et al. 2003).

These data suggest the need for conducting well-designed controlled clinical trials of various pharmacological agents in the treatment of pathological gambling, according to different clinical presentations and comorbidities. Treatment should ultimately target all symptom domains within the individual patient that contribute to compulsive gambling, including common comorbid conditions such as bipolar spectrum disorder, ADHD, and substance abuse/dependence disorders.

Psychotherapy

Treatment modalities for pathological gambling are similar to those of other substance abuse disorders and were created based on the addiction model, such as self-help groups, inpatient treatment programs, and rehabilitation programs. Essential features of any therapeutic intervention for pathological gambling include the need to establish both a therapeutic alliance and network, address the underlying pathology, interrupt the behavior and maintain abstinence, problem-solve, and improve quality of life.

The most popular intervention for problem gambling is Gamblers Anonymous (GA), which is similar to Alcoholics Anonymous and Narcotics Anonymous. However, evidence suggests that GA may not be very effective when used without other treatment modalities (Petry and Armentano 1999). Retrospective studies show a dropout rate of up to 70% within the first year (Stewart and Brown 1988), and overall dropout rates range from 75% to 90% (Moody 1990). Only 8% of GA members report total abstinence at 1-year follow-up and 7% at 2-year follow-up (Brown 1985). Although participation in GA's spousal component, Gam-Anon, may be helpful for some family members,

little evidence suggests that it reduces disordered gambling (Petry and Armentano 1999).

Inpatient programs for pathological gambling have included various combinations of individual and group psychotherapy and substance use treatment (Taber 1981), and most strongly encouraged or required attendance at GA meetings. Many patients improved in all programs, and outcome studies have shown 55% of patients reporting abstinence at 1-year follow-up (Russo et al. 1984; Taber et al. 1987). Although methodologically flawed, these reports suggest that professionally delivered multimodal therapy programs, given alone or in combination with GA, may be more effective than GA alone. Self-help manuals may also be useful for some (Dickerson et al. 1990), and studies comparing their effectiveness with professionally delivered CBT are ongoing (Petry and Armentano 1999).

Early reports in the psychoanalytic literature suggest that problem gambling is regressive and representative of various pregenital and genital instincts, unconscious conflicts, or painful affects. Most studies that report good outcomes are based on single-case studies, and some authors believe that purely psychodynamic treatment of pathological gambling is difficult. Rosenthal and Rugle (1994) published a contemporary psychodynamic approach to pathological gambling treatment that integrates traditional psychodynamic psychotherapy with an addiction model.

Behavioral, cognitive, and combined cognitive-behavioral methods have been used in treating pathological gambling. Aversive therapy has been employed to reach the goal of total abstinence of gambling, as have behavior monitoring, contingency management, contingency contracting, covert sensitization, systematic desensitization, imaginal desensitization, in vivo exposure, imaginal relaxation, psychoeducation, cognitive restructuring, problem-solving skills training, social skills training, and relapse prevention. Use of cognitive restructuring facilitates a decrease in the frequency of gambling and irrational verbalizations associated with gambling (Ladouceur 1990).

Trichotillomania

Definition and Diagnostic Criteria

Trichotillomania is a chronic ICD characterized by repetitive pulling out of one's own hair, resulting in noticeable hair loss. The DSM-IV-TR criteria for trichotillomania are listed in Table 19-15.

Criteria B and C are somewhat controversial in

TABLE 19–15. DSM-IV-TR diagnostic criteria for trichotillomania

- A. Recurrent pulling out of one's hair resulting in noticeable hair loss.
- B. An increasing sense of tension immediately before pulling out the hair or when attempting to resist the behavior.
- C. Pleasure, gratification, or relief when pulling out the hair.
- D. The disturbance is not better accounted for by another mental disorder and is not due to a general medical condition (e.g., a dermatological condition).
- E. The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

light of data suggesting that a significant minority of individuals who pull their hair do not report experiencing these feelings (Christenson et al. 1991a; Hanna 1997; King et al. 1995; Schlosser et al. 1994). These findings suggest that the current diagnostic classification of trichotillomania may be overly restrictive, particularly with respect to pediatric samples. As of now, it is unclear whether chronic hair pulling is best conceptualized as a single entity or as a symptom with myriad root causes yet to be identified, with little unifying these subtypes theoretically.

Approximately 75% of adult trichotillomania patients report that most of their hair-pulling behavior takes place “automatically” or outside of awareness, whereas the remaining 25% describe themselves as primarily focused on hair pulling when they pull (Christenson and Mackenzie 1994). However, some patients engage in both types of hair pulling. Compared with unfocused hair pullers, the subset who primarily engage in focused hair pulling are more likely to pull hair from the pubic area and to report shame as a result of their hair pulling (du Toit et al. 2001). Some suggest that trichotillomania patients who engage primarily in focused hair pulling are more similar to patients with OCD and may be more responsive to pharmacological interventions found effective for OCD (Christenson and O’Sullivan 1996; du Toit et al. 2001). The issue of trichotillomania subtyping is one of both considerable importance and ongoing debate, and no formal subtyping system incorporating affective correlates of pulling has been advanced.

The few published data on how trichotillomania presents in children and adolescents suggest similari-

ties to adult hair pulling. As with adults, the scalp is the most common pulling site in children and adolescents, followed by eyelashes and eyebrows (Hanna 1997; Reeve 1999). In one study, almost half of children and adolescents described having a ritual or routine involved in pulling their hair (Hanna 1997). The absence of body hair on younger children precludes pulling from certain sites, but clinical work with adolescents appears consistent with the adult data in that pulling from sites other than the face and scalp is also common. When completed, ongoing research regarding the preferred pulling sites of children and adolescents will add to this knowledge base (M.E. Franklin et al. 2002; Tolin et al. 2002).

Epidemiology

Early clinical studies suggested that trichotillomania was extremely rare; however, survey research with nonclinical samples has indicated that hair pulling is more common than originally suggested. In studies involving college samples, 10%–13% of students reported hair pulling, with the prevalence of clinically significant pulling ranging between 1% and 3.5% (Christenson et al. 1991c; Rothbaum et al. 1993). A large epidemiological study of trichotillomania and skin picking using self-report instruments is under way in a large sample of college freshmen (Hajcak et al. 2006). Epidemiological research on trichotillomania is extremely limited both in terms of the number of studies and methodology. One epidemiological survey of 17-year-old adolescents in Israel suggests a prevalence rate of 1% for current or past hair pulling, with fewer reporting noticeable hair loss or distress from these symptoms (King et al. 1995). There is a need for more epidemiological research on trichotillomania.

Comorbidity

Psychiatric comorbidity is quite common among adults with trichotillomania. Christenson et al. (1991a) found that approximately 82% of an adult sample with trichotillomania met criteria for a past or current comorbid Axis I disorder, the most common being affective, anxiety, and addictive disorders. Of the patients with comorbid disorders, there was a lifetime prevalence rate of 65% for mood disorders, 57% for anxiety disorders, 22% for substance abuse disorders, 20% for eating disorders, and 42% for personality disorders. The most frequently cited comorbid personality disorders are histrionic, borderline, and obsessive-compulsive (Christenson et al. 1992; Schlosser et al. 1994; Swedo and Leonard 1992). In a larger sample of adults

seeking treatment for trichotillomania, Christenson (1995) found comorbidity rates of 57% for major depression, 27% for generalized anxiety disorder, 20% for eating disorders, 19% for alcohol abuse, and 16% for other substance abuse. In a mixed sample of children, adolescents, and adults with trichotillomania, Swedo and Leonard (1992) found comorbidity rates of 39% for unipolar depression, 32% for generalized anxiety disorder, 16% for OCD, and 15% for substance abuse.

Reeve et al. (1992) and King et al. (1995) found that 7 of 10 and 9 of 15 children with trichotillomania had at least one comorbid Axis I disorder, respectively. M. E. Franklin et al. (2002) and Tolin et al. (2002) reported little comorbidity in their pediatric treatment-seeking samples, suggesting that comorbidity may develop secondarily in the wake of trichotillomania. Sampling issues most likely underlie these observed differences. Nevertheless, if it is indeed the case that children and adolescents with trichotillomania are less comorbid than adults with trichotillomania, successful early intervention in children and adolescents with trichotillomania may help reduce the rates and severity of later adult psychiatric comorbidity and functional impairment (Keuthen et al. 2002). More longitudinal and psychopathology research is needed.

A key debate in the field is whether trichotillomania should be conceptualized as an ICD or a variant of OCD. In support of the classification as an obsessive-compulsive spectrum disorder is the apparent similarity between compulsions and the repetitive and perceived uncontrollable nature of hair pulling and accompanying anxiety relief (Swedo 1993; Swedo and Leonard 1992), the possible selective responsiveness of trichotillomania to serotonin reuptake inhibitors, and the elevated rates of OCD in patients with trichotillomania (Christenson et al. 1991a). Others argue that trichotillomania and OCD are separate diagnoses because trichotillomania is not characterized by persistent intrusive thoughts regarding hair pulling, hair pulling often occurs outside awareness, and the repetitive behavior in trichotillomania is generally limited to hair pulling whereas compulsions in OCD often consist of a variety of anxiety-relieving behaviors. Those with OCD also describe their compulsions as unpleasant but necessary to reduce negative affect (i.e., maintained by negative reinforcement), whereas most subjects with trichotillomania describe hair pulling as pleasurable or satisfying (i.e., maintained by positive reinforcement). Furthermore, OCD patients' age at onset is generally later (Himle et al. 1995; Swedo 1993; Tukul et al. 2001), they report higher levels of overall anxiety (Himle et al. 1995; Tukul et al. 2001),

and they have a more restricted range of affective states than trichotillomania patients (Stanley and Cohen 1999). The proposed difference between OCD and trichotillomania has led to the use of disparate CBT strategies for each.

Many authors (e.g., Christenson and Mansueto 1999) have noted similarities among skin picking and severe nail biting as well as common co-occurrence. If the skin picking and nail biting appear to be largely negatively reinforcing—reducing anxiety associated with specific obsessional thoughts and/or reducing the likelihood of feared outcomes—they may be better conceptualized as OCD behaviors. Clinical experience suggests these conditions are much more likely to formally resemble trichotillomania. More research is needed to determine whether they are all one entity or distinct conditions.

Pathogenesis

Figure 19–3 shows a schematic diagram of a preliminary biopsychosocial model of trichotillomania. This model is preliminary, because the available experimental and descriptive psychopathology research in trichotillomania is sparse. This model is heuristic rather than explanatory, but it is hoped it will stimulate new studies on the mechanisms of trichotillomania and be modified as new data become available.

Biological Vulnerability

Biological vulnerabilities likely increase the probability that a person will develop trichotillomania. Familial research suggests that trichotillomania may be associated with increased rates of OCD or other excessive habits among first-degree relatives (Bienvenu et al. 2000; King et al. 1995). This is consistent with the notion of a genetic basis for a spectrum of excessive grooming behaviors that include trichotillomania, but environmental factors such as social learning cannot be ruled out.

Neuroimaging has demonstrated hyperactivity in the left cerebellum and right superior parietal lobe (Swedo et al. 1991) and possible structural abnormalities in the left putamen (O'Sullivan et al. 1997), left inferior frontal gyrus, and right cuneal cortex (Grachev 1997). Trichotillomania patients have also shown errors in spatial processing (Rettew et al. 1991), divided attention (Stanley et al. 1997), nonverbal memory, and executive functioning (Keuthen et al. 1996), although in the latter study, Bonferroni correction for multiple comparisons would have made these differences nonsignificant. Studies like these do not necessarily imply that preexisting brain abnormalities cause the symp-

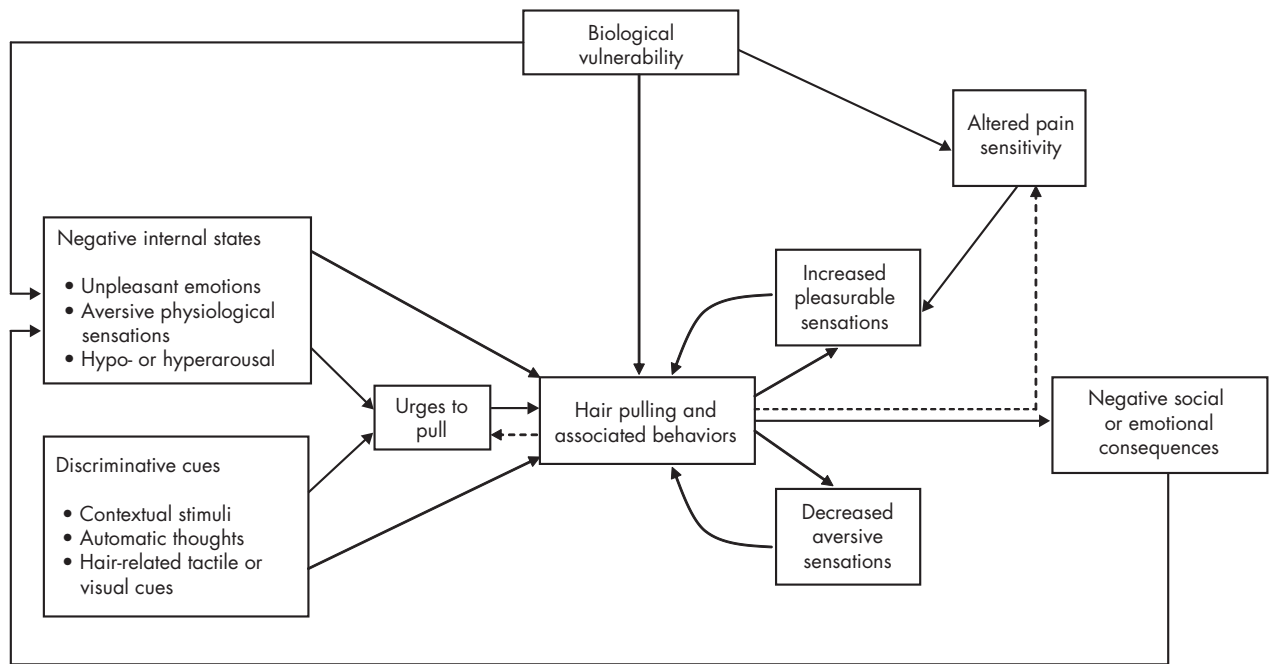


FIGURE 19–3. Schematic diagram of a preliminary biopsychosocial model of trichotillomania.

Source. Reprinted from Franklin ME, Tolin DF, Diefenbach GJ: Trichotillomania, in *Clinical Manual of Impulse-Control Disorders*. Edited by Hollander E, Stein DJ. Washington, DC, American Psychiatric Publishing, 2006, pp. 149–173. Copyright 2006, American Psychiatric Publishing. Used with permission.

toms of trichotillomania; it may be that chronic trichotillomania or its associated features lead to changes in brain structure or function or that both trichotillomania and the brain abnormalities are caused by a third, unknown, variable.

Altered Pain Sensitivity

Individuals with trichotillomania often report that hair pulling is not painful (Christenson et al. 1991c) or in many cases that it feels good or pleasurable (Stanley et al. 1992). To date, no studies have compared hair-pulling sensations between those with trichotillomania and those without, but it is suspected that those without trichotillomania generally do not derive pleasure from pulling; rather, they are likely to describe it as painful. Thus, alterations in pain sensitivity may influence the reinforcing quality of pulling behavior. One possible mechanism for such alterations is upregulation of the endogenous opioid system; this model has not been supported by challenge tasks (Frecska and Arato 2002), although some evidence suggests that pulling may decrease with administration of opiate receptor antagonists (Carrion 1995; Christenson et al. 1994a). Trichotillomania patients do not appear to show reduced pain in nonpulling areas such as the fin-

gertips (Christenson et al. 1994b), so pain sensations are not globally altered in trichotillomania but rather are diminished only at the sites of pulling. This may result from habituation of the pain response caused by repeated pulling over time, although pain absence has been noted even in young children with short pulling histories (Chang et al. 1991). To date, no studies of pain tolerance at the preferred pulling site have been conducted. For those patients who do experience pulling-related pain, the pain itself may be reinforcing because it distracts the individual from negative emotional or physiological states (Christenson and Mansueto 1999).

Hair-Pulling Cues

The behavioral model of hair pulling suggests that pulling begins as a normal response to stress but eventually becomes associated with a variety of internal and external cues through conditioning (Mansueto et al. 1997). Christenson et al. (1993) identified two facets of hair-pulling cues: negative affect (hyperarousal) and sedentary/contemplative cues (hypoarousal, e.g., boredom, fatigue, sedentary activities like reading, television). Physical sensations such as skin sensitivity, itching, irritation, pressure, and burning sensations preceding pulling episodes have also been identified

as arousal cues for hair pulling (Mansueto 1990).

Cognitions may serve as cues and consequences to the behavioral sequence. Negative cognitions about the pulling habit itself, such as fear of negative evaluation or worry that the urges to pull will never go away or will get stronger until they pull, resulting in negative emotion, may also increase urges to pull. Belief in the positive effects of hair pulling (e.g., “Hair pulling will make me feel better”) or pulling-facilitative thoughts (e.g., “I’ll just pull one”) may also cue pulling episodes (Gluhoski 1995).

Common contextual cues linked with pulling or pulling-related feelings by conditioning include visual signs that hair is misshapen or unattractive, tactile sensations like feeling a coarse hair, places or activities where pulling has previously occurred, being alone, or the presence of pulling implements like tweezers.

Reinforcement

Hair pulling is often preceded by negative internal states such as unpleasant emotions, aversive physiological sensations, or dysregulated arousal. Hair pulling appears to result in a decrease of these states. Over time, hair-pulling urges that are reinforced by pulling lead to stronger urges to pull, which perpetuates the behavioral cycle. Trichotillomania patients report retrospectively that pulling leads to reduced feelings of tension, boredom, and anxiety, and nonclinical hair pullers also report reductions in sadness and anger (Stanley et al. 1995). In these cases, hair pulling is negatively reinforced and is thus somewhat similar to the compulsive behaviors seen in OCD (Tolin and Foa 2001). However, to the extent that hair pulling evokes pleasurable sensations (Stanley et al. 1992), the habit may also be strengthened via *positive* reinforcement (Mansueto et al. 1997). Pleasure may be obtained not only through pulling but also through associated behaviors like playing with or inspecting the hair, oral stimulation, or trichophagia (Christenson and Mansueto 1999). Thus, pulling may be maintained by either negative or positive reinforcement. Some trichotillomania patients may experience one or the other form of reinforcement, or different kinds of reinforcement may be active for the same person at different times. Careful attention to hair-pulling contingencies is important in planning therapeutic interventions for trichotillomania.

Course

Age of onset usually ranges from early childhood to young adulthood. Initial onset after young adulthood

is uncommon, but there have been reports of onset as early as 14 months and as late as 61 years. Peak age of onset in children is at about age 5–8 years, whereas for patients who present to clinicians in adulthood, the mean age of onset is about 13 years (Rothbaum et al. 1993; Swedo et al. 1989).

Transient periods of hair pulling in early childhood may be considered benign and usually have a self-limited course, with most cases remitting spontaneously by the teenage years. Circumscribed periods of hair pulling (weeks to months) followed by complete remission are common among children. This may be because it usually represents a “habit” without the presence of an obvious precipitant or a transient behavior in response to a psychosocial stressor.

Trichotillomania in adolescents and adults typically follows a chronic course, involves multiple hair sites, and is associated with high rates of psychiatric comorbidity (Christenson et al. 1991a). The chronic course may take one of two patterns: in one, the frequency and severity of hair pulling wax and wane over months, without any true remissions; in the other, episodes are characterized by frequent hair pulling separated by long periods of remission (Moore and Jefferson 2004). Some have continuous symptoms for decades. For others, the disorder may come and go for weeks, months, or years. Sites of hair pulling may vary over time. Progression of the condition seems to be unpredictable.

Treatment

The treatment literature for trichotillomania is generally made up of case studies, with progressively more controlled investigation in recent years. In general, knowledge about trichotillomania treatments is limited by small sample sizes, lack of specificity regarding sample characteristics, nonrandom assignment to treatment, dearth of long-term follow-up data, exclusive reliance on patient self-report measures, and lack of information regarding rates of treatment refusal and dropout. Studies of treatment approaches for trichotillomania are summarized in Table 19–16.

Pharmacotherapy

Of the six randomized controlled trials evaluating the efficacy of pharmacotherapy conducted to date, five involved serotonin reuptake inhibitors. This may reflect the previously prevailing view that trichotillomania is a variant of OCD and thus ought to be responsive to the same pharmacological agents proven successful in OCD. In sum, results from these con-

TABLE 19-16. Trichotillomania: treatment summary

Authors	Treatment	Description
Pharmacotherapy		
Swedo et al. 1989, 1993	Clomipramine vs. desipramine	10-week double-blind crossover; women with severe trichotillomania ($n=13$); greater improvement and reduced severity of symptoms with clomipramine vs. desipramine; patients reported that compulsion decreased in intensity, and they were more able to resist the urge to pull out their hair with clomipramine; 40% (moderate) reduction in severity of symptoms at a mean of 4.3 years of follow-up
Christenson et al. 1991c	Fluoxetine	18-week placebo-controlled, double-blind crossover; adult chronic hair pullers ($n=15$ completers); short-term efficacy of fluoxetine in the treatment of trichotillomania was not demonstrated
Streichenwein and Thornby 1995	Fluoxetine	Long-term (31-week) double-blind, placebo-controlled crossover; adult chronic hair pullers ($n=16$ completers); efficacy of fluoxetine in the treatment of trichotillomania was not demonstrated
Ninan et al. 2000	CBT vs. clomipramine	Placebo-controlled, randomized, parallel treatment; patients with trichotillomania ($n=16$ completers); CBT dramatically reduced symptoms and was significantly more effective than clomipramine or placebo; clomipramine reduced symptoms more than placebo, but the difference was not significant
van Minnen et al. 2003	Behavior therapy vs. fluoxetine vs. wait list	12-week randomized, wait list-controlled; patients with trichotillomania ($n=40$ completers); behavioral therapy was highly effective for reducing symptoms of trichotillomania in the short term, while fluoxetine was not
Epperson et al. 1999	SSRIs plus risperidone	Open-label; 3 patients with serotonin reuptake inhibitor-refractory trichotillomania; all 3 patients had a robust decrease in hair pulling measured by clinician-rated instruments
Stein and Hollander 1992	SSRIs plus pimozide	Open-label; improvement in hair pulling in 6 of 7 trichotillomania patients; response was sustained in those able to tolerate their medication
Stewart and Nejtcek 2003	Olanzapine	3-month open-label; patients with trichotillomania ($n=16$ completers at 1 week); decreased hair pulling and anxiety, with global improvement

TABLE 19-16. Trichotillomania: treatment summary (continued)

Authors	Treatment	Description
Christenson et al. 1994b	Naltrexone	Placebo-controlled, double-blind; reducing trichotillomania symptoms in patients with trichotillomania
Pollard et al. 1991	Clomipramine	Open-label; 3 of 4 patients with trichotillomania relapsed completely at 3-month follow-up, although still taking previously effective levels of the drug; remaining patient relapsed for 2 weeks but regained initial treatment benefits
Iancu et al. 1996	Serotonergic drugs	Open-label; 12 patients with trichotillomania; treatment response not maintained at follow-up
Psychotherapy		
Ninan et al. 2000 van Minnen et al. 2003	CBT	See above
Azrin et al. 1980	Habit reversal	Habit reversal was more effective than negative practice in treating trichotillomania
Lerner et al. 1998 Keuthen et al. 2001 Mouton and Stanley 1996	CBT	Significant relapse following CBT

Note. CBT=cognitive-behavioral therapy; SSRI=selective serotonin reuptake inhibitor.

trolled studies of serotonin reuptake inhibitors are equivocal at best, although in view of the small sample sizes more controlled research should be conducted to determine their efficacy more definitively (Christenson et al. 1991b; Ninan et al. 2000; Streichenwein and Thornby 1995; Swedo et al. 1989, 1993; van Minnen et al. 2003). Perhaps important differences between OCD and trichotillomania underlie this apparent difference in treatment response. However, several case studies indicated that augmentation of SSRIs with atypical neuroleptics may be beneficial (Epperson et al. 1999; Stein and Hollander 1992), and an open trial suggested that olanzapine may be efficacious as a monotherapy for trichotillomania (Stewart and Nejtck 2003). Interestingly, naltrexone, an opioid-antagonist thought to decrease positive reinforcement, has also been found superior to placebo in reducing trichotillomania symptoms (Christenson et al. 1994a).

Although no double-blind discontinuation studies have been conducted in trichotillomania, evidence from open studies suggests that treatment response gained from pharmacotherapy may not be maintained in the long run (Iancu et al. 1996; Pollard et al. 1991). The absence of a single randomized, controlled trial in pediatric trichotillomania limits treatment recommendations for this population.

Psychotherapy

With respect to behavioral approaches and CBT, a variety of specific techniques have been applied, including awareness training, self-monitoring, aversion, covert sensitization, negative practice, relaxation training, habit reversal, competing response training, stimulus control, and overcorrection. Although the state of the CBT literature justifies only cautious recommendations, habit reversal, awareness training, and stimulus control are generally purported as the core efficacious interventions for trichotillomania.

Successful outcome has been reported with several of the aforementioned interventions. However, because the vast majority of the literature consists of uncontrolled case reports or small case series, confident conclusions cannot be drawn about the specificity of the observed reductions. This is evidenced by the three randomized trials with adults that have been conducted regarding the efficacy of CBT. Ninan et al. (2000) found CBT superior to clomipramine and placebo at posttreatment, and the same pattern was reported by van Minnen et al. (2003) in their randomized, controlled trial of CBT, fluoxetine, and a wait-list condition. Azrin et al. (1980) found that habit reversal was more effective than negative practice. The significant problem of relapse following CBT has been high-

lighted in several studies (Keuthen et al. 2001; Lerner et al. 1998; Mouton and Stanley 1996).

The limited and equivocal treatment literature suggests that there is neither a universal nor a complete response to any treatment for trichotillomania. Controlled studies examining the efficacy of CBT treatments involving habit reversal, pharmacotherapy, and their combination are needed.

Impulse-Control Disorders Not Otherwise Specified

As mentioned earlier, there are a number of other disorders that are not included as a distinct category but are categorized as ICDs not otherwise specified in DSM-IV-TR. These include sexual compulsions (impulsive-compulsive sexual behavior), compulsive shopping (impulsive-compulsive buying disorder), skin picking (impulsive-compulsive psychogenic excoriation), and Internet addiction (impulsive-compulsive computer usage disorder). We briefly describe the nature of each of these behaviors and discuss provisional diagnostic criteria that fall within the framework of ICDs.

Sexual Compulsions

Sexual compulsions have been conceptualized in several compelling ways, most commonly as addictive, impulsive, or compulsive disorders. There is no universal agreement on the nature, or even the definition, of sexual compulsions, but at the core are sexual thoughts, urges, and behaviors that are difficult to resist. Sexual compulsions can be categorized as either paraphilias or paraphilia-related disorders, sometimes referred to as nonparaphilic sexual addictions, with the division between the two based on whether the particular sexual compulsion is outside societal norms, although this boundary is not always easily placed. Whether looking at clinical or forensic populations, it is clear that many individuals have an inability to control their sexual thoughts, urges, and behaviors, despite negative consequences.

The paraphilias form a recognized category of disorders in DSM-IV-TR. These disorders all entail socially deviant sexual thoughts, urges, or behaviors that are generally intense and persistent and either involve nonconsenting persons or lead to distress or impairment in functioning. In contrast, paraphilia-related disorders involve sexual thoughts, urges, and behaviors that are normative but occur with such frequency

or intensity that they lead to distress or impairment in functioning (Kafka 1994, 1997). None of the paraphilia-related disorders are designated as specific disorders in DSM-IV-TR.

Sexual compulsions can be conceptualized as being on an obsessive-compulsive spectrum (Bradford 2001; Hollander and Wong 1995a) because, like OCD, sexual compulsions are characterized by obsessive preoccupations (sexual thoughts, fantasies, and urges) and compulsive repetitive behaviors (in this case, sexual behaviors). The compulsive sexual behaviors differ somewhat from OCD ritual compulsions. OCD rituals are not pleasurable activities engaged in for their own sake but rather are neutral or often irritating and unpleasant behaviors that are engaged in to reduce anxiety. Sexual behaviors generally have an element of pleasure, at least initially, although they may lose their pleasurable quality over time; in this regard, they are more similar to addictions and to ICDs such as pathological gambling.

Sexual obsessions can be a presentation of OCD. Paraphilias and paraphilia-related obsessions can be distinguished from the sexual obsessions that are part of OCD because the latter are characterized by recurrent intrusive sexual thoughts and/or images that are ego dystonic, are morally repugnant, induce anxiety, and usually lead to avoidance and nonsexual rituals rather than to sexual behaviors (Hollander and Wong 1995a). The obsessive thoughts often include the fear or belief that one may actually have somehow committed the feared sexual behavior without knowing it. Therefore, the rituals that accompany sexual obsessions in OCD are repetitive behaviors of a nonsexual nature meant somehow to avoid or undo the distressing sexual thoughts or fears. These rituals can take almost any form. For example, someone with an OCD sexual obsession may have intrusive thoughts of having sexual relations with a stranger he passed on the street and may feel compelled to confess to his wife that he may have had sexual relations with the stranger.

Compulsive Shopping

Many terms have been used for compulsive shopping, including *pathological spending*, *compulsive consumption*, *addictive compulsion*, *addictive shopping*, *uncontrolled buying*, *shopaholism*, and even *mall mania*. The most widely used terms are *compulsive shopping* and *compulsive buying*. There has been considerable debate in the professional literature about the appropriate classification of compulsive shopping. Some investigators have suggested that compulsive shopping is similar to drug or alcohol addiction (Glatt and Cook

1987; Krych 1989), whereas other researchers have focused on the disorder's similarity to OCD, placing it within the obsessive-compulsive spectrum (Frost et al. 1998; Hollander 1993). Hollander (1993), for example, described an impulsive-compulsive spectrum of behavior involving many disorders that he related to OCD, including compulsive shopping. Yet other investigators, following in the tradition of Kraepelin and Bleuler, have favored its classification as a disorder of impulse control similar to disorders such as pathological gambling (Black et al. 2001), whereas some have related compulsive buying to the mood and anxiety disorders (Lejoyeux et al. 1996; McElroy et al. 1995). There is currently little evidence to favor the classification of compulsive shopping as an addiction, an obsessive-compulsive spectrum disorder, or a mood disorder (Black 2000).

Currently, although DSM-IV-TR has no diagnostic category for compulsive buying, individuals with the disorder can be placed in the residual category "disorders of impulse control not otherwise specified." In DSM-IV-TR, ICDs share an inability to resist an "impulse, drive, or temptation to perform an act that is harmful to the person or to others" (p. 663). One could debate whether compulsive shopping is a harmful act, but its secondary consequences frequently are harmful to the individual.

Following the tradition of criteria-based diagnoses, McElroy et al. (1994) developed an operational definition of compulsive shopping for both clinical and research use (Table 19-17). Their definition recognizes that compulsive buying has both cognitive and behavioral components, each potentially causing impairment manifested through personal distress; social, marital, or occupational dysfunction; or financial or legal problems. They exclude from the definition persons whose excessive buying occurs in the context of mania or hypomania. The definition developed by these investigators is now widely used by psychiatric researchers.

Self-Injurious Behavior

Self-injurious behavior (SIB) can be defined as any behavior involving the deliberate infliction of direct physical harm to one's own body without any intent to die as a consequence of the behavior. DSM-IV-TR offers a few diagnoses under which many, but not all, SIBs can be "fitted": trichotillomania or ICDs not otherwise specified (both under Axis I ICDs); Axis II BPD under the criterion "recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior" (p. 710); and stereotypic movement disorder with self-injurious behavior (disorders of infancy, childhood, or adolescence).

TABLE 19-17. Diagnostic criteria for compulsive buying

1. Maladaptive preoccupation with buying or shopping, or maladaptive buying or shopping impulses or behavior as indicated by at least one of the following:
 - a. Frequent preoccupation with buying or impulses to buy that are experienced as irresistible, intrusive, and/or senseless
 - b. Frequent buying of more than can be afforded, frequent buying of items that are not needed, or shopping for longer periods of time than intended
2. The buying preoccupations, impulses, or behaviors cause marked distress, are time-consuming, significantly interfere with social or occupational functioning, or result in financial problems (e.g., indebtedness or bankruptcy).
3. The excessive buying or shopping behavior does not occur exclusively during periods of hypomania or mania.

Source. Reprinted from McElroy S, Keck PE Jr, Pope HG Jr, et al.: "Compulsive Buying: A Report of 20 Cases." *Journal of Clinical Psychiatry* 55:242-248, 1994. Copyright 1994, Physicians Postgraduate Press. Used with permission.

More recently, Simeon and Favazza (2001) proposed a phenomenologically based and clinically relevant comprehensive schema for the classification of all SIBs. They proposed four major categories: stereotypic, major, compulsive, and impulsive. *Stereotypic* behaviors refer to highly repetitive, monotonous, fixed, often rhythmic, seemingly highly driven, and usually content-less (i.e., devoid of thought, affect, and meaning) acts that can range widely in self-inflicted tissue injury from mild to severe or even life-threatening at times. These appear more strongly biologically driven than other types of SIB and are frequently associated with mental retardation (estimates of SIB in people with mental retardation range from 3% to 46% [Bodfish et al. 1995; Winchel and Stanley 1991]), autism, and syndromes such as Lesch-Nyhan, Cornelia de Lange's, and Prader-Willi.

Major SIBs include dramatic and often life-threatening forms of self-injury and involve major and often irreversible destruction of body tissue such as castration, eye enucleation, and amputation of extremities. They are most frequently associated with psychotic states such as schizophrenia but also with intoxications, neurological conditions, bipolar disorder, severe personality disorders, and transsexualism. Common themes involve sin, sexual temptation, punishment,

and salvation. Religious delusions are quite common (Nakaya 1996).

Compulsive self-injury includes repetitive, often ritualistic behaviors that typically occur multiple times daily, such as trichotillomania (hair pulling), onychophagia (nail biting), and skin picking or skin scratching (neurotic excoriations). Of these, trichotillomania is by far the most extensively investigated and the only one diagnostically classified as a discrete disorder in DSM-IV-TR. Compulsive SIBs other than hair pulling, such as skin picking and nail biting, appear to be quite common but have received much less attention in the psychiatric literature.

Impulsive SIBs include skin cutting, skin burning, and self-hitting. These behaviors may be broadly conceptualized as acts of impulsive aggression, not unlike impulsive suicide attempts, where the target of the aggression is the self. These behaviors frequently permit those who engage in them to obtain rapid but short-lived relief from a variety of intolerable states, serving a pathological but life-sustaining function. To maximize treatment effectiveness, patients' highly complex determinants, motivations, and precipitants need to be thoroughly understood at a descriptive and motivational level on an individual basis.

Five descriptive stages have been delineated in impulsive self-injury (Leibenluft et al. 1987). The precipitating event often involves real or perceived loss, rejection, or abandonment. It is followed by the escalation of various types of intolerable affects. After failed attempts to forestall the SIB, the behavior is executed and is typically followed by short-lived emotional relief. Individuals describe various self-states and associated motivations leading up to the self-injury (Favazza 1989, 1996; Leibenluft et al. 1987).

Impulsive SIBs are at times so habitual and repetitive that they can occur on a daily basis without major precipitants, becoming in a sense "compulsions." Indeed, there is some evidence that patients with impulsive SIBs who have obsessional traits are more likely to engage in repetitive self-injury (McKay et al. 2000). Thus, it is probably more accurate to conceptualize impulsive self-injury as encompassing some obsessive-compulsive traits, just as compulsive self-injury may encompass some impulsive traits. Both types of traits facilitate the perpetuation of SIBs via the difficulty in controlling impulses and the tendency to repeat.

Problematic Internet Use

Behavioral problems associated with the Internet have been described with various terms such as *computer addiction*, *Internet addiction (disorder)*, *Internetomania*,

pathological Internet use, and *problematic Internet use*. There are few studies on problematic Internet use, and most have been conducted online and have lacked control populations (DeAngelis 2000). Given these limitations, it appears that between 6% and 14% of those who use the Internet may be problematic users (DeAngelis 2000). In a study of inappropriate Internet use in the workplace, 60% of the participating corporations reported employees engaged in various improper Internet use, and 30% of the companies reported terminating employees for their behavior (Greenfield 2000). In two online studies of Taiwanese college students, including one with more than 753 participants, more than 10% of the students were noted to be potential "Internet addicts" (Chou 2001; Tsai and Lin 2001). With the continued rapid expansion and increased availability of the Internet, the number of Internet-associated behavior problems is likely to grow.

Problematic Internet use can be typified by the Internet user's inability to limit Internet use, which leads to psychological impairment as well as social, educational, and occupational dysfunction (Shapira et al. 2000). Use of the Internet may be associated with numerous risks to the user. The Internet may be utilized by some to access areas that may be a manifestation of their psychiatric illness, for example, compulsive gambling, paraphilias, and compulsive buying. Some small psychiatric studies have demonstrated that many of the evaluated individuals with problematic Internet use have comorbid psychiatric illnesses, especially mood and anxiety disorders (Black et al. 1999; Shapira et al. 2000). These same studies revealed significant distress and daily dysfunction among problematic users. Individuals with problematic Internet use have been noted to prefer computer activities that entail large amounts of interpersonal interaction, such as E-mail, chat rooms, and interactive gaming (Black et al. 1999; Chou 2001; Griffiths 1995, 1996; Shapira et al. 2000; Young 1998). Studies demonstrate that Internet usage is gender and age dependent, with females and mature "addicts" preferring chat rooms that contain sexual material and males and younger "addicts" more tempted by pornographic and gaming sites (Mitchell 2000).

Whether these aberrant and problematic behaviors are the result of a unique disorder or are simply a manifestation of other psychiatric illnesses remains to be seen. Thus, it is imperative that suggested criteria be able to distinguish problematic Internet use from other psychiatric illnesses.

"Behavioral addiction," a variant of the classic addiction model, has been proposed as a way of concep-

tualizing Internet dependence (Bradley 1990; Marks 1990) as well as other disorders such as compulsive shopping, compulsive gambling, compulsive sexual behavior, kleptomania, and overeating. In a survey of 129 college students, Greenberg et al. (1999) found that students with behavioral addictions (Internet, exercise, gambling) tended to also be addicted to substances, including nicotine and alcohol. Psychological dependence is present in both substance and behavioral addictions (Bradley 1990; Marks 1990). In the early 1990s, the term “technological addiction” was coined by Griffiths (1995, 1996) to describe “nonchemical (behavioral) addictions which involve human–machine interaction” (Griffiths 1995, p. 15). Some suggest that technological addictions, like computer and Internet addiction, may be similar to drug addiction due to the presence of common components such as euphoria, tolerance, withdrawal, and relapse (Griffiths 1995). “Technological addictions” have been studied primarily in gamblers addicted to slots or “fruit machines” that use operant conditioning to train gamblers to expect a reward for their behavior (Donegan et al. 1983). Not every pull of the lever results in a reward, and thus the gamble ensues (Griffiths 1993). Griffiths (1995) proposed that two kinds of people become addicted to the Internet: those who are intrinsically attracted to the technology and those who use the technology as a diversion from life’s displeasures. As the technology improves (increased speed, improved graphics), more individuals may be adversely affected by the Internet (Griffiths 1995; Griffiths and Parke 2002).

Conclusion

This chapter has focused on disorders found in the DSM-IV-TR section on ICDs not otherwise classified:

IED, kleptomania, pathological gambling, pyromania, and trichotillomania. Nevertheless, pathological impulsivity may be a crucial construct in understanding a broad range of psychiatric disorders, ranging from common psychotic disorders (e.g., bipolar disorder) to a range of conditions that have emerged together with new lifestyles and technologies (e.g., compulsive shopping, Internet addiction). The development of reliable diagnostic criteria for ICDs has been extremely useful in promoting research on these disorders and has provided a basis for epidemiological work demonstrating the prevalence of these disorders, their high comorbidity and morbidity, and their significant social costs. At the same time, advances in basic research on impulsivity and addiction, together with new methods in clinical research, have led to increased understanding of the overlapping neurocircuitry and neurochemistry that may be involved in a range of these conditions, and this in turn may ultimately lead to a revised nosology of these conditions. Developments in psychometrics and psychobiology have in turn encouraged researchers to conduct rigorous randomized clinical trials of a range of medications and psychotherapies in the ICDs, and a number of effective strategies are now available. Nevertheless, the range of clinical trials in this area remains comparatively limited, and for now clinicians are required to adopt a flexible approach that includes multiple modalities of intervention in the management of the ICDs. Although many patients can be helped by such an approach, much further work is needed to delineate fully the psychobiology of these disorders and to develop effective treatments. There is also a need to develop coordinated approaches to the prevention of disorders, such as pathological gambling, which are influenced greatly by the availability of particular facilities or technologies.

Key Points

- Pathological impulsivity is a useful construct in understanding a broad range of psychiatric symptoms and disorders, including the ICDs not otherwise specified.
- ICDs are highly prevalent and associated with significant disability and costs but receive disproportionately little attention from clinicians and researchers.
- There are now structured diagnostic instruments and standardized rating scales that allow reliable diagnosis and assessment of the ICDs.
- There have been significant advances in our understanding of the neuronal circuitry that mediates impulsivity, as well as in the delineating of the contributing genes and proteins in this circuitry.
- Ultimately, a better understanding of the psychobiological underpinnings of impulsivity, behavior addiction, and other related constructs may lead to changes in our classification of these disorders.
- Although no medication is registered for the treatment of ICDs, a number of randomized, controlled trials have demonstrated the potential value of pharmacotherapy.
- Current clinical practice also emphasizes the need for a comprehensive approach to management that includes psychotherapy and family intervention. Additional work is needed to improve efficacy.

Suggested Readings

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