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≡ The Oxford Handbook of
**IMPULSE CONTROL
DISORDERS**

The Oxford Handbook of Impulse
Control Disorders

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Oxford New York

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Kuala Lumpur Madrid Melbourne Mexico City Nairobi
New Delhi Shanghai Taipei Toronto

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Copyright © 2012 by Oxford University Press, Inc.

Published by Oxford University Press, Inc.
198 Madison Avenue, New York, New York 10016
www.oup.com

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Library of Congress Cataloging-in-Publication Data

The Oxford handbook of impulse control disorders / edited by Jon E. Grant, Marc N. Potenza.

p. cm.

ISBN 978-0-19-538971-5

1. Impulse control disorders—Handbooks, manuals, etc. 2. Compulsive behavior—Handbooks, manuals, etc. I. Grant, Jon E. II. Potenza, Marc N., 1965-

RC569.5.I46O94 2012

616.85'84—dc22

2010054118

9 8 7 6 5 4 3 2 1

Printed in the United States of America on acid-free paper

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PART 1

Introduction and Historical Perspectives on Impulsivity

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Overview of the Impulse Control Disorders Not Elsewhere Classified and Limitations of Knowledge

Jon E. Grant and Marc N. Potenza

Abstract

Several disorders have been classified together in the American Psychiatric Association's *Diagnostic and Statistical Manual* (4th ed.; DSM-IV) as impulse control disorders not elsewhere classified. These impulse control disorders have been grouped together based on perceived similarities in clinical presentation and hypothesized similarities in pathophysiologies. The question exists whether these disorders belong together or whether they should be categorized elsewhere. Examination of the family of impulse control disorders generates questions regarding the distinct nature of each disorder: whether each is unique or whether they represent variations of each other or other psychiatric disorders. Neurobiology may cut across disorders, and identifying important intermediary phenotypes will be important in understanding impulse control disorders and related entities. The distress of patients with impulse control disorders highlights the importance of examining these disorders. More comprehensive information has significant potential for advancing prevention and treatment strategies for those who suffer from disorders characterized by impaired impulse control.

Keywords: impulse control disorders, neurobiology, obsessive-compulsive spectrum

Several disorders have been classified together in the American Psychiatric Association's *Diagnostic and Statistical Manual* (4th ed.; DSM-IV) as impulse control disorders not elsewhere classified (APA, 2000). These impulse control disorders have been grouped together based on perceived similarities in clinical presentation and hypothesized similarities in pathophysiologies. These disorders include pathological gambling, kleptomania, trichotillomania, pyromania, intermittent explosive disorder, and impulse control disorder not otherwise specified. This last diagnosis can be used for other disorders characterized by impaired impulse control that are not defined elsewhere in the DSM, including pathological skin picking, compulsive buying, compulsive sexual behavior, and problematic Internet use.

Pathological Gambling

Pathological gambling is characterized by persistent and recurrent maladaptive patterns of gambling behaviour. It is associated with impaired functioning, reduced quality of life, and high rates of bankruptcy, divorce, and incarceration (Grant & Kim, 2001). Pathological gambling usually begins in adolescence or early adulthood, with males tending to start at an earlier age than females (Ibáñez et al., 2003; Shaffer et al., 1999). Many pathological gamblers engage in illegal behavior, such as stealing, embezzlement, and writing bad checks, to fund their gambling or in an attempt to recover money lost to gambling. Suicide attempts are common and have been reported in 17% of individuals in treatment for pathological gambling (Petry & Kiluk, 2002).

Kleptomania

Kleptomania is characterized by repetitive, uncontrollable stealing of items not needed for their personal use (APA, 2000). Although kleptomania typically begins in early adulthood or late adolescence (McElroy et al., 1991a), the disorder has been reported in children as young as 4 years old and in adults as old as 77 years old (Grant, 2006). Intense guilt and shame are commonly reported by those suffering from kleptomania. Items stolen are typically hoarded, given away, returned to the store, or thrown away (McElroy et al., 1991b). Many individuals with kleptomania (64% to 87%) have been apprehended at some time due to their stealing behavior (Grant, 2006).

Trichotillomania

Pathological hair pulling, trichotillomania, has been defined as repetitive, intentionally performed pulling that causes noticeable hair loss and results in clinically significant distress or functional impairment (APA, 2000). The mean age at onset for trichotillomania is approximately 13 years of age (Christenson & Mansueti, 1999). People report pulling from any body area, most commonly the scalp. The feel of the hair's texture is a frequent trigger. Hair pulling is subject to great fluctuations in severity, with worsening of symptoms often related to stress. Individuals with trichotillomania often pull for more than 1 hour each day, may pull hair from a spouse or children, and often have rituals surrounding the pulling. For example, they may play with the hair, rub it on the face, and occasionally eat the root or hair shaft. Significant social and occupational disability is common, with 34.6% of individuals reporting daily interference with job duties and 47% reporting avoidance of such social situations as dating or group activities (Woods et al., 2006).

Pyromania

The DSM-IV describes pyromania as a preoccupation with fire setting and characterizes the behavior with the following diagnostic criteria: (1) deliberate and purposeful fire setting on more than one occasion; (2) tension or affective arousal before the act; (3) fascination with, interest in, curiosity about, or attraction to fire and its situational contexts; and (4) pleasure, gratification, or relief when setting fires or when witnessing or participating in their aftermath (APA, 2000). The mean age of onset is generally in late adolescence, and the behavior appears to be chronic if left untreated (Grant & Kim, 2007). The urge to set fires is common, and the fire setting

is almost always pleasurable. Severe distress follows the fire setting, and individuals with pyromania report significant functional impairment (Grant & Kim, 2007).

Intermittent Explosive Disorder

Intermittent explosive disorder is characterized by recurrent, significant outbursts of aggression, often leading to assaultive acts against people or property, which are disproportionate to outside stressors and not better explained by another psychiatric diagnosis (APA, 2000). The symptoms of intermittent explosive disorder tend to start in adolescence (approximately age 16) and appear to be chronic (Coccaro et al., 2004; McElroy et al., 1998). Individuals suffering from intermittent explosive disorder regard their behavior as distressing and problematic (McElroy et al., 1998). Outbursts are generally short-lived (usually less than 30 minutes in duration) and frequent (several times per month) (McElroy et al., 1998). Legal and occupational difficulties are common (McElroy et al., 1998).

Pathological Skin Picking

Pathological skin picking is characterized by the repetitive or compulsive picking of skin to the point of causing tissue damage. Individuals with pathological skin picking frequently report shame and embarrassment and the avoidance of social situations (Arnold et al., 2001). People who engage in this behavior typically spend a significant amount of time picking, often several hours each day. Most often they pick the face, but any body part may be the focus—for example, the torso, arms, hands, or legs. The picking behavior may result in significant tissue damage and scarring, sometimes even warranting reconstructive surgery, and in rare cases can be life-threatening (Odlaug & Grant, 2008).

Compulsive Buying

The onset of compulsive buying typically occurs during late adolescence or early adulthood (Black, 1996). The disorder appears to be more common among females than males (Black, 1996; Christenson et al., 1994). Individuals with compulsive buying report repetitive, intrusive urges to shop that are often triggered by being in stores and worsen during times of stress, emotional difficulties, or boredom. Compulsive buying regularly results in substantial financial debt, marital or familial disruption, and legal consequences (Christenson et al., 1994). Guilt, shame, and embarrassment typically follow the buying episodes. Most items are not used

or even removed from the packaging (Christenson et al., 1994).

Compulsive Sexual Behavior

Compulsive sexual behavior is characterized by inappropriate or excessive sexual behaviors or thoughts that lead to subjective distress and/or impaired functioning (Black et al., 1997). Compulsive sexual behavior can involve a wide range of sexual behaviors, often including a mixture of paraphilic and nonparaphilic behaviors (Coleman, 1992; Kafka & Prentky, 1994). Nonparaphilic compulsive sexual behavior involves conventional sexual behaviors (for example, masturbation, promiscuity, pornography) that have become excessive or uncontrolled. Although the compulsive sexual acts are gratifying, the behavior is typically followed by remorse or guilt (Barth & Kinder, 1987). The behavior is often driven by either pleasure seeking or anxiety reduction (Coleman, 1992).

Problematic Internet Use

Problematic Internet use, also termed *Internet addiction*, is characterized by either interfering preoccupations with use of the Internet or excessive use of the Internet for longer periods of time than planned (Shapira et al., 2003). Internet use or the preoccupation leads to clinically significant distress and/or impaired functioning (Shapira et al., 2003). Most people who suffer from Internet addiction have some college education and are employed (Black et al., 1999; Shapira et al. 2000). Individuals with this disorder have reported spending a mean of 27 hours per week on the Internet for “nonessential” purposes (Black et al., 1999). Consequently, these individuals tend to report significant social impairment, financial difficulties, and vocational impairment (Shapira et al., 2000). Individuals with Internet addiction also report that the behavior provides distraction from other concerns, helps them feel more social, and relieves anxiety (Black et al., 1999).

Impulsivity as a Construct Underlying Impulse Control Disorders

Impulsivity has been defined as a predisposition toward rapid, unplanned reactions to either internal or external stimuli with diminished regard for negative consequences (Moeller et al., 2001). Given this definition, multiple psychiatric disorders might be characterized by impulsivity. Many of these disorders are distributed throughout the DSM-IV; for example, bipolar disorder, substance use disorders,

and Cluster B personality disorders all display elements of impulsivity but are categorized in multiple sections of the DSM-IV. Although the impulse control disorders described in the paragraphs above are presently grouped together (if one considers using the “not otherwise specified” category for such disorders as pathological skin picking, compulsive buying, compulsive sexual behaviors, and problematic Internet use), the question exists whether these disorders belong together or whether they should be categorized elsewhere. Should the four disorders currently lacking individualized diagnostic criteria in DSM-IV be operationalized and formally included in this group? Might there be other disorders—relating to self-injurious behavior, binge eating, obesity, hoarding, or cellular phone use—that share features with impulse control disorders and might be considered within this category?

Examination of the family of impulse control disorders generates questions regarding the distinct nature of each disorder: whether each is unique or whether they represent variations of each other or other psychiatric disorders. As mentioned above, impulsivity has relevance to substance use disorders, bipolar disorder, and cluster B personality disorders. Should impulse control disorders be considered nonsubstance addictions, disorders of mood regulation, or disorders of temperament? Why are some of the disorders described as “compulsive”? Are they related to obsessive-compulsive disorder? These questions offer more than just theoretical debates; their conceptualization has implications for the development and implementation of prevention and treatment strategies.

Consider the following case:

Andrew, a 32-year-old single male, described himself as being compulsive. He reports a history, beginning in late adolescence, of uncontrollable sexual behavior. Over the course of a few months, he reports that he became “obsessed” with using pornography online, thinking about it “all day.” He states that his use of pornography started when he began using the Internet for school, and over a period of a few months, it developed into an almost daily ritual. Andrew reports that he currently spends 4–6 hours each day looking at pornography. He reports experiencing a “high” or a “rush” each time he opens an online site. He usually looks at images until he finds the “right” one. In addition, Andrew describes daily thoughts and urges about sex that preoccupy him for 3–4 hours each day. He may even leave work early with projects unfinished so that he can return home and look at pornography or

he looks at pornography at work against his workplace rules. He was recently reprimanded by his supervisor for downloading pornography at work. In addition, he lies to his fiancé, telling her that he is checking sports scores when he is online for long periods of time. Andrew reports feeling “compelled” to look at pornography.

Does Andrew suffer from compulsive sexual behavior? From obsessive-compulsive disorder? Is there a difference? Is his behavior compulsive, impulsive, or both? How might conceptualization of Andrew’s behavior influence its treatment?

Obsessive-Compulsive Spectrum Model

As the case vignette indicates, compulsive sexual behavior, like behaviors in other impulse control disorders, is characterized by repetition and impaired inhibition. Difficulties in demonstrating self-control in compulsive sexual behavior suggest a similarity to the frequently excessive, unnecessary, and unwanted rituals of obsessive-compulsive disorder.

Studies examining rates of comorbid disorders in impulse control disorders have found that 5% to 35% of individuals with an impulse control disorder have co-occurring obsessive-compulsive disorder (Black et al., 1997; Christenson et al., 1991; McElroy et al., 1998; Schlosser et al., 1994), with some having much higher rates of co-occurrence than others (35% of compulsive buyers have obsessive-compulsive disorder compared to only 4.5% of pyromaniacs). Subjects with impulse control disorders may also score high on measures of compusivity (Blanco et al., 2009). Additionally, individuals with some impulse control disorders (kleptomania, compulsive buying) frequently report hoarding symptoms that resemble those of individuals with obsessive-compulsive disorder (Christenson et al., 1994; Goldman, 1991).

There are, however, some clear differences between impulse control disorders and obsessive-compulsive disorder. For example, most individuals with impulse control disorders report their behaviors as pleasurable (Black et al., 1997; Grant & Kim, 2002; McElroy et al., 1998; Schlosser et al., 1994; Stanley et al., 1992) and as often not elicited by an obsession (Grant & Potenza, 2006). In addition, neurobiological studies suggest that individuals with impulse control disorders may have neural features (relatively decreased activation within cortical, basal ganglionic, and thalamic brain regions) distinct from the brain activation patterns observed in cue-provocation studies of obsessive-compulsive disorder (relatively increased cortico-basal-ganglionic-thalamic activity).

Individuals with impulse control disorders are generally sensation seeking, whereas those with obsessive-compulsive disorder are generally harm avoidant, with a compulsive risk-aversive endpoint to their behaviors (Kim & Grant, 2001). Perhaps most important, however, is that impulse control disorders’ response to pharmacotherapy (particularly serotonergic medications), unlike the response of obsessive-compulsive disorder, has been equivocal at best (Grant & Potenza, 2006).

Although the obsessive-compulsive spectrum model has multiple strengths, differences between impulse control disorders and obsessive-compulsive disorder have raised the question of whether other reasonable alternative understandings of these disorders should be entertained (see below). Other considerations include heterogeneities within groups of individuals with impulse control disorders and obsessive-compulsive disorder with the potential for greater overlap within subgroups (Potenza et al., 2009). Recent research has suggested that the obsessive-compulsive spectrum disorders may have various clusters that cohere based on clinical and possibly genetic grounds (Lochner et al., 2005). Further research examining the function of impulsive-compulsive neurocircuitry in multiple disorders, as well as other biological and behavioral features, may lead to findings of subtypes of impulse control disorder that are more like subtypes of obsessive-compulsive disorder and others that have more in common with other disorders such as addictions.

Behavioral or Nonsubstance Addiction Model

To examine whether impulse control disorders should be considered addictions, clinical similarities between these disorders and substance use disorders can be considered. Many, but not all, individuals with impulse control disorders report an urge to engage in a behavior with ultimately negative consequences, mounting tension unless the behavior is completed, and reduction of the urge and/or pleasure after completion of the behavior. These features have led to a description of impulse control disorders as “addictions without the drug,” “behavioral addictions,” or “nonsubstance addictions.”

Unlike compulsions in obsessive-compulsive disorder, behaviors in impulse control disorders typically have a pleasurable quality. Many people with an impulse control disorder report an urge or a craving state prior to their behavior, as do individuals with substance use disorders prior to drinking or using drugs. Additionally, the impulse control

behavior decreases anxiety, as often drinking alcohol does, for example, in alcohol use disorders. Similar to individuals with substance use disorders, many people with impulse control disorders also have problems inhibiting or delaying impulses or gratification.

Strong evidence for considering impulse control disorders as addictions would come from genetic and/or pathophysiology studies. A possible genetic link between impulse control disorders and substance abuse is supported by data indicating that substance use disorders are more common in relatives of subjects with impulse control disorders than in the general population. Studies that have used control groups (trichotillomania, pathological gambling, kleptomania, and compulsive buying) have found that the first-degree relatives of subjects with impulse control disorders were significantly more likely to have substance use disorders.

Consistent with the notion that impulse control disorders share possible neurobiological links to drug addictions, studies have demonstrated frequent co-occurrence between some impulse control disorders (e.g., pathological gambling) and substance use disorders. In addition, a growing body of literature implicates multiple neurotransmitter systems (e.g., serotonergic, dopaminergic, noradrenergic, opioidergic), as well as familial and inherited factors, in the pathophysiology of impulse control disorders, and many findings are consistent with those in drug addictions (Potenza, 2008).

Impulse control disorders have also demonstrated a response to opioid antagonists, drugs that have been successful in treating substance use disorders (Grant et al., 2006; Kim et al., 2001). The finding that impulse control disorders may be responsive to antiaddiction medications may support the inclusion of impulse control disorders within an addictive spectrum.

Ultimately, an assessment of the relationship of impulse control disorders to established addictive disorders needs to consider the respective etiologies. Unfortunately, knowledge of these psychiatric disorders is not yet advanced enough to provide a definite answer to such questions. Nonetheless, evidence suggests that corticostriatal circuitry mediates multiple processes (e.g., reward evaluation and decision making) relevant to impulsivity and compulsion in impulse control disorders and addictions (Chambers et al., 2003; Fineberg et al., 2010).

Affective Disorders Models

Although much data from diverse sources support a close relationship between impulse control disorders

and either obsessive-compulsive disorder or substance use disorders, other nonmutually exclusive models have been proposed. The association of impulse control disorders with mood disorders has led to their hypothesized grouping as affective spectrum disorders (McElroy et al., 1996).

Many people with impulse control disorders report that the pleasurable yet problematic behaviors alleviate negative emotional states. Many individuals with impulse control disorders report symptoms consistent with either depression or a mixed manic state during the impulsive behavior. If impulsive behaviors are associated with an affective state, are they merely symptoms of the primary mood disorder? There is currently no research describing whether impulse control behaviors differ when they appear secondary to mood disorder compared to being independent disorders. Although mood symptoms are common in individuals with impulse control disorders, it is not always clear whether these symptoms precede the impulse control disorder or may be considered reactive.

Because behaviors in impulse control disorder may be risky or self-destructive, the question has been raised whether impulse control disorders reflect subclinical mania or cyclothymia. The elevated rates of co-occurrence between impulse control disorders and depression and bipolar disorder may support their inclusion within an affective spectrum. Additional support for the affective disorders model comes from early reports of a treatment response to antidepressants and mood stabilizers (McElroy et al., 1991a; McElroy et al., 1996).

Several issues complicate the argument for an affective disorders model of impulse control disorders. Depression in impulse control disorders may be distinct from primary or uncomplicated depression; for example, depression in impulse control disorders may represent a response to shame and embarrassment (Grant & Kim, 2002). In addition, rates of co-occurrence of impulse control disorders and bipolar disorder may not be as high as was initially thought (Grant & Kim, 2001, 2002). Finally, the response to antidepressants and mood stabilizers may not be as robust as was initially anticipated (McElroy et al., 1991b), and impulse control disorders respond to opioid antagonists, which have not demonstrated efficacy in mood disorders.

Nonetheless, brain imaging studies have found common regional brain activity differences distinguishing bipolar subjects from controls and pathological gambling subjects from controls during a cognitive task involving attention and response

inhibition (Blumberg et al., 2003); Potenza et al., 2003). Other studies have found a close link between mood symptoms and gambling urges in individuals with pathological gambling (Rømer Thomsen et al., 2009), and the relationship between pathological gambling and major depression appears to be largely related to shared genetic factors (Potenza et al., 2005). For these reasons, the relationship between impulse control disorders and mood disorders requires further investigation and clarification, particularly as appropriate classification has implications for treatment development.

Complexity of Impulse Control Disorders

Although people with impulse control disorders share various symptoms, there also exist substantial heterogeneities. In addition to the models described, other models may also be applicable to impulse control disorders. Further, the models need not be mutually exclusive. On the diagnostic level, perhaps some impulse control disorders would better explained by an addiction model, while others may have more in common with obsessive-compulsive disorder (Potenza et al., 2009). Toward that end, research has tried to find similarities between various impulse control disorders. A cluster analysis has demonstrated that kleptomania, compulsive buying, and intermittent explosive disorder may belong to an “impulsive” subgroup, while trichotillomania, pathological gambling, and compulsive sexual behavior may belong to a “reward deficiency” group (Lochner & Stein, 2006). But even this explanation will likely be too simple, as heterogeneities within each diagnostic group likely contribute to how we might best conceptualize and approach impulse control disorders (van Holst et al., 2010).

On an individual level, perhaps some people with pathological gambling may have more in common neurobiologically with some people with compulsive sexual behavior than they do with other gamblers. Neurobiology may cut across disorders, and identifying important intermediary phenotypes will be important in understanding impulse control disorders and related entities (Fineberg et al., 2010). This may explain why one person with pathological gambling seems to fit the affective model well but another fits the addiction model. A particular impulse control behavior, such as gambling, may be a manifestation of several pathophysiologies. Family history, comorbidity, genetics, neuroimaging, and the treatment response represent means by which the complexity of impulse control disorders may be understood.

Effective Treatment May Depend on Proper Conceptualization

It is likely that heterogeneity exists in impulse control disorders, and that some individuals with these disorders might be more similar to those with obsessive-compulsive disorder and may respond to serotonin reuptake inhibitors, while others might be more similar to those with addictions and may respond to opioid antagonists. While these notions remain speculative and require additional studies to examine their appropriateness, the use clinical symptomatology, including symptoms relating to co-occurring disorders, to guide pharmacological treatment selection is gaining empirical support for impulse control disorders.

Similarly, it could be hypothesized that behavioral therapy selection might be based in part on clinical symptomatology. For example, the prominence of urges and withdrawal symptoms may suggest the use of relapse prevention techniques. Further research is needed to empirically validate specific treatments for individuals with impulse control disorders and help guide the selection of specific treatments for specific groups of people with the disorder. Subtyping impulse control disorders based on clinical similarities to other disorders, the existence of co-occurring conditions, or core features of the behavior (e.g., cravings) may all be useful ways to decide on treatment interventions. Although subtyping needs more research, the early studies suggest that looking beyond the DSM-IV diagnostic criteria may be clinically helpful.

Conclusions

Which disorders to include among impulse control disorders has been a matter of debate. For example, drug addiction could be considered an impulse control disorder given the urge to use drugs, hedonic aspects of drug use, subsequent feelings of guilt or remorse, and limited self-control over stopping. It is possible that one common neurobiological dysfunction may give rise to multiple behavioral symptoms. Some support for this suggestion comes from the fact that many people with one impulse control disorder also report problems with another impulse disorder during their lives, suggesting common etiologies across disorders. A counterargument to this theory, however, is that many people have only one impulse disorder and never have any desire to engage in other behaviors. If the same biology was underlying these behaviors, wouldn't most people switch back and forth between several of the behaviors? May environmental factors, such as

availability of different outlets for impulse control disorder behaviors, influence the resulting expression of a particular condition? Without a clear understanding of the neurobiology that underlies these behaviors, it is not yet possible to say which behaviors share a common etiology.

Andrew's case illustrates not only the similarities among impulse control disorders, obsessive-compulsive disorder, and addictive disorders but also the complexity of behaviors related to impulse control disorders in general. The distress of patients with impulse control disorders highlights the importance of examining these disorders. More comprehensive information has significant potential for advancing prevention and treatment strategies for those who suffer from disorders characterized by impaired impulse control.

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Historical Perspectives on Impulsivity and Impulse Control Disorders

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Abstract

At a recent meeting of the International Society for Research on Impulsivity (ISRI), there were a number of presentations describing different behavioral laboratory measures of impulsivity. After one of the presentations, a question was raised about how a specific behavioral laboratory measure captured the construct of impulsivity. At that point, it was said that perhaps the term *impulsivity* had outlived its usefulness and another, more specific term was needed. This discussion is part of a long history of the study of impulsivity and impulse control disorders and points out an interesting feature of impulsivity: While the clinical literature is consistent in describing the importance of impulsivity in a number of psychiatric disorders, there has been much more inconsistency in the definition and measurement of impulsivity. In spite of this inconsistency, there has been a large body of work on impulsivity over the last 2500 years. This chapter will focus on historical aspects of the study of what impulsivity is, how it should be measured, and what causes it. This review will be of necessity an abridged discussion of the history of impulsivity. To complete an exhaustive review of this literature would require an entire book. While it is not feasible to include all of the work that has been done in this area, an attempt will be made to present a cross section of the vast literature on impulsivity. In this chapter, authors have been grouped together based on theoretical frameworks used to approach impulsivity. Based on this historical perspective, an attempt will be made to answer the question raised above: Has the term *impulsivity* lost its usefulness?

Keywords: impulsivity, history

The Humorists

Any historical perspective on the study of impulsivity must include the Greek physician Hippocrates (ca. 460 BC–ca. 370 BC), considered by many to be the father of modern medicine. Hippocrates devised a system of personality based on four bodily fluids or *humors*. The Hippocratic view of personality, as described and supported by Galen (AD 129–ca. AD 200), continued to have a significant impact on theories of behavior into the nineteenth century. Within this system, personality or *temperament* was based on the relative proportion of four bodily humors: yellow bile, black bile, blood, and phlegm. Impulsive behavior would be most closely aligned

with choleric temperament, which was caused by excessive yellow bile. Individuals with this type of temperament are prone to be aggressive, tense, impulsive, and restless. This personality type was seen as being the opposite of the phlegmatic type, attributed to an excess of phlegm, or mucus. The phlegmatic individual was seen as dull, lazy, slow, controlled, and careful (Titchener, 1921). Although temperament is largely based on the underlying level of bodily humors, environmental factors could have an impact as well, such as climate and diet. Since choleric temperament was seen as “warm and dry,” individuals became more choleric in a warmer climate. Likewise, the phlegmatic temperament was

seen as “cold and moist,” so individuals are more prone to a phlegmatic temperament in a colder climate (Fowler, 1908). Humorism and its thoughts on temperament largely fell out of favor by the end of the nineteenth century.

The Phrenologists, Psychiatrists, and Early Neuroscientists

In the late eighteenth century, Franz Joseph Gall formulated the theory that personality traits can be determined based on the shape of a person’s skull, termed *phrenology* from the Greek *phren*, meaning “mind,” and *logos*, meaning “knowledge.” While this theory would be subsequently discredited, it should be noted that Gall never thought that the skull was “the cause of different dispositions of the mind; he referred the organic influence, whatever it was, to the brain” (Spurzheim, 1846, p. 10). Gall believed that certain brain regions had specific functions and that the levels of these functions could be determined by measuring the bones over the brain regions, since the growth of these bones was determined by the size of the underlying part of the brain. By measuring the size of different skull bones, one could determine “external signs of internal capacities” (Spurzheim, 1846, p.12). Using the size of various parts of the cranium as a guide, Spurzheim proposed a series of 35 fundamental faculties of the mind through which an accurate picture of a personality could be developed. The list of powers proposed by Spurzheim includes regions that have impulsive tendencies, such as “destructiveness,” along with others, such as “cautiousness,” that would increase impulsivity by their absence (Spurzheim, 1834). In a later development, Eulogio Prieto, a Cuban phrenologist, developed a phrenology bust that included six brain regions. One region, named the *impulsive* region or *selfish propensities* comprised seven traits including “vitativeness, alimentiveness, destructiveness, combativeness, acquisitiveness, secretiveness, and cautiousness” (*The Phrenological Journal*, 1905).

Further support for localization of behavior to the brain was provided by the Scottish neurologist and psychologist David Ferrier (1843–1928). In his lectures delivered to the Royal College of Physicians (Ferrier, 1878, p.445), he describes in detail the case of Phineas P. Gage, age 25. “While he was engaged in turning a blasting charge in a rock with a pointed iron bar 3 feet 7 inches in length, 1 and 1 quarter inches in diameter, weighing 13 and a quarter pounds, the charge suddenly exploded. The iron bar was propelled with its pointed end first, entering at

the left angle of the patient’s jaw and passed clear through the head near the sagittal suture in the frontal region, and was picked up at some distance covered with ‘blood and brains.’ The patient was for the moment stunned, but within an hour after the accident he was able to walk up a long flight of stairs and gave the surgeon an intelligible account of the injury he had sustained. His life was naturally for a long time despaired of; but he ultimately recovered and lived twelve and a half years afterwards. He then died (of epileptic convulsions) at a distance from medical supervision, and no post-mortem examination of the brain was made.” Ferrier goes on to point out that although some authors have used the lack of gross motor symptoms in Gage to argue that he “suffered no damage bodily or mental,” Gage’s physician, John Harlow, spoke of dramatic changes in Gage’s personality after the injury: “His contractors, who regarded him as the most efficient and capable foreman in their imply [employ] previous to his injury, considered the change in his mind so marked that they could not give him his place again. The equilibrium or balance, so to speak, between his intellectual faculties and animal propensities seems to have been destroyed. He is fitful, irreverent, indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned in turn for others appearing more feasible” (p. 446). Ferrier goes on to point out that there are similarities in behavior between several patients who suffered isolated prefrontal brain injuries and monkeys after removal of the frontal lobes. These findings would be used later to support a role for the prefrontal cortex in impulsivity and impulse control disorders (described below). However, in the early twentieth century, other theories regarding the origin of personality dominated psychiatry.

One of the earliest psychiatrists to describe what would later become impulse control disorders was Jean-Étienne Dominique Esquirol (1772–1840). In his book *A Treatise on Insanity* (1845), Esquirol described a group of disorders that he termed *monomania*. These disorders were characterized by chronic symptoms without fever and a “partial lesion of the intelligence, affections or will” in which patients “seize upon a false principle, which they pursue without deviating from logical reasonings, and from which they deduce legitimate consequences, which

modify their affections, and the acts of their will” (p. 320). One class of monomania that most closely resembles impulse control disorders was instinctive monomania, which Esquirol described as follows: “The patient is drawn away from his accustomed course, to the commission of acts, to which neither reason nor sentiment determine, which conscience rebukes, and which the will has no longer the power to restrain. The actions are involuntary, instinctive, irresistible” (p. 320). One of the instinctive monomanias, termed by Esquirol *Incendiary Monomania*, characterized by an “instinctive desire to burn” (p. 357), would encompass the impulse control disorder pyromania. Although the monomanias as defined by Esquirol included many of the symptoms associated with the current conceptualization of impulse control disorders, one main difference between his diagnostic system and our current diagnostic system is that Esquirol included patients with and without psychotic symptoms under the same diagnostic category, as can be seen from this quote: “Monomaniacs, like other insane persons, are subject to illusions and hallucinations, which often alone characterize their delirium, and are the cause of the perversions of their affections, and the disorder of their actions” (p. 328).

Clinical Psychology and Psychiatry

One of the most influential theories of personality in the early twentieth century was psychoanalysis. In his book *Three Contributions to the Theory of Sex* (1920), Sigmund Freud describes cruel impulses, stating, “As we know, a thorough psychological analysis of this impulse has not as yet been successfully accomplished; we may assume that the cruel feelings emanate from the impulse to mastery and appear at a period in the sexual life before the genitals have taken on their later role.” Later, a follower of Freud, Otto Fenichel, described an *impulse neurosis*, in which a person had intense ego-syntonic pathological impulses that are often irresistible.

Patients with an impulse neurosis are described as having difficulty postponing immediate reactions or actions. Fenichel saw as the source of these impulses a fusion of instinctual urges and defensive strivings (Fenichel, 1945). As can be seen from Fenichel’s description, the concept of impulse neurosis focuses on two constructs regarding impulsivity: first, that it is driven by underlying impulses and, second, that individuals with impulse neuroses cannot postpone actions or reactions. These two concepts are consistently seen in disorders that today are known as *impulse control disorders*.

Emil Kraepelin, in his book *Lehrbuch Der Psychiatrie*, as abstracted and translated into English by Defendorf (1904), describes *impulsive insanity*, in which there is a “development of morbid tendencies and impulses which may predominate over volition or appear only in paroxysms. These acts, which appear without motive, are performed because of an irresistible impulse. These impulses do not arise because of a conscious plan, but appear suddenly, and are quickly executed, and often quite indistinct, thereby causing the actions to appear unpremeditated, purposeless, and even absurd” (p. 389). Kraepelin goes on to describe cases of impulsive insanity, including such disorders as fire setting (pyromania), stealing (kleptomania), and morbid impulses to destroy and kill. He separates impulsive insanity cases from common criminal behavior through the fact that the “criminal sets fire, kills, and steals, but he does it from selfish motives, and for some definite purpose, perhaps to do some one injury. . . on the other hand, impulsive insanity approaches very closely some forms of compulsive insanity” (p. 391). In this definition of impulsive insanity, Kraepelin lays out a list and description of disorders that are grouped together as Impulse Control Disorders Not Otherwise Specified in the *Diagnostic and Statistical Manual of Mental Disorders*, version IV (APA, 2000). Interestingly, Kraepelin goes on to state: “It is of the greatest importance that the patients do not become addicted to the use of alcohol” (p. 391). An association between impulse control disorders and substance abuse that has been noted by modern authors (reviewed in Moeller, 2009).

In his book *Textbook of Psychiatry* (1924), Eugene Bleuler describes a number of different impulses, including the nutrition impulse related to eating, the suicide impulse, the sex impulse, and the ethical impulse. However, he describes a specific group of impulses, termed *morbid impulses*, that most closely match the modern construct of impulsivity. Morbid impulses are “impulses for actions, which are accomplished unexpectedly, without real reflection or with inconsistent reflection, or without the assent of the whole personality” (p. 149). Bleuler goes on to say that morbid impulses are often “distinguished by violence, hastiness, skill and regardlessness of the interests of others as well of their own” (p. 149). In agreement with Kraepelin, Bleuler also describes morbid impulses to set fires (pyromania) and to steal (kleptomania). He also classifies kleptomania and pyromania under the heading of *impulsive insanity*, as did Kraepelin.

Bleuler further expounds on the broader category of *impulsive actions*, which “have one thing in common, namely, they are carried out without the cooperation of reflection and aimful willing” (p. 149). He defines impulsive actions as “various indefinable kinds of actions carried out suddenly and without proper deliberation” (p. 149).

Individual Differences Psychology

One of the founding fathers of individual differences psychology, Hans Eysenck (1916–1997), described a theory of personality structure based on factor analysis. At the lowest level in the analysis were specific responses to an experimental test or experiences of everyday life, which may not be characteristic of the individual; at the second level were habitual responses, which tend to recur under similar circumstances; and at the third level were organization of habitual acts into traits. *Traits* were theoretical constructs based on observed intercorrelations of habitual responses. Finally, at the highest level, traits are organized into a general type, based on observed intercorrelations of traits. Based on this type of factor analysis, Eysenck initially described two dimensions of personality, extraversion and neuroticism (*Dimensions of Personality*, 1947). Later, a third dimension, psychotism, was added. *Psychotism* referred to a trait that was not limited to clinical psychosis, and was developed based on studies of prisoners and criminals. In the system of personality as formulated by Eysenck, extraversion was characterized as being talkative, high on positive affect, and outgoing.

Neuroticism was characterized as being high in negative affect, such as anxiety and depression. Psychotism was characterized by nonconformity, hostility, and impulsivity. Eysenck developed the Eysenck Personality Inventory (EPI) to measure components of personality. The EPI measured the two global aspects of personality, extraversion and neuroticism. Within the EPI, impulsivity was one of the traits that make up extraversion, along with sociability. Later, the Eysenck Personality Questionnaire (EPQ) was developed, including all three personality traits: extraversion, neuroticism, and psychotism. Within the EPQ, impulsivity items are located within the psychotism questions. In Eysenck's system of personality, impulsivity was seen as a combination of four habits: impulsivity narrow, nonplanning, liveliness, and risk taking (Action, 2003). Later, a specific scale to measure impulsiveness was developed. The I₇ scale (Eysenck & Eysenck, 1985) is composed of 54 items with

subscales for impulsivity, venturesomeness, and empathy.

Jeffrey Gray (1934–2004) proposed a variant model of personality based on Eysenck's model. Gray's theory (known as *Reinforcement Sensitivity Theory* [RST]) consisted of two main aspects of emotion that determine behavior: a behavioral approach system (BAS) and a behavioral inhibition system (BIS). The BAS is sensitive to reward and activates behaviors in response to reward cues. The neurotransmitter dopamine was thought to play a role in the BAS. Extraversion, impulsivity, and sensation seeking were associated with the BAS.

The BIS is sensitive to punishment and fear-related stimuli and is theorized to be associated with the neurotransmitter serotonin.

Other researchers developed models of personality based on theoretical relationships to underlying biology, including C. Robert Cloninger (1944–) with his Tridimensional Personality Questionnaire (TPQ) and his Temperament and Character Inventory (TCI) and Marvin Zuckerman (1928–) with his Sensation Seeking Scale (SSS) and the Zuckerman-Kuhlman Personality Questionnaire (ZKPQ). The TCI is composed of items that measure seven subscales, including novelty seeking, harm avoidance, reward dependence, persistence, self-directedness, cooperativeness, and self-transcendence. Impulsiveness is a subscale of novelty seeking. The ZKPQ assesses five basic dimensions of personality, including activity, aggression-hostility, impulsive sensation seeking, neuroticism-anxiety, and sociability.

Scott J. Dickman defined impulsivity as “the tendency to act with less forethought than do most individuals of equal ability and knowledge” (Dickman, 1993 p. 151). In his research, Dickman found evidence of two distinct subtypes of impulsivity, which he termed *functional* and *dysfunctional*. The functional impulsive individual acts with little forethought and is rewarded for this type of behavior, while the dysfunctional impulsive individual acts with little forethought in spite of the frequently negative consequences of the behavior. Dickman (1990) developed the Functional and Dysfunctional Impulsivity Inventory to measure both aspects of impulsivity.

Dysfunctional impulsivity, as measured by the scale, correlates highly with other questionnaire measures of impulsivity, such as the I₇, while functional impulsivity does not. Based on this fact, it has been argued that dysfunctional impulsivity more closely approximates the clinical construct of

impulsivity, whereas functional impulsivity could be more appropriately called *spontaneity* (Eysenck, 1993). The notion that impulsivity is by definition dysfunctional is also more in keeping with the historical use of the term both clinically and by the lay public.

One of the most widely cited authors on impulsivity is Ernest S. Barratt (1925–2005). Barratt formulated a theory of impulsivity integrating cognitive, behavioral, environmental, and biological factors into a general systems model. Studying impulsivity as a construct for over 40 years, using questionnaires as well as behavioral and electro-physiological measures, Barratt believed that one of the key features of impulsivity was a deficit in cognitive tempo and rhythm. However, he saw impulsivity as a multidimensional construct with three subdivisions, as measured by the Barratt Impulsiveness Scale (BIS). The current version of the BIS (BIS-11) has become one of the most widely used measures of impulsivity. The BIS-11 is a 30-item questionnaire with three subscales, Nonplanning, Attentional, and Motor impulsivity. Barratt believed that a true measure of impulsivity should have construct validity and thus should show elevated impulsivity in clinical groups that are known to have higher impulsivity.

Barratt used this method to refine his BIS in 11 different versions, administering the scale to incarcerated subjects who had a history of impulsive violence and to substance-abusing subjects as well as healthy controls (Patton et al., 1995). Since then, the scale has been administered to many groups of subjects who are clinically known to have a problem with impulsivity, including those with borderline personality disorder, antisocial personality disorder, and bipolar disorder, all showing elevated impulsivity as measured by the BIS (Fossati et al., 2004; Moeller et al., 2002; Swann et al., 2003). In spite of the utility of the BIS in distinguishing impulsive groups from less impulsive groups of subjects, Barratt argued that the best measure of impulsivity for individual subjects was a battery of measures that included behavioral, questionnaire, and physiological measures. He believed that ultimately, an *impulsivity index* could be obtained using such a battery that would have much greater clinical utility than any one measure.

Although Barratt studied impulsivity for over 40 years, he did not propose a formal definition for it until late in his career. Working with Moeller, Barratt, Dougherty, Schmitz, and Swann (2001), Barratt proposed what he called a *clinical* definition

of impulsivity: “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others” (p. 1784).

This definition incorporated several features of impulsivity as previously described by Bleuler, in which impulsivity has the features of rapid action without forethought, “accomplished unexpectedly, without real reflection,” along with the lack of regard for future consequences. This real or apparent lack of regard for negative future consequences of behavior has come to be the focus of a behavioral definition of impulsivity, as described below.

Behavioral Psychology

Based on the work of B. F. Skinner on experimental analysis of behavior through the use of operant methods, several behavioral tasks have been used to study impulsivity. A definition of impulsivity was developed based on performance on one type of these tasks. Ainslie (1974) defined an *impulse* as the choice of a small, short-term gain at the expense of a large, long-term loss. In developing this definition, Ainslie cites the work of Mowrer and Ullman (1945), who pointed out the need for examination of the temporal pattern of punishment and reward to explain “persistent non-integrative behavior” in which behavior with predominantly painful consequences persists indefinitely. The exact nature of this temporal effect is as follows:

“If a response is immediately followed by a rewarding state of affairs (drive reduction), the tendency for that response to occur in the same problem-situation in the future is reinforced more than if there is a temporal delay between the occurrence of the response and the reward” (p. 66). Likewise, if a response is immediately followed by punishment, the effect is greater than if the punishment is delayed.

This leads to the conclusion that if an act has two consequences, one rewarding and the other punishing, which would be equal if they occurred simultaneously, the effect of the order of the consequences will be a significant determinant of the behavior: If the punishment occurs first and the reward later, the response will be inhibited, and if the reward comes first and the punishment later, the response will be reinforced. A number of behavioral tasks have been developed to measure the preference for choosing a small reward at a short delay versus a larger reward at a longer delay, which can be used in both humans and animals. Using these behavioral tasks, a subject

(animal or human) will choose a smaller, sooner reinforcer over a larger, delayed reinforcer based directly on the size of the reinforcers and inversely on the delay between reinforcers. These functions describe a hyperbolic curve that can be used as a quantifiable measure of self-control.

The work of Ainslie, Howard Rachlin, Alexandra Logue, and others focuses on self-control, which they considered to be the opposite of impulsivity: commitment to a course of action with long-term benefits over short-term gains. This definition of impulsivity treats impulsivity as a unitary construct, that is useful for measurement but is not consistent with clinical aspects of impulsivity previously described. One aspect of the clinical construct of impulsivity, which is not captured by this definition, is the rapid, unrestrained action component.

Other behavioral tasks have been developed that capture this aspect of impulsivity. However, as will be discussed, these measures often depend on other aspects of behavior besides impulsivity, such as attention and memory.

Developed as a test of brain damage in human subjects by Rosvold et al. (1956), the continuous performance test (CPT) as initially formulated presented two attentional tasks, labeled X and AX, with the AX task designed to be the more difficult version. The task originally used a revolving drum on which two series of 31 letters each were mounted side by side. One of these series presented the X task, and the other presented the AX task. The drum revolved at a rate such that the letters came into view every 0.92 seconds. Subjects were instructed to press a response key if the letter X appeared in the X task and if the letter X appeared after the letter A in the AX task. A response was scored as correct if the response key was pressed within 0.69 second after the presentation of the correct letter, with a 0.23-second interstimulus interval. The length of testing on the task was 5 minutes for each set of stimuli in children and 10 minutes for each set of stimuli in adults. Using this task, Rosvold et al. showed that brain-damaged adults showed a rate of correct detections in the X task similar to that of non-brain-damaged adults. However, brain-damaged adults showed a significantly lower number of correct responses on the AX task compared to non-brain-damaged adults, even after adjusting for differences in IQ. Rosvold et al. hypothesized that the performance differences between the two groups on the task were due to impairment in attention in the brain-damaged individuals.

Later studies would use a computerized variant of the CPT to study children with attentional deficit hyperactivity disorder (ADHD). This test also showed a poorer performance in ADHD children compared to non-ADHD controls (Halperin et al., 1990; Seidel & Joschko, 1990).

In addition to the deficit in attention seen in ADHD subjects, impulsivity is a well-described phenomenon in these individuals (Buttross, 1988). In their study of ADHD children, Halperin et al. (1988) noted that there were several types of information that could be obtained from the CPT. The number of correct detections of matching stimuli or "hits," as well as the number of errors of omission or "misses," could be determined. In addition, the number of commission errors or "false alarms," in which the subject responded to an inappropriate stimulus, could be determined. Halperin et al. argued that commission errors represented a response to a stimulus prior to the complete processing of information. This "acting without thinking" behavior is consistent with impulsivity. By examining correct detections, omission errors, and commission errors, information about impulsivity as opposed to attention and memory can be obtained (Halperin et al., 1988).

Several variants of the CPT have been developed. One variant is the immediate and delayed memory task (IMT/DMT; Dougherty et al., 2002). Studies have shown higher numbers of commission errors on the IMT/DMT in clinical groups that exhibit impulsive behaviors, such as children with conduct disorder, adults with substance abuse, and adults with bipolar disorder (Dougherty et al., 2003; Moeller et al., 2005; Swann et al., 2003). Other studies have shown a modest but significant correlation between commission errors on the IMT/DMT and impulsivity measured by questionnaires such as the BIS, supporting commission errors as capturing at least a component of the clinical construct of impulsivity (Moeller et al., 2004).

Tests of reaction time date to at least the mid-nineteenth century. One of the early behavioral scientists to study reaction time was Franciscus Donders (1818–1889). Donders developed tasks to measure and compare reaction time between simple response tasks and choice tasks. These tasks developed into two tasks that are now widely used as measures of impulsivity: go/no-go tasks and go/stop or stop signal tasks. Logan, Cowan, and Davis (1984) proposed a model of response inhibition using a reaction time task that occasionally presented a "stop signal" notifying the subjects to

inhibit their response. They proposed a model of response inhibition that assumed that the “processes responding to the reaction time stimulus race against the processes responding to the stop signal” (p. 277). Thus, it would be possible to measure a *stop signal reaction time* to gauge the effectiveness of processes responsible for behavioral inhibition. Logan et al. noted that stop signal reaction times became shorter as the delay between the go stimulus and the stop stimulus increased.

Further, it is harder to suppress a response when the stop stimulus is presented closer to the go stimulus. Thus, by varying the interval between the two stimuli (*stimulus onset asynchrony* [SOA]), the shortest period in which subjects could inhibit a response could be determined. Logan, Schachar, and Tannock (1997) would later propose a definition of impulsivity based on performance on the task: “We have found it useful to operationalize impulsivity in terms of the ability to inhibit prepotent courses of action: People who are impulsive have trouble inhibiting action, whereas people who are not impulsive find it easier to do so” (p. 60). Logan et al. (1997) supported this definition of impulsivity based on the fact that, in healthy controls, subjects with higher scores on a subset of questions from the EPI related to extraversion also had longer stop signal reaction times. Impaired performance on the go-stop task has been shown in subject groups known to have higher impulsivity, such as children with ADHD (Pliszka et al., 1997) and drug abusers (Fillmore & Rush, 2002).

It has been noted that the go/no-go task is a variant of the go/stop task in which the SOA is always zero (Band & van Boxtel, 1999). Newman, Widom, and Nathan (1985) administered a go/nogo discrimination task to psychopathic subjects and controls. The task involved a successive series of stimuli. Subjects were rewarded for responding to correct cues and punished for responding to incorrect cues.

Newman et al. (1985) defined passive avoidance, or errors of commission in which subjects fail to withhold a response to a no-go stimulus, and errors of commission in which subjects fail to respond to an appropriate go stimulus. Results of the study showed that psychopathic subjects demonstrated a passive avoidance deficit when avoidance required subjects to inhibit a response that frequently led to reward. Errors of commission have since then been used as a measure of impulsivity in a number of studies in patient groups, including children with ADHD (Trommer et al., 1988) and persons with drug dependence (Lane et al., 2007).

An additional utility of go/no-go tasks is that they lend themselves to use in functional neuroimaging and neurophysiology in order to localize the brain regions associated with behavioral inhibition. A large number of studies have used go/no-go tasks to assess brain function associated with behavioral inhibition during functional magnetic resonance imaging (fMRI). Functional MRI studies in healthy controls support the importance of the prefrontal cortex, especially the orbital cortex, in response inhibition (Horn et al., 2003; Tamm et al., 2002). Other studies have found differences in brain activation between impulsive patient groups, including Bipolar and Personality Disordered subjects (Altshuler et al., 2005; Vollm et al., 2004), supporting the utility of the task.

Animal Research

In addition to the utility of behavioral tasks as tools for functional imaging and neurophysiology studies, an additional advantage of behavioral tasks over self-report measures of impulsivity is that they can be administered to animals. This allows for direct examination of effects of experimental manipulation of brain structure and function on behavior that cannot be done in human research.

One of the more widely used behavioral measures for animal studies relies on the choice of a small reward with a short delay or a larger reward with a longer delay. This measure is analogous to the tasks used in humans described above, in which impulsivity was defined as a “small, short-term gain at the expense of a large, long-term loss” (Ainslie, 1974, p. 485). Using an adjusting amount procedure in which rats chose between a delayed, fixed (large) amount of water and an immediate, adjusting (small) amount of water. Richards, Sabol, and de Wit (1999) showed that rats given an acute dose of methamphetamine valued delayed large rewards more highly than after they were given saline doses. However, after large, behaviorally disruptive doses of methamphetamine were given, rats valued delayed large rewards less than after saline treatment. These studies pointed out the importance of dose and frequency on behavioral effects of stimulants on impulsivity. Other studies have used these types of delayed reward task to examine the effect of brain lesions on performance.

Cardinal, Pennicott, Sugathapala, Robbins, and Everitt (2001) showed that selective lesions of the nucleus accumbens core lead to persistent choice of smaller, shorter delayed rewards in rats. In contrast, damage to the anterior cingulate cortex and medial

prefrontal cortex had no effect on choice behavior. Based on this finding, the authors suggested that “dysfunction of the nucleus accumbens core may be a key element in the neuropathology of impulsivity” (p. 2499).

The effect of experimental manipulation of brain neurotransmitters on behavior on the delayed reward task has also been examined. Winstanley, Dalley, Theobald, and Robbins (2003) gave rats infusions of the serotonergic neurotoxin 5,7-Dihydroxytryptamine (5,7-DHT) and explored the effect of serotonergic lesions on choice behavior in a delayed reward task. Results showed that although the 5,7-DHT-treated rats showed a depletion of forebrain serotonin levels of over 85%, there was no effect on choice behavior. However, the effect of amphetamine on increased choice for the large reward was attenuated by 5-HT depletion. The authors concluded that “the ability of amphetamine to decrease impulsivity is not solely due to its effects on dopaminergic systems, but may also depend on serotonergic neurotransmission” (p. 320). Another study by the same author (Winstanley et al., 2006) examined levels of dopamine, serotonin, and their metabolites in the medial prefrontal cortex and orbitofrontal cortex of rats using *in vivo* microdialysis during a delay-discounting model of impulsive choice.

That study found significant increases in serotonin in the medial prefrontal cortex and increased dopamine metabolite levels in the orbitofrontal cortex during task performance compared to that of yoked control animals. The authors interpreted these findings as suggesting “a double dissociation between serotonergic and dopaminergic modulation of impulsive decision-making within distinct areas of frontal cortex” (p. 106). These studies point out the complexity of neurotransmitter interactions on impulsive choice.

Other tasks that have analogies with human response inhibition tasks have been developed. One of the more widely used tasks of this type is the five-choice serial reaction time task (5-CSRT; (Carli et al., 1983). In this task, rats are trained to detect 0.5-second flashes of light presented randomly in one of five locations with a fixed intertrial interval of 5 seconds. Correct responses are rewarded with food and incorrect responses are punished by a time-out. The 5-CSRT has been used as a screening tool for medication development. Day et al. (2007) evaluated the effects of ciproxifan, a nicotine, and the histamine H₃ receptor antagonist in rodents using the 5-CSRT. Results showed that ciproxifan

reduced premature responding, which the authors argued provides evidence that ciproxifan may reduce impulsivity. Other authors have used the 5-CSRT to study the role of serotonin function in impulsivity.

Winstanley, Theobald, Dalley, Glennon, and Robbins (2004) studied the effects of serotonin manipulation on performance of the 5-CSRT using the serotonin neurotoxin 5,7-DHT and serotonin 2A (5-HT-2A) and serotonin 2C (5-HT-2C) receptor antagonists. Results showed that 5,7-DHT lesions significantly reduced forebrain levels of serotonin and increased levels of premature responding, whereas the serotonin 2A antagonist M100907 decreased premature responding in sham-operated controls but had no effect in serotonin-depleted rats. The serotonin 2C antagonist SB242084 increased premature responding in all animals. These findings are consistent with human studies linking serotonin to impulsive behaviors (Linnoila et al., 1983).

One type of task that has been used extensively in animal behavioral studies is the differential reinforcement for low rate of responding (DRL) described in Monterosso and Ainslie (1999). In this task, the animal is trained to respond at a set rate for a reinforcer. Premature responses reset the time required between behaviors. In this model, the premature responses are considered the impulsive response. DRL tasks have been used in human studies. Children with ADHD ages 6–8 years performed more poorly on a DRL 6-second schedule than matched controls (Gordon, 1979). Evenden (1998a) used a version of the DRL, the fixed consecutive number schedule (FCN), as a measure of impulsivity. In the FCN, rats are required to press one lever (the FCN lever) in a two-lever operant chamber a fixed minimum number of times before pressing the other (reinforcement) lever.

If the rat presses the reinforcement lever prior to completing the required number of responses on the FCN lever, a time-out occurs and the rat must restart the sequence on the FCN lever. Evenden (1998b) used the FCN procedure to examine the effects of drugs on impulsivity. He found that the tricyclic antidepressant imipramine increased the average number of responses in the FCN and produced a shift in the responses, which he interpreted as “possibly reflecting a reduction in impulsivity” (p. 295).

Discussion

As can be seen from this historical review on impulsivity, over the last century three overall perspectives

on impulsivity have been developed. In one perspective, there is a body of clinical literature that has focused on the categorization and symptoms of pathological groups who clinically have problems with impulsive behavior. This literature, which can be traced back to the writings of Ferrier, Kraepelin, and Bleuler, describes impulsivity in patients with certain conditions or behavioral problems, such as brain injury, kleptomania, and pyromania. Impulsivity here is seen as a categorical construct. In another perspective, impulsivity is seen as a facet of personality, usually as a subfacet of another dimension, such as extraversion or sensation seeking. In this perspective, impulsivity is a continuous variable ranging in level from high to low. Finally, there is a behavioral perspective, in which impulsivity is defined based on performance on various behavioral tasks. In this perspective, impulsivity is again a continuous variable.

Definitions of impulsivity in the three perspectives have ranged from Bleuler's "actions that are carried out without the cooperation of reflection and aimful willing" and Dickman's "tendency to act with less forethought than do most individuals of equal ability and knowledge" to behavioral definitions based on the "ability to inhibit prepotent courses of action" and "choice of a small, short-term gain at the expense of a large, long-term loss" of Ainslie and Logan.

Each of these approaches has some merit. Impulsivity as a clinical construct has clinical utility in diagnosis and treatment and is widely accepted by the lay public. However, impulsivity as a clinical construct is multidimensional and, as such, is less likely to be associated with a specific neurotransmitter, gene, or brain region. Impulsivity as a behavioral construct allows for the measurement of impulsive behavior in animals and in brain imaging. This provides important information about the biological basis of behavior. However, each of these behavioral measures appears to capture only a subset of the behavioral problems associated with the clinical construct of impulsivity.

In the final analysis, it may be that a historical perspective on impulsivity provides the most complete picture of what impulsivity is. Impulsivity is both a clinical disorder and a personality trait. It is a multidimensional construct that is associated with several discrete types of behavior. Returning to the question raised at the beginning of this chapter, has the term *impulsivity* lost its meaning? I believe, based on this review, that it is clear that impulsivity remains an important clinical

construct that spans a long history of human thought.

However, there are important issues that have been raised regarding the lack of consistency in the use of the term *impulsivity*. In the future study of impulsivity and impulse control disorders, it will become increasingly important to be specific in defining what is being referred to. As more behavioral and biological measures are developed, each of which may be called "impulsivity," it will be important to retain the clinical perspective on what kinds of behavioral problems patients with impulse control disorders have. At the same time, use of more than one measure in patient populations is more likely to give a complete picture of behavior in these subjects. Returning to the recommendations of Barratt, perhaps an impulsivity index would provide the best measure of impulsivity.

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PART **2**

Phenomenology and
Epidemiology of
Impulse Control
Disorders Not
Elsewhere Classified

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Current Classification of Impulse Control Disorders: Neurocognitive and Behavioral Models of Impulsivity and the Role of Personality

Ashwini K. Padhi, Ali M. Mehdi, Kevin J. Craig, and Naomi A. Fineberg

Abstract

Impulse control disorders (ICDs) are common disabling disorders that have impulsive behavior as a core feature. They emerge early in life and run a chronic lifelong course. They are assumed to lie at the severest end of a continuum of impulsivity that connects normal with pathological states. People with ICDs experience a drive to undertake repetitive acts. Although the consequences are damaging, performance of the impulsive act may be experienced as rewarding, or alternatively may relieve distress, implicating dysfunction of the neural circuitry involved in reward processing and/or behavioral inhibition. Clinical data are increasingly pointing toward an etiological association between some ICDs, such as pathological gambling and addiction, and others, such as trichotillomania and compulsive disorders. Comorbidity with other psychiatric disorders is also common, and hints at overlapping psychobiological processes across several diagnostic groups. The results of neurocognitive studies suggest that impulsivity is multidimensional and comprises dissociable cognitive and behavioral indices governed by separate underlying neural mechanisms. For example, trichotillomania may primarily involve motor impulsivity, whereas problem gambling may involve reward impulsivity and reflection impulsivity. Exploring neurocognitive changes in individuals with ICDs and other mental disorders characterized by poor impulse control, and among their family members, may help to elucidate the underpinning neurocircuitry and clarify their nosological status.

Keywords: impulse control disorders, ICD, impulsivity, neurocognitive, personality

Introduction

The impulse-control disorders (ICDs) constitute a group of psychiatric disorders characterized by the repeated inability to resist an impulse, drive, or temptation to perform an act that is harmful to the person or others (APA, 2000). The act may be unplanned or premeditated but usually has the qualities of impetuosity and lack of deliberation. Individuals with ICDs experience extreme difficulty in controlling impulses despite their negative consequences. These disorders appear to be relatively common, frequently go unrecognized, and may constitute greater threats to personal health than is often appreciated (Potenza & Hollander 2002). Recently, however, the public health implications of

disorders such as problem gambling have become the focus of media attention, driving initiatives to better understand and treat these disorders.

Impulse control disorders have always been problematic in terms of psychiatric classification. Historically, they have constituted a miscellaneous group of disorders, arguably with little in common apart from the core feature of impulsive behavior. As a group, ICDs have also attracted relatively little scientific interest. However, as cognitive neuroscience begins to unmask the mechanisms underlying behavioral inhibition and decision making, our understanding of these disorders is growing. Some disorders, such as pathological gambling, appear to be driven by reward-dependent learning and are

thought to have much in common with addiction. The compulsive aspects of many ICDs have become another area of interest. For example, trichotillomania has been compared to obsessive-compulsive disorder in an effort to understand the mechanisms underlying compulsive behaviour (Chamberlain et al., 2007a; Fineberg et al., 2010).

From a sociological perspective, it has been suggested that the societal norms supporting impulse control and delay of gratification are weakening in favor of present-oriented expression of impulses (Tavares, 2008). Therefore, ICDs represent the most extreme form of this progression. As opportunities for the expression of impulsive behavior increase, so does the number of candidate ICDs. For example, recent research has described compulsive-impulsive Internet usage (Block, 2007).

It is timely, therefore, to review the current clinical classification of ICDs in order to examine areas of strength and weakness in our knowledge and consider whether some disorders may be better described. Given that disorders characterized by impaired impulse control are also currently classified outside the DSM-IV-TR ICD grouping, it is relevant to (1) review the evidence that supports a close nosological relationship between ICDs as currently defined; (2) identify the key cognitive and behavioral dimensions of “impulsivity” that may lie at the heart of each of these disorders; (3) identify comorbidity patterns and associated “symptom domains” that may hint at relationships with disorders classified elsewhere; and (4) consider whether the arguments are strong enough to support additional individual diagnoses such as impulsive-compulsive shopping, Internet use, or compulsive sexual behavior or (5) whether a different conceptualization for ICDs, as disorders defined by a common core of “behavioral impulsivity” and accompanied

by a range of associated behaviors that may vary according to personal and contextual factors, including acts as yet not defined, might be of relevance in advancing our understanding and future research in this ubiquitous and evolving family of disorders. Such research needs to focus on the neural systems involved so that we can explain the ICDs in terms of the anatomy and function of the relevant brain areas. Greater understanding of the similarities and differences among these disorders may challenge our existing classification system.

History

In 1816, Matthey coined the term *klopemanie* to indicate a condition characterized by the compelling impulse to steal a worthless object “borne out of pure insanity” (Segrave, 2001). At around the same time, impulsive fire setting, or *impulsive incendiарism* (Meckel, 1820, cited in Lewis & Yarnell [1951]) was classified as pyromania (Marc, 1833). Esquirol was probably the first author to provide a nosological framework for ICDs (Esquirol, 1838). His monomanias describe a number of disorders, including arsonism, alcoholism, impulsive homicide, and kleptomania, which share the common features of a compelling or irresistible urge and a tendency to act impulsively (Tavares, 2008). In the early twentieth century, Kraepelin (1915) reported similarities between excessive buying in women (oniomania) and men’s unrestrained gambling in the context of pathological impulses. Bleuler (1924) refined the concept and included oniomania along with pyromania and kleptomania. Frosch and Wortis (1954) divided disorders of impulse control into two types based upon whether the impulsivity constituted a specific symptomatic disturbance or a pervasive character disorder (Figure 3.1). The former type, defined as *impulse neurosis*, includes

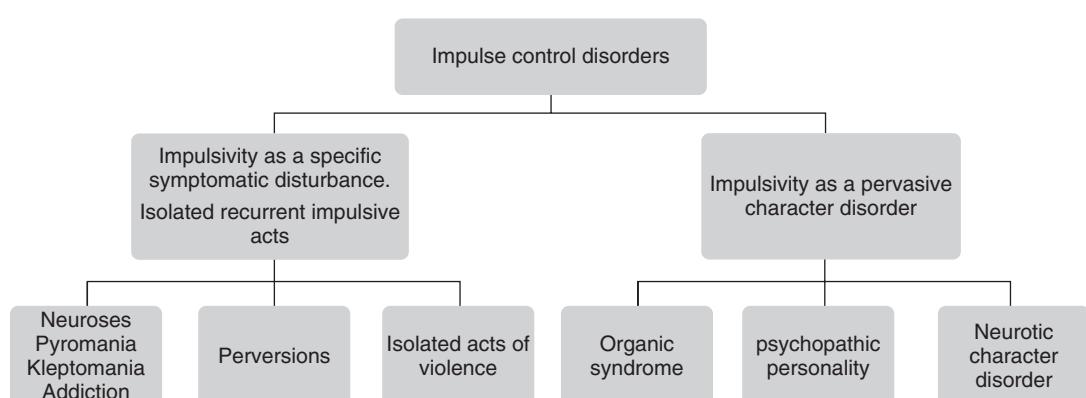


Fig. 3.1 The classification of ICDs according to Frosch and Wortis (1954).

isolated, recurrent impulsive acts (e.g., pyromania, kleptomania, and addictions), perversions (e.g., paraphilia), or catathymic crisis (isolated, nonrepetitive acts of violence). The last type is characterized by diffuse and nonspecific impulsivity pervading the total personality and behavior of the individual. It is divided into three subgroups: organic syndrome, psychopathic personality, and neurotic character disorder. The authors concluded that no single factor determines the development of an impulse disorder. Their classification, which still has relevance for psychiatry, encompasses a wide array of clinical syndromes that have, as their common denominator, a disorder of impulse control.

Current Classification of ICDs

DSM-IV-TR

According to the DSM-IV-TR, ICDs comprise a cluster of disorders linked by an irresistible urge to perform an act that results in negative consequences to the individual or others. This classification is operationally defined by and largely based on symptom similarity rather than pathophysiological mechanisms or treatment response. The category includes pathological gambling, intermittent explosive disorder, kleptomania, pyromania, trichotillomania, and ICDs not otherwise specified (ICD NOS; Table 3.1). The ICDs currently classified as ICD NOS, but under consideration as distinct disorders for the next iteration of the DSM, include compulsive-impulsive sexual behaviors, compulsive-impulsive shopping, compulsive-impulsive Internet usage and compulsive-impulsive skin picking (Dell'Osso et al., 2006). Other psychiatric disorders in which impulsivity may also play a crucial role (e.g., substance-related disorders, bipolar disorder, Tourette's syndrome, binge-eating disorder,

paraphilic, conduct disorders, cluster B personality disorders, attention-deficit hyperactivity disorder, and neurological disorders with disinhibition) are classified elsewhere in the DSM-IV-TR.

ICD-10

The *International Statistical Classification of Diseases and Related Health Problems, 10th Revision* (ICD-10; WHO, 1992) classifies these disorders as “habit and impulse disorders” (Table 3.1). According to the ICD-10, they are characterized by repeated acts that have no clear rational motivation, cannot be controlled, and generally harm the patient’s own interests as well as those of other people. The ICD-10 emphasises the poorly understood nature of ICDs and explains “that they are grouped together because of broad descriptive similarities, not because they are known to share any other important features” (pp. 241–242). However, the conceptualization of an association between disorders of impulse control and disorders of habit regulation is consistent with translational research linking pathophysiological aspects of habit formation, compulsivity, and impulsivity (Brewer & Potenza, 2008). Unlike the DSM-IV-TR, in ICD-10 intermittent explosive disorder is mentioned under “Other habit and impulse disorders” and does not qualify as a distinct category. In addition, impulse and habit disorders involving sexual behavior (F65) are classified elsewhere and are specifically excluded from this group.

Core Features of DSM-IV ICDs

The ICDs are typically characterized by a common core of clinical features:

1. A compelling and irrepressible urge to perform an act that is potentially harmful to self or others

Table 3.1 Classification of ICDs

DSM-IV-TR	ICD-10:DCR-10
Impulse Control Disorders Not Elsewhere Classified	Habit and Impulse Disorders
312.31 Pathological gambling	F63.0 Pathological gambling
312.32 Kleptomania	F63.2 Pathological stealing [kleptomania]
312.33 Pyromania	F63.1 Pathological fire setting [pyromania]
312.39 Trichotillomania	F63.3 Trichotillomania
312.34 Intermittent explosive disorder	F63.8 Other habit and impulse disorders [intermittent explosive disorder]
312.30 Impulse-control disorder not otherwise specified	F63.9 Habit and impulse disorder, unspecified

2. Repeated performance of the problematic behavior
 3. Progressive loss of control over the behavior
- Afflicted individuals may pass through five stages of symptomatic behavior (Figure 3.2).

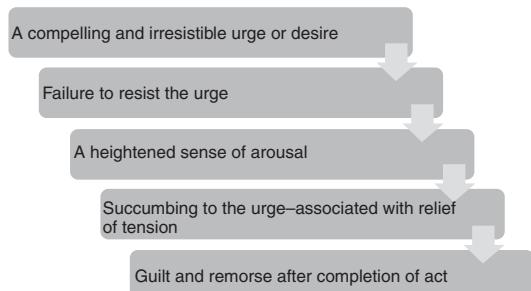


Fig. 3.2 Stages of symptomatic behavior seen in ICDs (Hollander & Stein, 2005).

ICDs and Behavioral Addiction

It has been argued that ICDs share analogous behavior with substance addictions. Like people with substance addictions, those with ICDs, such as pathological gambling, trichotillomania, pyromania, and kleptomania, experience a growing urge beforehand and relief, gratification, and/or pleasure afterward. The drive to undertake a repetitive act that is to some extent rewarding may be termed a *behavioral addiction*, and implicates dysfunction of neural circuitry involved in reward processing and top-down inhibitory control (Grant et al., 2007).

ICDs and Compulsion

Some ICDs, such as pathological gambling, trichotillomania, and the candidate ICD-NOS, are characterized by compulsion as well as impulsivity. Compulsions are repetitive acts that are designed to reduce discomfort or distress. In this sense, individuals with these ICDs may have a compulsive tendency to repeat certain acts in an attempt to decrease dysphoria, such as to reduce the unpleasant feeling of craving in pathological gambling or to alleviate distress in trichotillomania (Hollander & Stein, 2005). According to this model, gambling or hair pulling may also be considered a pathological habit, possibly driven by neural activity within the basal ganglia, with cortical nodes unable to exert sufficient inhibitory influence (Chamberlain et al., 2007a).

ICDs, Impulsivity, and Models of Personality

Impulsivity may be conceptualized as a normal personality trait that contributes to adaptive functioning as well as psychopathology. It is often assumed that people with impulsivity lie on a continuum of severity, from low trait-impulsive individuals, through more impulsive nonclinical groups, to those with diagnosable ICDs. Personality theories, largely based upon self-report and observational studies in humans and animals, have incorporated trait impulsivity into their models. According to Eysenck (Eysenck & Eysenck, 1977), impulsivity (labeled *Broad Impulsiveness*) contributes to several personality domains and may be fractionated into replicable factors, including narrow impulsiveness, risk taking, nonplanning, and liveliness. Factors such as risk taking, nonplanning, and liveliness are positively correlated with extraversion, whereas narrow impulsiveness correlates with neuroticism and psychotism (Eysenck & Eysenck, 1985). Gray's Reinforcement Sensitivity Theory (RST; Gray, 1982, 1987) posits that personality dimensions derive from fundamental punishment and reward sensitivities (Gray, 1981). In the RST model, trait impulsivity is closely related to extraversion and is linked to an overactive behavioral approach system, whereas trait anxiety is closely related to neuroticism and to an under- or overactive behavioral inhibition system (Gray, 1987). Cloninger (1986) postulated a neurobiological basis for phenotypic variation in personality and emphasized the importance of gene-environment interactions. He proposed several genetically independent but functionally interactive personality dimensions, of which novelty seeking, comprising behavioral activation in response to novelty, an extravagant approach to reward, and a tendency to lose one's temper, appear to be conceptually related to Gray's behavioral approach and Eysenck's extraversion models. Individuals scoring highly on the novelty-seeking scale of the Tridimensional Personality Questionnaire (Cloninger, 1987; Cloninger et al., 1991) are described as "impulsive, exploratory, fickle, excitable, quick-tempered, extravagant, and disorderly." Thus, impulsivity may be conceptualized as a biologically driven tendency to respond dramatically to novel stimuli.

Zuckerman (1984) introduced the concept of sensation seeking as a genetically determined trait with biological correlates (e.g., high levels of gonadal

hormones, monoamine oxidase, and exaggerated cortical evoked potentials; Zuckerman & Cloninger, 1996; Zuckerman et al., 1993). The Sensation Seeking Scale Form V (SSS-V; Zuckerman et al., 1978) comprises subscales that correspond to experience seeking, disinhibition, thrill and adventure seeking, and boredom susceptibility. Sensation seeking is associated with a variety of impulsive behaviors such as substance abuse (Stephenson et al., 2002), sexual risk taking (Donohew et al., 2000), reckless driving (Heino et al., 1996), smoking (Zuckerman et al. 1990), and alcohol misuse (Stacy et al., 1993). In contrast, Tellegen (1982, 1985) represented personality as three dimensions, of which *constraint avoidance*, measured on a control-versus-impulsiveness scale, is related to an individual's tendency to engage in risky behavior, inability to express caution or restraint, and inability to accept conventional norms. The Five Factor Model (Costa & McCrae, 1992) provides a conceptual framework for integrating research findings and empirical theories of human personality. Studies using the Five Factor model have decomposed impulsivity into four dimensions that are largely compatible with the models previously described: (1) urgency (a tendency to act rashly in a positive or negative mood state), linked to neuroticism; (2) lack of premeditation (a tendency to act without forethought) and (3) lack of perseverance (inability to remain focused despite distraction), linked to conscientiousness; and (4) sensation seeking (a tendency to seek out novel or thrilling stimulation), linked to extraversion (Whiteside & Lynam, 2001).

In summary, impulsivity is considered an important component of normal personality. It may be fractionated into narrower behavioral constructs, some of which appear to be on the whole adaptive and others pathological. Systematic investigation of impulsive traits in healthy volunteers and mental disorders characterized by impulsive acts, from a neurosciences perspective, may identify dissociable neuropsychological components of normal and pathological impulsivity and may broaden our understanding of the role of impulsivity in human development and ICDs.

The Multifaceted Nature of Impulsivity

As a construct, impulsivity may be approached from various angles within a neurocognitive framework. Early definitions invoking "action without forethought" (Hinslie & Shatzky, 1940) were superseded by a focus on the "quality of the decision,"

such that quick *and incorrect* decisions that led to an undesirable outcome were classified as impulsive (Kagan & Kogan, 1970). Work by Barratt, including studies among the prison population, explored the components of pathological impulsivity. Barratt surmised that impulsiveness was composed of three subtraits: cognitive impulsiveness (making quick cognitive decisions), motor impulsiveness (acting without thinking), and nonplanning impulsiveness (lack of "futuring" or planning; Barratt, 1985). The Barratt Impulsiveness Scale (BIS, version 10) was designed to measure these subtraits (Barratt, 1985). Later studies (Luengo et al., 1991; Patton et al., 1995) refined the cognitive factor, which was relabeled *attentional impulsiveness* and defined as an inability to focus attention or concentrate. The current 30-item BIS-11 (Patton et al., 1995) is arguably the most commonly administered self-report measure for the assessment of impulsiveness in both research and clinical settings. However, this conceptualization of impulsivity focuses exclusively on those aspects that are undesirable. An alternative view, proposed by Dickman (1990), introduces the interesting concept of *functional impulsivity*, related to swift decision making in situations that may lead to personal benefit. In behavioral and cognitive experiments, functional impulsivity is associated with speed of information processing, whereas *dysfunctional impulsivity* is associated with an inability to inhibit competing responses. However, there are indications that, on some tasks, the two tendencies may have moderating effects on each other (Brunas-Wagstaff et al., 1996).

Endeavoring to take into account these complexities, Moeller and colleagues (2001) defined impulsivity as "a predisposition towards rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others" (p. 1784). They proposed that the expression of impulsive behavior involves behavioral (motor) and/or cognitive (choice/decision making/reflection) aspects that may be separable (Bechara & Damasio, 2002; Brunner and Hen, 1997; Evenden, 1999). State- and trait-dependent variables are likely to be involved, and ICDs may differ in the extent to which these separate aspects are expressed (see below) (Moeller et al., 2001).

Diagnostic Validators for ICDs

There has been discussion in the literature as to which diagnostic validators are most important

for the purposes of classification (Hyman, 2007). Ideally, the classification should optimize diagnostic validity (reflecting advances in our understanding of the underpinning neurobiology) as well as clinical utility (to allow appropriate assessment and treatment). Similarity of symptoms, gender ratio, age of onset and course of illness, comorbidity patterns, familial transmission, neurocognitive and imaging markers, and treatment response may be of relevance in identifying common underpinning pathophysiological mechanisms that support clustering disorders together within a diagnostic group. By the same token, recognition of extensive areas of overlap, relative to unique features of specific disorders, together with an ever-expanding increase in the number of diagnostic categories, has raised concern that current systems for classification may lack discriminant validity, and that we are erroneously distinguishing symptoms and disorders that are, in reality, minor variations of broader syndromes (Tyrer, 1989).

In the following sections, we review each ICD in relation to such factors as far as the evidence allows. However, much of the relevant data are patchy or sparse, largely because many of the extensive epidemiological studies of the 1980s and 1990s did not include ICDs (Kaplan, 2005), and in those studies that did, standardized diagnostic criteria were not always applied (Tavares, 2008). A small amount of research has investigated neurocognitive and neuroanatomical changes associated with ICDs. Future extension of such research across the whole range of ICDs, and into the unaffected first-degree relatives of ICD probands, similar to approaches applied in addiction and obsessive-compulsive disorder (OCD) research (reviewed in Fineberg et al., 2010), may contribute to a more accurate definition of these disorders and shed light on their heritable components.

Pathological Gambling

Pathological gambling is defined by persistent and recurrent maladaptive gambling that disrupts personal, family, or vocational pursuits (DSM-IV-TR). Several etiological models have been proposed (Dannon et al., 2006). Some authors suggest that pathological gambling, along with other ICDs, represents a form of “affective spectrum disorder” (McElroy et al., 1992). Others focus on compulsive elements and view pathological gambling as an impulsive subtype of the “obsessive-compulsive spectrum” (Hollander & Wong, 1995). Still others prioritize the role of impulsivity, as a common

denominator linking gambling and substance addiction, and consider it a “behavioral addiction” (Potenza, 2008). Indeed, higher levels of impulsivity, measured by the BIS (Barratt, 1985), have been reported in persons with pathological gambling even compared to cocaine abusers, alcoholics, and polysubstance abusers, as well as depressed patients (DeCaria et al., 1996).

The lifetime and past-year prevalence rates of pathological gambling in adults have been estimated at 1.6% and 1.14%, respectively (Shaffer et al., 1999). These rates are relatively elevated in psychiatric patients (>12%) and adolescents (>6%) (George & Murali, 2005). Increased access to gambling, through casinos, the Internet, and interactive television, may be contributing to an ongoing rise in incidence, at least in the U.S. and U.K. general populations (Christiansen/Cummings Associates, 1996; Griffiths, 2004; Volberg, 1994, 1996). Males are overrepresented by a ratio of 2–3:1 (Shaffer et al., 1999; Sommers, 1988). Black race and divorced/separated/widowed marital status are also associated with an increased risk for pathological gambling (Petry et al., 2005). The peak onset typically occurs in early adolescence in males (Ladouceur et al., 1994) and later in females (Lesieur et al., 1991), who have a quicker progression (Rosenthal, 1992; Volberg, 1994). The disorder tends to run a chronic, progressive course with multiple periods of abstinence and relapse (George & Murali, 2005).

Comorbidity with drug addiction is particularly high. The recent U.S. National Epidemiologic Survey reported that almost three-quarters (73.2%) of pathological gamblers had a concurrent alcohol use disorder, 38.1% had a drug use disorder, and 60.4% had nicotine dependence. Moreover, 60.8% had a personality disorder (Petry et al., 2005). In a nontreatment-seeking population of problem gamblers, borderline personality disorder was the most prevalent personality disorder at 16%, followed by antisocial, paranoid, narcissistic, and personality disorder not otherwise specified (Fernández-Montalvo & Echeburúa, 2004), although other community studies have found a substantial prevalence for antisocial personality disorder (15%; Slutske et al., 2001). In addition, almost half of pathological gamblers were found to have a mood disorder, with one study showing a particularly strong association with mania (Petry et al., 2005). Similarly, a systematic review of studies of mood disorders among patients with pathological gambling identified a relatively high prevalence of manic

(25%) as well as depressive disorder (50%; Kim et al., 2006). Population surveys of pathological gambling also show elevated rates of anxiety disorders (40%–60%), including panic disorder (15%–25%), generalized anxiety disorder (GAD, 5%–15%), posttraumatic stress disorder (15%), and obsessive-compulsive disorder (OCD, 2.5%–20%) (Bland et al., 1993; Cunningham-Williams et al., 1998; Kessler et al., 2008; Linden et al., 1986; Petry et al., 2005; Specker et al., 1996). The rate of suicidal ideation in pathological gamblers has been estimated to range from 20% to 80% and that of suicide attempts from 4% to 40% (George & Murali, 2005).

Problem gambling is associated with elevated rates of other ICDs, with a prevalence ranging from 18% to 43% for one or more comorbid ICDs. Comorbidities with compulsive-impulsive shopping (8%–23%) and compulsive-impulsive sexual behavior (9%–17%), consistent with their inclusion in the ICD group, are noteworthy (Black & Moyer, 1998; Grant & Kim, 2001). Comorbid attention deficit hyperactivity disorder (ADHD) has been reported in 13.4%–20% of cases (Kessler et al., 2008; Specker et al., 1995), suggesting a possible attentional component to pathological gambling.

Kleptomania

Kleptomania, defined as a recurrent failure to resist the impulse to steal items not needed for personal use (DSM-IV-TR), is characterized by an urge to perform an act that is pleasurable at the moment but later causes significant distress and dysfunction (Aboujaoude et al., 2004). Kleptomania can be distinguished from antisocial personality disorder by the presence of guilt or remorse and the lack of a theft motive. The prevalence has been estimated at 6 per 1000 (Goldman, 1991). However, a study of 204 psychiatric inpatients with multiple disorders revealed that 7.8% and 9.3% demonstrated symptoms consistent with a current or lifetime diagnosis of kleptomania, respectively (Grant et al., 2005). Kleptomania is more common in females (2:1 prevalence in clinical samples), and the mean age of onset is in late adolescence or puberty (Aboujaoude et al., 2004; Grant, 2006; Presta et al., 2002). The disorder is usually chronic and the value of the stolen items increases over time, suggesting the development of tolerance, akin to that of substance addiction (Grant & Kim, 2002). Lifetime rates of substance abuse disorders (29%–50%) are elevated in kleptomania (Grant, 2003; McElroy

et al., 1991). In addition, individuals with kleptomania commonly have first-degree relatives with substance abuse, implying a familial association between these disorders (Grant & Potenza, 2004). Studies have also found elevated rates of affective disorder, ranging from 45% to 100% (Bayle et al., 2003; Dannon et al., 2004; McElroy et al., 1991; Presta et al. 2002). Bipolar affective disorder appears to be particularly overrepresented, occurring in up to 27% of cases (Bayle et al., 2003). Elevated lifetime comorbidity was also found for personality disorder (55%), suicidal behavior (36%), anxiety disorders (18%), and other ICDs (36%), in particular those with a compulsive component such as compulsive-impulsive shopping (18%) and trichotillomania (9%; Bayle et al., 2003). However, inconsistent rates of concurrent OCD have been reported, with some studies showing relatively high rates (45%–60%; McElroy et al., 1991; Presta et al., 2002) and others low rates (0%–6.5%; Bayle et al., 2003; Grant 2003). Fifteen percent of individuals with kleptomania met diagnostic criteria for ADHD in their lifetime (Presta et al., 2002).

Intermittent Explosive Disorder

In intermittent explosive disorder (IED), discrete episodes of aggression occur that result in serious assaultive acts or destruction of property. The degree of aggression is out of proportion to any provocation or precipitating stressor and is not better accounted for by other mental disorders or physical factors. The violent outbursts are brief (<1 hour) and are often associated with somatic anxiety (e.g., sweating, chest tightness, palpitations) as well as by a sensation of relief and, in some cases, pleasure, but accompanied by remorse after the act, with no signs of aggression between the episodes (McElroy et al., 1998). Changes in the DSM criteria have affected the interpretation of research findings in IED. For example, the exclusion of comorbid personality disorder in DSM-IV excludes up to 40% of individuals who might previously have met the diagnostic criteria (Coccaro et al., 1998). Moreover, studies have usually reported on impulsive aggression rather than IED per se. The onset of IED is usually abrupt, and it runs a chronic course. Although some studies indicate a male preponderance (Boyd, 2008; Coccaro et al., 1998; McElroy et al., 1998), others have reported a near-equal prevalence between the sexes (Coccaro et al., 2004, 2005). The mean age of onset is in adolescence (16.2 +/– 8.5 years), with an earlier onset in males (12.9 +/– 5.3 years) compared

to females ($19.0 +/ - 9.7$ years; Coccaro et al., 2005; Kessler et al., 2006).

In the recent extensive U.S. National Comorbidity Survey Replication (NCS-R; Kessler et al., 2006), the 12-month and lifetime prevalence estimates of DSM-IV IED were 3.9% and 7.3%, respectively, emphasizing the fact that IED is more common than was previously recognized (Posternak & Zimmerman, 2002). In a study of 1300 psychiatric outpatients, Coccaro et al. (2005) reported similar current and lifetime prevalence estimates for IED of 2.6%–3.6% and 5.6%–7%, respectively. In a survey of probationers, 7.4% were positive for IED and its presence was notably higher among participants who were positive for ADHD (18%) than among those who were ADHD negative (4.7%; Mann et al., 2009).

The majority (81.8%) of NCS-R respondents with lifetime IED (DSM-IV defined) met criteria for at least one other lifetime DSM-IV disorder (Kessler et al., 2006). Comorbidity was raised for anxiety disorders (58.1%), mood disorders (37.4%), substance misuse (35.1%), and ADHD (19.6%). Roughly one-quarter of the respondents had a past history of oppositional defiant disorder (ODD; 24.6%) or of conduct disorder (CD; 24.2%). When ADHD, ODD, and CD were grouped as ICDs, comorbidity with IED reached 44.9%. However, in the clinical sample reported by Coccaro and colleagues (2005), the co-occurrence of ICDs (7.3%) was lower. Another study of 27 IED cases reported 44% with a comorbid ICD (McElroy et al., 1998). The same study reported rates of OCD at around 22%, but the NCS-R (Kessler et al., 2006) and another study (Fontenelle et al., 2005) did not find elevated rates of OCD in individuals with IED. The prevalence of suicide attempts and nonlethal self-injurious behaviors is estimated to be 12.5% and 7.4%, respectively (McCloskey et al., 2008).

Pyromania

Pyromania is defined by multiple episodes of deliberate and purposeful fire setting without external reward (DSM-IV-TR). Pyromaniacs experience tension or affective arousal before setting the fire; pleasure, gratification, or relief when setting fires or when witnessing or participating in their aftermath; and fascination with or attraction to fire and the activities and equipment associated with firefighting. Pyromania differs from arson in that the latter is perpetrated for financial gain, revenge, or another advantage and is planned beforehand (Dell'Osso et al., 2006).

Pyromania is understudied in the adult community. Most research focuses on fire setting in children and adolescents. The prevalence of fire setting among child psychiatric outpatients is estimated at 2.4%–3.5% (Jacobson, 1985; Kolko & Kadzin, 1988). Grant and Kim (2007) reported the mean age of onset as 18 ± 5 years in a small study of adults and adolescents, with a male:female ratio of approximately 8:1. Fire setting is typically episodic, though the long-term course of pyromania is poorly understood.

Comorbidity with other ICDs is high. For instance, in a recent small study of individuals with pyromania ($n = 21$; Grant, & Kim, 2007), 47.6% met criteria for a current ICD, with lifetime comorbidities of 23.8% for kleptomania, 9.5% each for pathological gambling and IED, and 4.8% for trichotillomania. The same study reported significant comorbidity with current substance use (33.3%) and mood disorder (61.9%). Anxiety disorders were only modestly overrepresented (19%). No subject met criteria for ADHD or antisocial personality disorder, although 9.5% met criteria for borderline personality disorder.

Trichotillomania

In trichotillomania, recurrent hair pulling resulting in noticeable hair loss is preceded by a buildup of tension and is followed by pleasure, gratification, or relief. Hair pulling may be either *automatic* or *focused*. Automatic pulling occurs without awareness, while focused pulling exhibits a compulsive quality that may incorporate a conscious response to a negative emotional state (e.g., anxiety, stress, anger), an intense thought or urge, or an attempt to establish symmetry (Flessner et al., 2009). Trichotillomania has also been conceptualized as an obsessive-compulsive spectrum disorder (Swedo & Leonard, 1992), a pathological form of grooming behavior (Grant & Christenson, 2007), or a body-focused habit disorder (Stein et al., 2007).

A survey of 2500 college students reported a lifetime prevalence of trichotillomania of 0.6% when all diagnostic criteria were met. However, “clinically significant” hair pulling occurred in 1.5% of males and 3.4% of females (Christenson et al., 1991a). In adult recruitment studies, females typically outnumber males by at least 3:1 (Chamberlain et al., 2007b). Trichotillomania typically begins in childhood or adolescence. The mean age of onset is 12–13 years (Christenson, 1995), and the disorder usually runs a chronic course with waxing and waning symptoms (Walsh & McDougle, 2001).

Compared to females, males with grooming disorders including trichotillomania experience a later onset and greater functional impairment, and are more likely to suffer from a comorbid anxiety disorder (Grant & Christenson, 2007). In a study of 60 trichotillomania subjects, 82% qualified for past or current Axis I diagnoses other than trichotillomania (Christenson et al., 1991b). Subjects reported an elevated lifetime prevalence of anxiety disorder (57%), major depressive disorder (55%), substance misuse (22%), and OCD (15%). An increased prevalence of OCD has also been reported in first-degree relatives of individuals with trichotillomania compared to normal controls (Lenane et al., 1992). Hanna et al. (2005) found that grooming disorders (trichotillomania, skin picking, nail biting) indexed familial vulnerability to OCD. Conversely, Bienvenu et al. (2000) reported increased rates of grooming disorders in probands with OCD compared with controls, as well as in first-degree relatives of OCD probands compared with controls. Bienvenu et al. (unpublished results) recently replicated these results using a larger sample of families; the prevalence of trichotillomania and skin picking was higher in relatives of OCD-affected probands than in control relatives, independent of trichotillomania or skin picking in probands and OCD in relatives, supporting a familial association between these disorders. Lifetime rates of compulsive skin picking occurred in approximately 10% patients with trichotillomania (Arnold et al., 1998; Simeon et al., 1997). Lifetime rates of trichotillomania may be elevated in some ICDs such as compulsive-impulsive sexual behavior (6%; Black et al., 1997a) and kleptomania (10%; McElroy et al., 1991). Although systematic comparisons with control groups are needed, relatively little comorbidity between trichotillomania and other ICDs, such as pathological gambling (Grant & Kim, 2003, 2007; McElroy et al., 1998) or substance use disorders (Lochner et al., 2005), has so far been demonstrated. Swedo and Leonard (1992) found Axis II disorders in 38% of their sample, the most prevalent being histrionic (26%), borderline (18%), and passive/aggressive personality disorder (16%).

Comparing Key Clinical Validators Across ICDs

Table 3.2 summarizes the clinical and demographic characteristics of the major ICDs. According to current data, most ICDs develop in late childhood or adolescence and run a chronic relapsing course, compatible with the theory that trait impulsivity,

developing early in life, plays an important role in their ontogenesis. Some ICDs are more common in males, such as pyromania, IED, and pathological gambling, whereas others, such as kleptomania and trichotillomania, have a higher prevalence in females. A plausible explanation is that gender may play a part in shaping the direction of the expressed behaviour. The ICDs are characterized by arousal at around the time the impulsive act is performed, though some, such as trichotillomania, pathological gambling, IED, and kleptomania, are more clearly linked to subsequent feelings of shame and remorse than others, such as pyromania. All (or almost all) disorders show some evidence of increased comorbidity with DSM-IV Axis I disorders of impulse control, mood, substance addiction, personality, anxiety, and attention, although most of the studies lacked a clinical control group for comparison. Moreover, we should be cautious in interpreting comorbidity per se as evidence of shared psychobiology, since the elevated rates may, in some cases, simply represent the influence of one disorder on the development of another (e.g., the consequences of gambling may result in depression and anxiety). Alternatively, comorbidity may simply reflect an artifact of overlapping diagnostic criteria, such as may occur between ICDs and borderline personality disorder. Evidence of cosegregation of a comorbid disorder in relatives unaffected by the index disorder, such as trichotillomania (and compulsive skin picking) in relatives of people with OCD, may represent more robust evidence of a shared etiology. Notwithstanding the limitations of the data, subtle differences among the comorbidity patterns of the individual ICDs are becoming apparent. For example, relative to the other ICDs, pathological gambling and kleptomania appear to be strongly associated with substance addiction and bipolar affective disorder, whereas trichotillomania appears to be strongly linked with OCD and compulsive-impulsive skin picking and less strongly associated with other ICDs, such as pathological gambling, bipolar affective disorder, or substance addiction. These differences imply diverging as well as overlapping psychobiology for the ICDs as currently defined and provide limited support for their classification as separate diagnoses within a common grouping.

Impulse Control Disorders Not Otherwise Specified (ICD NOS)

Examination of the ICD NOS group (Table 3.3) also reveals interesting overlap and variation, although the data remains scarce and preliminary.

Table 3.2 Clinical and Demographic Characteristics of the Major DSM-IV ICDs (data derived from studies reported in the text)

ICDs Validators	Pyromania	IED	PG	Kleptomania	Trichotillomania
Demographics					
Sex ratio (♂:♀)	8:1	Inconsistent data	2:1 to 3:1	1:2	1:3
Age of onset	Late adolescence	Early to mid-adolescence; males earlier than females	Early adolescence in males; later in females but quicker progression	Late adolescence	Late childhood or early adolescence
Course	Chronic, fluctuating	Chronic, fluctuating	Chronic, fluctuating	Chronic, fluctuating	Chronic, fluctuating
Symptoms					
Arousal before act	Yes	Yes	Yes	Yes	Yes
Pleasure during act	Yes	Yes	Yes	Yes	Yes
Remorse/distress after act	Yes but also pleasure or satisfaction	Yes	Yes	Yes	Yes
Comorbidity with					
ICDs	47.6% PG: 9.5% Klep: 23.8% IED: 9.5% Trich: 4.8% C-I S: 19% C-I SB: 4.8% C-I SP: 9.5%	7%–44%	18%–43% IED: 2%–13% C-I S: 8%–23% C-I SB: 9%–17%	36% Pyromania: 15% Trich: 9% C-I S: 18%	? Klep: 4.1% IED: 6.1% Pyromania: 2% C-I S: 4.1% C-I SP: 10%
Any mood disorder	61.9%	37.4%–75.6%	50%–60%	≥50%	65%

Major depression	47.6%	37.3%	≥50%	45%	55%
Bipolar affective disorder (BPAD)	BPAD I: 4.8% BPAD II: 9.5%	?	25%	27%	0%
Any anxiety disorder	19%	58.1%–78%	40%–60%	18%	57%
OCD	4.8%	0%–22% Results inconsistent	2.5%–20% Results inconsistent	0%–60% Results inconsistent	15%
Any substance misuse	33.3%	35.1%–59.8%	50%	29%–50%	22%
Personality disorder	9.5%	32.7%	60%	55%	38%
ADHD	0	19.6%	13.4%–20%	15%	?
Self-harm	?	Suicide attempt: 12.5% Self-injury: 7.4%	4%–40%	36%	?

? = not known; ADHD = attention deficit hyperactivity disorder; BPAD = bipolar affective disorder; C-I S = compulsive-impulsive shopping; C-I SB = compulsive-impulsive sexual behavior; C-I SP = compulsive-impulsive skin picking; ICDs = impulse control disorders; IED = intermittent explosive disorder; Klep = kleptomania; OCD = obsessive-compulsive disorder; PG = pathological gambling; Trich = trichotillomania.

Table 3.3 Clinical and Demographic Characteristics of the DSM-IV ICDs Not Otherwise Specified (data derived from studies reported in the text)

ICD Validators	Compulsive-Impulsive Skin Picking	Compulsive-Impulsive Sexual Behaviors	Compulsive-Impulsive Shopping	Compulsive-Impulsive Internet Usage
Demographics				
Sex ratio (♂:♀)	F>M	M > F	F > M	M > F
Age of onset	Childhood and early adolescence	?	Late adolescence, early adulthood	Any age
Course	Chronic	?	Chronic	?
Symptoms				
Arousal before act	Yes	Yes	Yes	Yes
Pleasure during act	Yes	Yes	Yes	Yes
Remorse/distress after act	Yes	Not always	Yes	No
Comorbidity with				
ICDs	++ Mainly trichotillomania	?	+	++ CI-S, C-ISB, PG, and pyromania
Any mood disorder	+	+	++	+
Major depression	?	+	++	?
Bipolar affective disorder (BPAD)	?	?	?	+
Any anxiety disorder	+	+	++	?
OCD	+	+	+	+
Any substance misuse	+	+	++	++
Personality disorder	+ (OCPD)	?	?	?
ADHD	?	+	?	++
Self-harm	++	?	?	?

+= present or some evidence; ++= strongly present or much evidence; ?= not known; CI-S = compulsive-impulsive shopping; C-I SB = compulsive-impulsive sexual behavior; OCPD = obsessive-compulsive personality disorder; PG = pathological gambling.

Compulsive-Impulsive Internet Usage

Also known as *Internet addiction*, C-I Internet usage represents excessive, uncontrollable, and harmful use of the Internet (Dell'Osso et al., 2006). Descriptions are based on modifications of the DSM-IV (American Psychiatric Association, 1995) criteria for pathological gambling (Beard & Wolf, 2001; Block, 2007; Young, 1998a) and include (1) excessive use, with loss of the sense of time or neglect of basic drives; (2) withdrawal symptoms,

such as anger, tension, and/or depression, when the computer is inaccessible; (3) tolerance, including the need for better computer equipment, more software, or more hours of use; and (4) negative repercussions, including arguments, lying, poor achievement, social isolation, and fatigue. Some authors believe that Internet addiction is better conceptualized as a pattern of dependent behaviors contingent upon "virtual" reinforcers that are similar to real-world addictions, such as money or sex, rather

than as a separate psychiatric disorder (Yellowlees & Marks, 2007). Subtypes have been proposed, such as excessive gaming, sexual preoccupations, and e-mail/text messaging (Block, 2007). The lack of a standardized definition or assessment tools has hampered evaluation of the true extent of this problem, which is estimated to affect 3% to as many as 11% of Internet users (DeAngelis, 2000; Kershaw, 2005). Individuals are unforthcoming about problematic Internet use, as they may be embarrassed or lack awareness of the disorder (Niemz et al., 2005; Yip & Potenza, 2009). With increased availability, problematic Internet use has been reported across most age, gender, social, educational, and socioeconomic groups (Chou et al., 2005; Young, 1998b). Males may use the Internet differently from females and appear more likely to be subject to Internet addiction and to explore sexual fantasies online (Chou et al., 2005).

Psychiatric comorbidity is common (Shaw & Black, 2008). Black and colleagues (1999) found that 38% of a case series of compulsive Internet users had at least one ICD, including compulsive buying (19%), pathological gambling, pyromania, and compulsive-impulsive sexual behavior (10% for each disorder). Other studies reported a high prevalence (50%–60%) of comorbid substance misuse (Bai et al., 2001; Shapira et al., 2000; Young, 1996), OCD (10%–20%; Black et al., 1999; Shapira et al., 2000, 2003), and ADHD (18%; Ko et al., 2008). One small study also found a high prevalence of comorbid bipolar affective disorder (Shapira et al., 2000). In adolescents with Internet addiction, depression is the most frequently reported psychiatric symptom (van den Eijnden et al., 2008; Whang et al., 2003; Yen et al., 2007). However, a 2-year follow-up study of 2293 adolescents found that hostility and ADHD were the most significant predictors of Internet addiction (Ko et al., 2009).

Compulsive-Impulsive Shopping

This disorder is characterized by the recurrent and irresistible impulse to buy items that are either not needed or cannot be afforded (Dell'Osso et al., 2006). A prepurchase urge, which culminates in a purchase and a sense of fulfillment, is followed by guilt and remorse (Black, 2007). Koran and colleagues (2006) surveyed 2513 U.S. adults by telephone and estimated a 5.8% prevalence. The majority of these people (80%–95%) are female (Black, 2001; Faber & O'Guinn, 1989, 1992; Koran et al., 2006). An onset in the late teens or early 20s is usually reported, and the disorder is

generally chronic (Christenson et al., 1994; McElroy et al., 1994; Schlosser & Black, 1994). Some evidence suggests that compulsive-impulsive shopping runs in families and that within these families mood, anxiety, and substance use disorders are also overrepresented (Black et al., 1998; McElroy et al., 1994). Black et al. (1998) reported that compulsive buyers ($n = 33$) were more likely than comparison subjects ($n = 22$) to have any Axis I disorder (78.8% vs. 54.6%) or more than one other psychiatric disorder (48.5% vs. 13.6%). Several studies of compulsive-impulsive shopping have shown high rates of comorbid mood disorders (21%–100%), anxiety disorders (41%–80%), substance use disorders (46%), eating disorders (8%–35%), and OCD (3%–30%) (Black, 2001; Black et al., 1997b, 1998; Christenson et al., 1994; Koran et al., 2002; McElroy et al., 1994; Schlosser & Black, 1994). In contrast, studies of patients with OCD have not generally found elevated rates of compulsive buying (2.2%–10.6%; du Toit et al., 2005; Fontenelle, 2005), except for a study by Lejoyeux and colleagues (23.3%; 2005). Disorders of impulse control are also relatively common in individuals with compulsive-impulsive shopping (21%–40%; Christenson et al., 1994; McElroy et al., 1994).

Compulsive-Impulsive Sexual Behaviors

Compulsive-impulsive sexual behaviors include repetitive sexual thoughts and acts that may or may not cause subjective distress but interfere with several aspects of the patient's life, causing social or occupational impairment or legal and financial consequences (Black et al., 1997a; Dell'Osso et al., 2006). Compulsive-impulsive sexual behaviors may involve a broad range of paraphilic (sexual arousal to unconventional stimuli such as exhibitionism, voyeurism etc.) or nonparaphilic symptoms (excessive conventional sexual behaviors; Coleman, 1991). Given the secretiveness associated with the condition and the heterogeneity of the disorder, the true prevalence remains uncertain, with estimates ranging from 5% to 6% of the U.S. population (Coleman, 1991; Schaffer & Zimmerman, 1990). There is a male preponderance (Weissberg & Levay, 1986), but this could represent ascertainment bias (Dell'Osso et al., 2006). In a study of 120 consecutively evaluated male outpatients with paraphilic and related disorders (including 60 sex offenders), the most prevalent Axis I disorders were mood disorders (71.6%), especially dysthymic disorder (55%); anxiety disorders (38.3%), especially social phobia (21.6%); and substance abuse (40.8%). Attention deficit hyperactivity

disorder was retrospectively diagnosed in 35.8% (Kafka & Hennen, 2002). Other studies have reported rates of 12% and 14% comorbid OCD (Black et al., 1997a; Kafka, & Prentky, 1994).

Compulsive-Impulsive Skin Picking

Compulsive impulsive skin picking is defined by a repetitive tendency to pick skin that results in tissue damage leading to significant distress, dysfunction, or disfigurement. Most patients experience increasing tension before the act and/or relief after the act (Grant & Odlaug, 2009; Simeon & Favazza, 1995). Based on phenomenological and clinical similarities between trichotillomania and compulsive-impulsive skin picking, it has been suggested that the two disorders can be grouped within a spectrum of body-focused repetitive behavior (Bohne et al., 2002). In a nonclinical, community sample ($n = 354$), 62.7% endorsed some form of skin picking and 5.4% reported clinical levels of skin picking and associated distress/impact (Hayes et al., 2009). Grant and colleagues (2007) reported a prevalence of 11.8% in an adolescent psychiatric inpatient sample, while other studies have shown that approximately 2% of dermatology patients (Griesemer, 1978) and 4% of college students (Keuthen et al., 2000) suffer from the disorder. In a treatment-seeking population ($n = 40$), 47.5% reported the onset of skin picking before 10 years of age; these individuals were less likely to seek treatment than those with later onset (Odlaug & Grant, 2007). Skin picking may persist with a waxing and waning lifetime course, with one study citing an average duration of 21 years (Wilhelm et al., 1999). Women are affected more than men and the face is the most common site of excoriation, though any part of the body can be affected (Arnold et al., 1998; Simeon, 1997). In studies of patients with trichotillomania, lifetime rates of skin picking were approximately 10% (Arnold et al., 1998; Simeon, 1997). Increased rates of grooming disorders (trichotillomania, skin picking, nail biting) were reported in probands with OCD compared with controls, as well as in first-degree relatives of OCD probands compared with controls and independent of trichotillomania or skin picking in probands and OCD in relatives, supporting a familial association between these disorders (Bienvenu et al., 2000; unpublished results).

Impulse Control Disorders Not Otherwise Specified (ICD NOS) as Stand-Alone Disorders

The expansion of candidate ICD categories to include compulsive-impulsive Internet usage,

together with the lack of strict concordance on diagnostic criteria for the ICD NOS grouping and high rates of comorbidity among ICDs, as well as with other mental disorders typically associated with ICDs, such as depression, substance abuse, and anxiety, inevitably raises concerns about the discriminant validity of these categories and questions about how well some of these syndromes actually represent distinct diagnostic entities. Studies validating their diagnostic reliability, for example, using tests of interrater reliability, may improve confidence in this area. Despite the limitations of the data, compulsive-impulsive skin picking does appear to stand apart from the other ICD NOSs, as it shares a similar phenomenology, course, and specific familial comorbidity with trichotillomania, raising the possibility that skin picking may represent a forme-fruste of this disorder. Thus, compulsive-impulsive skin picking and trichotillomania could possibly be classified together as body-focused habit disorders. On the other hand, the phenotype in psychiatry is acknowledged to be an unreliable marker of the underpinning neurobiology and may confound our perception of the interrelatedness of individual disorders. Future research into the endophenotypes (intermediate phenotypes) associated with ICDs, such as the associated neuropsychological processes, neural circuitry, and its neurochemical modulation, is likely to be more helpful in defining the proximity of their relationships.

Neurocognitive Models of Impulsivity

Translational models of impulsivity have identified separate neurocognitive dimensions that may serve as a useful heuristic for exploring the pathophysiology of ICDs. *Motor impulsivity*, representing a tendency to prepotent motor disinhibition; *reward impulsivity*, representing difficulty in delaying gratification and choosing immediate small rewards despite negative long-term consequences; and *reflection impulsivity*, representing insufficient information sampling before making a choice, are each thought to be mediated by specific and dissociable failures in cortical “top-down” regulation of motor behavior involving dysregulation of frontostriatal neurocircuitry (Chamberlain & Sahakian, 2007; Fineberg et al., 2010)

Motor Impulsivity

Behavioral theory posits motor impulsivity as the inability to suppress prepotent responses (Barkley, 1997; Chamberlain & Sahakian, 2007) resulting from deficits in the underlying mechanisms of

behavioral inhibition (Gray, 1995; Rachlin, 2000). A prepotent response is one for which immediate reinforcement is available or has been previously associated with that response, such as responses that serve basic desires such as for food, sex, or escape from punishment (Barkley, 1997). Thus, motor impulsivity involves a failure to inhibit goal-driven behavior and encompasses a wide range of actions that are inappropriate for the context, premature, poorly planned, and result in adverse consequences (Daruna & Barnes, 1993; Evenden, 1999). Two of the most common measures of motor impulsivity are the go/no-go task and the stop-signal reaction time task (SSRT; Band & Van Boxtel, 1999). They have been used to study behavioral inhibition in both animals (Eagle & Robbins, 2003; Mishkin & Pribram, 1955) and humans (Costantini & Hoving, 1973; White, 1981). The SSRT depends upon the integrity of neural networks, including the pre-supplementary motor area, right inferior frontal gyrus, anterior cingulate cortices, and putamen (Aron, et al., 2003) and is modulated in rats and humans by norepinephrine (Chamberlain et al., 2007c; Cottrell et al., 2008), but not serotonin (Clark et al., 2005). Specific and substantial deficits in SSRT performance have been observed in several major mental disorders, including trichotillomania, OCD, and ADHD (Chamberlain et al., 2007b). The finding of SSRT impairment with intact cognitive flexibility in patients with compulsive-impulsive skin picking (Odlaug et al., 2010), draws interesting parallels with findings in trichotillomania and may indicate shared aspects of neurobiological dysfunction across these two disorders, while to some extent differentiating OCD, in which disorder cognitive inflexibility is also evident on attentional set-shifting tasks. Behavioral impairment on this test has also been identified in people with substance addiction (Monterosso et al., 2005), indicating potential neural overlap between substance and hair-pulling addictions. However, in a recent study, problem gamblers did not differ significantly from controls on the SSRT despite elevations in trait-impulsivity ratings (Lawrence et al., 2009), suggesting pathological gambling and trichotillomania may diverge in this aspect of impulsive responding.

Reward Impulsivity

In general, humans and animals, when offered the choice between a smaller, sooner reward and a larger, later reward, prefer the larger reward if the delay is sufficiently short. However, preference shifts to the smaller, immediate reward as the delay to the

larger reward increases, since delayed outcomes, despite their higher value, are perceived as less certain (Ainslie & Herrnstein, 1981; Kahneman & Tversky, 1979; Keren & Roelofsma, 1995). Thus, the appeal of a larger reward is discounted with increasing delay, a phenomenon termed *delay discounting* (Kirby & Santiesteban, 2003; Laibson, 1997). These judgments are thought to be mediated through orbitofrontal and related cortical circuitry under probable serotonergic modulation (Rogers et al., 1999) and subcortical circuitry under joint dopaminergic and serotonergic control (Winstanley et al., 2006). Reward impulsivity may be construed as the selection of small, immediate rewards over larger, later ones (Ho et al., 1998). This can be measured by decision-making or gambling tasks such as the Cambridge Gambling Task (Rogers et al., 1999) and the Iowa gamble task (Bechara et al., 1997) that compare the quality of decision making when individuals are asked to choose between small, likely rewards and large, unlikely rewards. Individuals with pathological gambling (Dixon et al., 2003) as well as substance misuse (Heila et al., 2006), alcohol misuse (Bobova et al., 2009), ADHD (Barkley et al., 2001) and smoking (Baker et al., 2003) show increased discounting of delayed rewards and perform disadvantageously on gambling tasks. Furthermore, in individuals with pathological gambling and substance misuse, neurocognitive performance correlates with clinical measures (Bechara, 2003; Petry, 2001), suggesting that reward impulsivity is a key and overlapping domain for both groups. In contrast, patients with trichotillomania were unimpaired on the Cambridge Gamble task (Chamberlain et al., 2007a).

Reflection Impulsivity

A tendency to consider alternative solutions in the presence of more than one option is described as *reflectivity*, a cognitive style that impacts favorably on the quality of problem solving (Kagan, 1966). Reflection impulsivity, representing insufficient information sampling before making a choice, represents another cognitive component of impulsivity and may be measured by tasks such as the Reflection Task (Clark et al., 2006), which is sensitive to substance abuse. In a study by Chamberlain et al. (2007a), patients with trichotillomania did not differ significantly from controls on the reflection impulsivity task (Clark et al., 2006). In contrast, a study that investigated reflection impulsivity in pathological gamblers, using the Matching Familiar Figures Test (Block & Harrington 1974),

demonstrated that pathological gamblers had significantly higher error rates than healthy controls.

Other factors such as faulty temporal judgments, resulting from increased allocation of attentional resources to the passage of time and/or faster cognitive processing secondary to heightened states of arousal, may also contribute to disadvantageous decision making (Barratt, 1983; Bschor et al., 2004; Wittmann & Paulus, 2008). Indeed, an altered sense of time may account for some of the impatience exhibited by impulsive individuals (Glicksohn et al., 2006; Takahashi, 2005). Studies on subjects with increased impulsiveness, such as those with borderline personality disorder (impulsive type; Berlin & Rolls, 2004), orbitofrontal cortex lesions (Berlin et al., 2004), sleep deprivation (Reynolds & Schiffbauer, 2004), and substance dependence (Wittmann et al., 2007), and children with disruptive behavior disorders (Dougherty et al., 2003) have shown that impulsive subjects overestimate timed intervals. Similarly, individuals with pathological gambling who scored higher on self-reported measures of impulsivity such as the BIS and showed deficits measured by a frontal behavior questionnaire considered to reflect prefrontal-cortical dysfunction had a faster subjective sense of time compared with controls (Berlin et al., 2008).

Summary

Impulse control disorders are poorly understood, prevalent, disabling disorders that are defined by the presence of impulsivity as a core feature. They tend to emerge early in life and run a chronic, though sometimes fluctuating, lifelong course. Comorbidity patterns provide some support for a nosological relationship between the ICDs as currently grouped. However, there are additional associations, for example with addictive, compulsive, and bipolar affective disorders, classified elsewhere in the DSM-IV, that may also have etiological relevance. Converging comorbidity and neurocognitive data suggest that some ICDs, such as pathological gambling, may be closely related to substance addiction, whereas evidence from controlled family studies suggests that trichotillomania is closely related to OCD. Impulse control disorders NOS remain scarcely researched, and though limited data hint at links between compulsive-impulsive shopping, sexual behavior, Internet usage, and the ICD group as a whole, compulsive-impulsive skin picking appears to be more closely associated with trichotillomania. Alternatively, these two disorders may be conceptualized as body-focused habit disorders.

To restrain the ongoing expansion of new diagnostic categories, a more parsimonious model for ICDs may be desirable. For example, ICDs could be defined simply, according to the presence of impulsivity as the core symptom, with associated behaviors (gambling, shopping, hair pulling, etc.) designated as diagnostic specifiers. On the other hand, neuropsychological evidence suggests that impulsivity is multidimensional and comprises dissociable cognitive and behavioral indices governed by separate neural mechanisms, the components of which vary from ICD to ICD. For example, trichotillomania may principally involve motor impulsivity, whereas pathological gambling appears to be a disorder of reward and reflection impulsivity. In addition to impulsivity, neuropsychological domains such as compulsivity, affect regulation, and habit formation may contribute differentially to the expression of certain ICDs. Future research exploring these components, using neurocognitive and imaging probes, should improve our classification of the ICDs and our understanding of how they relate to other psychiatric disorders.

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Expanding the Formal Category of Impulse Control Disorders

Jon E. Grant and Brian L. Odlaug

Abstract

In addition to the formal impulse control disorders (ICDs), high rates of trait impulsiveness, sensation seeking, and dissociative qualities have been noted in several other disorders that share similar phenomenological, clinical, and possibly biological characteristics. These proposed disorders include pathological skin picking, compulsive buying, problematic Internet use, and compulsive sexual behavior. Elevated rates of co-occurring ICDs within both the formal and proposed ICDs suggest that at least some of the proposed ICDs may share a common pathophysiology with some of the formal ICDs. Although research has only recently begun to explore pharmacological and psychosocial treatments for the ICDs, studies have shown a similar response between the formal and the proposed ICDs. Future development in understanding the relationship between these disorders will help to refine views of the psychopathology and taxonomy of these conditions.

Keywords: compulsive buying, comorbidity, etiology, kleptomania, impulse control disorder, intermittent explosive disorder, Internet addiction, pathological gambling, personality, sex addiction, skin picking, trichotillomania, treatment

Introduction

In the fourth edition of the *Diagnostic and Statistical Manual of the American Psychiatric Association* (DSM-IV-TR), the category of Impulse Control Disorders Not Elsewhere Classified currently includes pathological gambling, kleptomania, trichotillomania, pyromania, intermittent explosive disorder, and impulse control disorders (ICDs) not otherwise specified (American Psychiatric Association, 2000) (hereinafter referred to as the *formal ICDs*). These ICDs have been grouped together based on similarities in clinical presentation and hypothesized similarities in pathophysiology.

Impulsivity has been defined as a predisposition toward rapid, unplanned reactions to either internal or external stimuli without regard for negative consequences (Moeller et al., 2001). Given this definition

of impulsivity, multiple psychiatric disorders might be characterized as exhibiting problems with impulse control. Without clear evidence of a common pathophysiology linking these disorders, the question of which disorders should be included among the ICDs may depend in part on what one considers the core clinical and pathophysiological features of these disorders and whether these aspects are similar across disorders. Specific disorders have been proposed for inclusion based on perceived phenomenological, clinical, and possibly biological similarities: pathological skin picking, compulsive buying, problematic Internet use, and compulsive sexual behavior (hereinafter referred to as the *proposed ICDs*). The extent to which any or all of these ICDs share clinical, genetic, phenomenological, and biological features remains debated.

Heterogeneity of ICDs

Although people with ICDs have various features in common, our understanding of these disorders may need to address the heterogeneity across and within disorders. A cluster analysis has demonstrated that kleptomania, compulsive buying, and intermittent explosive disorder may belong to an *impulsive* subgroup, while trichotillomania, pathological gambling, and compulsive sexual behavior may belong to a *reward deficiency* group (Lochner & Stein, 2006). Even this type of analysis, however, may be too simple.

On an individual level, perhaps some people with pathological gambling have more in common neurobiologically with some people with compulsive sexual behavior than they do with other gamblers. Neurobiology may cut across disorders. A particular impulse behavior, such as gambling, may be a manifestation of several pathophysiologies. Family history, comorbidity, genetics, neuroimaging, and treatment response may all be means by which this complexity can be better understood.

Expanding the Formal Categories

Clinical Characteristics

One criterion that should be relevant to determining which disorders to include among ICDs is whether they share clinical characteristics. For many, but not all, individuals with both the formal and the proposed ICDs, the behavior is clinically characterized by certain core elements: (1) an urge to engage in a behavior with negative consequences; (2) mounting tension unless the behavior is completed; (3) rapid but temporary reduction of the urge after completion of the behavior; (4) return of the urge over hours, days, or weeks; (5) external cues unique to the behavior; (6) secondary conditioning by external and internal cues (dysphoria, boredom); and (7) hedonic feelings early in the behavior (Marks, 1990).

Most phenomenological data support a relationship between the proposed ICDs (compulsive buying, problematic Internet use, compulsive sexual behavior, and pathological skin picking) and the formal ICDs. Higher rates of the proposed ICDs have been observed in adolescents and young adults and lower rates among older adults, which suggests a natural history similar to that observed in formal ICDs (Chambers & Potenza, 2003; Grant, Williams, & Potenza, 2007). The natural histories of the proposed ICDs and the formal ICDs suggest that the disorders are chronic, relapsing conditions (Flessner & Woods, 2006; Grant, Odlaug, & Potenza, 2009;

Potenza et al., 2001) but that some people recover on their own (Hodgins & El-Guebaly, 2000; Nathan, 2003; Slutske, 2006; Slutske et al., 2003).

Like the formal ICDs, the proposed behaviors generally have a pleasurable quality (although for some people with pathological skin picking, the behavior is automatic, with little if any emotional quality (Christenson & Mansueto, 1999; Grant, Odlaug, & Potenza, 2007; Keuthen et al., 2010). Many people with the proposed ICDs report an urge or a craving state prior to initiating the behavior, as do individuals with the formal ICDs, such as the urges prior to gambling or stealing (Grant & Kim, 2002; Kim et al., 2001). Additionally, these behaviors often decrease anxiety, as the formal ICDs do, for example, in gambling or trichotillomania (Grant & Odlaug, 2009; Grant, Odlaug, & Kim, 2007). Emotional dysregulation may therefore play a large role in cravings in both the proposed and the formal ICDs. Although not examined for all behavioral addictions, many people with pathological gambling, kleptomania, compulsive sexual behavior, problematic Internet use, and compulsive buying report symptoms consistent with tolerance and withdrawal (Dell'Osso et al., 2008a; Goodman, 1993; Hollander & Allen, 2006; Kim et al., 2001; Rosenthal & Lesieur, 1992). As in the formal ICDs, financial and relationship problems are common in the proposed ICDs.

Personality

Individuals with the proposed ICDs (e.g., compulsive sexual behavior, problematic Internet use, and compulsive buying) and those with the formal ICDs (e.g., kleptomania, pathological gambling) appear to score high on self-report measures of impulsivity and sensation seeking (Kelly et al., 2006; Ko et al., 2010; Lejoyeux et al., 1997; Raymond et al., 2003). Trait impulsiveness may promote ICDs, and research has shown that pathological gamblers, individuals with compulsive sexual behaviors, and kleptomaniacs score high on measures of impulsiveness (Castanelli & Rugle, 1995; Grant & Kim, 2002; Miner et al., 2009; Steel & Blaszczynski, 1998).

Impulsive individuals may be highly responsive to positive reinforcement but rather insensitive to punishment (Goudriaan et al., 2006). They may have to struggle to imagine negative outcomes. Impulsive individuals may also lack the capacity to divide attention among competing stimuli and thus may be insensitive to internally generated cognitions focusing on restraint (Abrams & Kushner, 2004). As such, the initiation of an impulsive behavior may

quickly lead to a loss of control. Individuals high in sensation seeking search for novel, exciting experiences that may entail an element of physical or social risk. High levels of sensation seeking have been associated with pathological gambling (Coventry & Brown, 1993), kleptomania (Grant & Kim, 2002), compulsive sexual behavior (Raymond et al., 2003), compulsive buying (Lejoyeux et al., 1997), and problematic Internet use (Ko et al., 2010). Individuals high in sensation seeking may seek behaviors that provide excitement and produce high arousal (e.g., casino gambling, illicit sex) but may avoid others that do not.

Dissociation, which has been likened to a trance-like state, involves the separation of normally connected mental processes. It may promote ICDs. Dissociation has been reported in individuals with kleptomania (Grant, 2004), pathological gambling (Kofoed et al., 1997), and pathological skin picking (Odlaug & Grant, 2008).

In contrast to individuals with the formal or proposed ICDs, those with obsessive-compulsive disorder score high on measures of harm avoidance (Kim & Grant, 2001). Impulsivity and compulsivity are, of course, not mutually exclusive. Impaired inhibition of motor responses (impulsivity) has been found in individuals with obsessive-compulsive disorder and pathological skin picking (a behavioral addiction with arguably closer phenomenological links to obsessive-compulsive disorder), whereas cognitive inflexibility (thought to contribute to compulsivity) was limited to obsessive-compulsive disorder (Chamberlain et al., 2006; Odlaug et al., 2009).

Comorbidity

Another criterion that supports expanding the formal ICD category is the diagnostic overlap and

comorbidity among these disorders. If a relationship exists between the formal and the proposed ICDs, there should be evidence either that the proposed ICDs are overrepresented in patients with the formal ICDs and/or that the formal ICDs are overrepresented in patients with the proposed ICDs.

In terms of the formal ICDs, clinical samples of pathological gamblers demonstrate high rates of co-occurring lifetime compulsive sexual behavior (9.4%–17%) and compulsive buying (8.3%–25%; (Black & Moyer, 1998; Black, Monahan, & Gabel, 1997; Black, Kehrberg, Flumerfelt, & Schlosser, 1997; Grant & Kim, 2003; Specker et al., 1995). In individuals with kleptomania, elevated lifetime rates of the proposed ICDs are common (9.7% compulsive buying, 9.7% compulsive sexual behavior, 12.9% pathological skin picking; Grant, 2003; Presta et al., 2002). Similar findings have been reported for pyromania (19.0% compulsive buying, 9.5% compulsive sexual behavior, 9.5% pathological skin picking; Grant & Kim, 2007). Individuals with intermittent explosive disorder report high rates of all of the proposed ICDs, particularly compulsive buying (37%), and individuals with trichotillomania report elevated rates of pathological skin picking (53.5%; Lochner et al., 2005; McElroy et al., 1998; Stein et al., 2008; Table 4.1).

When we examine research on co-occurring ICDs among those with the proposed ICDs, we see generally the same elevated rates of comorbidity (Table 4.2). Studies of individuals with problematic Internet use have found high rates of compulsive buying (19%), pathological gambling (10%), pyromania (10%), and compulsive sexual behavior (10%; Black et al., 1999). Research on problematic Internet use has also found pornography to be the primary use in 8.1%–21% of Internet addicts (Dell'Osso et al., 2008b); Shapira et al., 2000).

Table 4.1 Lifetime Rates of the Proposed ICDs among Individuals with the Formal ICDs

Proposed ICDs	Pathological Gambling	Kleptomania	Pyromania	Intermittent Explosive Disorder	Trichotillomania
Problematic Internet use	10%	5%	10%	5%	0%
Compulsive buying	8.3%–25%	4.5%–9.7%	19.0%	37%	4.1%
Compulsive sexual behavior	9.4%–17%	9.1%–15%	9.5%	11%	*
Pathological skin picking	*	4.5%–12.9%	9.5%	4%	53.5%

*Unknown.

Table 4.2 Lifetime Rates of the Formal ICDs among Individuals with the Proposed ICDs

Formal ICDs	Problematic Internet Use	Compulsive Buying	Compulsive Sexual Behavior	Pathological Skin Picking
Pathological gambling	5%	5%–8.3%	4%–11%	*
Kleptomania	10%	4.2%–10%	13%–14%	1.7%–16%
Pyromania	*	0.0%–10%	8%	*
Intermittent explosive disorder	15%	4.2%–22%	3%–13%	*
Trichotillomania	*	4.2%–10%	6%	3.4%–38.3%

*Unknown.

High rates of co-occurring kleptomania (16%) and trichotillomania (9%–38.3%) have also been found in subjects with pathological skin picking (Arnold et al., 1998; Odlaug & Grant, 2008a; Wilhelm et al., 1999). Similarly, research on compulsive buying and compulsive sexual behavior has found that all of the proposed ICDs are overrepresented in these subjects (Black, Kehrberg, Flumerfelt, & Schlosser, 1997; Raymond et al., 2003).

These findings of elevated rates of co-occurrence may suggest that at least some of the proposed ICDs share a common pathophysiology with some of the formal ICDs. Studies have also found that pathological gamblers and individuals who are compulsively sexual (other ICDs have not been studied on this topic) have a greater incidence of attention deficit hyperactivity disorder (ADHD) as children (Kafka & Hennen, 2002; Specker et al., 1995). This suggests that in addition to possible shared pathophysiology between formal and proposed ICDs, there may be other variables (e.g., ADHD) that account for their co-occurrence as well.

Etiology

Ultimately, an assessment of the relationship of the proposed ICDs to the formal ICDs needs to consider their respective etiologies. Unfortunately, knowledge of these psychiatric disorders is not yet advanced enough to answer this question. Nonetheless, evidence suggests that corticostriatal circuitry mediates reward processes via dopamine activity within the ventral striatum (Chambers, Taylor, & Potenza, 2003). Several reports link dopamine agonist use in Parkinson's disease with a wide range of ICDs, both formal and proposed (pathological gambling, compulsive sexual behavior, compulsive buying, kleptomania, trichotillomania; Abosch et al., 2011;

Dodd et al., 2005; Driver-Dunckley, Samanta, & Stacy, 2003; Weintraub et al., 2010). These findings suggest limbic mechanisms in the association between both the formal and the proposed ICDs and dopamine agonist treatment. Furthermore, there is evidence that illicit drugs that increase the level of mesocorticolimbic dopamine (e.g., methamphetamine, cocaine) increase the symptoms of many of the ICDs (compulsive sexual behavior, skin picking), consistent with a possibly shared neurobiology (Grant & Potenza, 2006).

In addition to neurobiology, behavioral, cognitive, and dispositional attitudes may play a role in the etiology of both the formal and proposed ICDs. Behavioral and social learning theorists have focused on the role of direct and vicarious reinforcement in the development and maintenance of behaviors. Cognitive theorists have focused on information processing biases that inflate subjective estimates of succeeding in various behaviors (e.g., winning in gambling, finding sexual gratification) or that otherwise promote impulse behavior persistence. Dispositional traits such as impulsiveness, sensation seeking, neuroticism, and extraversion, as well as antisocial personality traits, have also been postulated as significant in the development of many of the formal and proposed ICDs (Baylé et al., 2003; Castellani & Rugle, 1995; Nower et al., 2004).

Positive reinforcement refers to the introduction of a hedonically positive consequence that strengthens a preceding response. The variable ratio of wins and losses built into gambling, or of success and failure built into shoplifting or compulsive sexual behavior, provides a particularly pathogenic formulation. The quintessential positive reinforcer of both formal and proposed ICDs is acquiring something rewarding (e.g., winning money, stealing/buying

an item, or performing sexual acts). The intermittent reinforcement (i.e., winning money, stealing items, or performing a sexual act on an unpredictably variable ratio) of most ICD behaviors describes a schedule of reinforcement that is particularly resistant to extinction, even in the absence of reinforcement over many trials.

There may be a range of reinforcers (other than the item of reward) available to people with ICDs that may serve to initiate and perpetuate the behavior (e.g., Ocean & Smith, 1993). These include social reinforcers (e.g., interaction with store employees for the compulsive buyer; Christenson et al., 1994), material reinforcers (e.g., drinks and other goods/services provided for gamblers), ambient reinforcers (e.g., a wide array of visual and auditory stimuli present in many stores for the kleptomaniac; Grant & Kim, 2002), cognitive reinforcers (e.g., near misses such as being one slot machine line away from a large payout for the pathological gambler; Griffiths, 1991), and even physiological arousal itself (e.g., the "rush" reported by people who set fires; Grant & Kim, 2007).

A negative reinforcement model based on the *self-medication* model may also apply to both the formal and the proposed ICDs. A number of studies have found elevated rates of depression and anxiety disorders in individuals with both formal and proposed ICDs. The lifetime prevalence of mood and anxiety disorders, depending upon the ICD, ranges from 28% to 100% and 33% to 80%, respectively (Black & Moyer, 1998; Christenson et al., 1991; Grant & Kim, 2001; McElroy, Pope Jr., Hudson, Keck, & White, 1991; McElroy, Hudson, Pope, & Keck, 1991; Schlosser et al., 1994). Depressed or anxious individuals may engage in the impulse behavior to distract themselves from life stressors and unpleasant cognitions (McCormick, 1994; Rosenthal & Lesieur, 1992). Persons who are depressed or anxious may also view gambling winnings, buying or stealing items, or having sex as a means of significant symptom relief and the risks of arrest, debt, or sexually transmitted diseases as a relatively minor setback. Ironically, problems resulting directly from ICDs (e.g., financial distress, relationship problems, criminal activity) may, in turn, lead to even more impulse control behaviors in a misguided attempt at symptom management.

An individual's need for tension reduction has been described as a possible contributing characteristic of both the formal and the proposed ICDs. For example, individuals who develop pathological gambling appear to have abnormal reactions to

stress (Goudriaan et al., 2004). Gambling behavior is thereby negatively reinforced in avoidance of negative affect or distress (Clarke, 2004). A study of New Zealand university students found that individuals who suffered from problem gambling were more depressed and impulsive than their non-problem-gambling peers (Clarke, 2004). Motivations reported by problem gamblers in this study were tension reduction, guilt, and feeling compelled to prove themselves to others. The need for tension reduction may be better conceptualized, in some individuals with ICDs, as an urge to engage in the problem behavior, which can be reinforcing.

In the case of pathological skin picking or trichotillomania, the impact of heightened levels of stress on psychosocial functioning has also been examined (Diefenbach et al., 2005; Odlaug & Grant, 2008b). These studies found that individuals with trichotillomania reported lower life satisfaction, higher levels of distress, and lower levels of self-esteem. The majority of subjects reported current problems with negative affect/negative self-evaluations. This affective experience is an important motivator for pulling or picking, serving as both a stimulus cue and a reinforcer of the behavior, and may apply to the other formal and proposed ICDs as well.

Responsiveness to Treatment

Based on the view that the proposed and the formal ICDs share common causal/maintaining factors, one could predict that these disorders would respond positively to the same treatments. In fact, various nonmedical treatment modalities that are effective in treating the formal ICDs are also helpful in treating the proposed ICDs. For example, 12-step approaches have been used in pathological gambling, compulsive sexual behavior, problematic Internet use, kleptomania, and compulsive buying. Cognitive behavioral therapies have also demonstrated benefit in controlled studies on pathological gambling, pathological skin picking, and compulsive buying (Grant, Donahue, Odlaug, Kim, Miller, & Petry, 2009; Mitchell et al., 2006; Mueller et al., 2008; Petry et al., 2006; Schuck et al., 2011; Teng et al., 2006). In addition, several influential psychosocial interventions for all of the ICDs rely on a relapse prevention model that encourages abstinence by identifying patterns of abuse, avoiding or coping with high-risk situations, and making lifestyle changes that reinforce healthier behaviors.

Although research has only recently begun to explore pharmacological treatment for ICDs, these studies have shown a similar response for the formal

and the proposed ICDs. Historically, the use of serotonergic medications for the formal ICDs (e.g., trichotillomania, pathological gambling, kleptomania) has resulted in mixed outcomes (Christenson et al., 1991; Grant et al., 2003; McElroy, Pope Jr., Hudson, Keck, & White, 1991; Saiz-Ruiz et al., 2005). Similarly, case reports and controlled studies of serotonergic medications in the treatment of the proposed ICDs show mixed results or limited efficacy (Bloch et al., 2001; Coleman et al., 2000; Keuthen et al., 2007; Koran et al., 2002, 2007; Simeon et al., 1997; Wainberg et al., 2006).

Unlike the serotonergic medications, other medications that have shown promise in treating the formal ICDs have also shown promise in treating the proposed ICDs. Given their ability to modulate dopaminergic transmission in the mesolimbic pathway, opioid receptor antagonists (naltrexone and nalmefene) have been investigated in the treatment of ICDs. Naltrexone, approved by the Food and Drug Administration for the treatment of alcoholism and opioid dependence, has been effective in reducing urges and the frequency of gambling in patients with pathological gambling (Grant et al., 2006, 2008; Kim et al., 2001) and the behaviors associated with kleptomania (Grant, Kim, & Odlaug, 2009). Although controlled studies are still lacking, naltrexone has shown promise in treating compulsive buying (Grant, 2003), compulsive sexual behavior (Raymond et al., 2002), problematic Internet use (Bostwick & Bucci, 2008), and pathological skin picking (Lienemann & Walker, 1989).

Agents that can improve glutamatergic tone within the nucleus accumbens (and thereby affect dopamine) may also reduce reward-seeking behavior (Kalivas & Volkow, 2005; Kalivas et al., 2005). Studies examining cocaine addiction in rats have demonstrated that *N*-acetyl cysteine, an amino acid, restores the extracellular glutamate concentration in the nucleus accumbens, blocks reconstitution of compulsive behaviors, and decreases cravings (Baker et al., 2003). One study examining *N*-acetyl cysteine in pathological gambling found that the medication reduced gambling urges and behavior (Grant, Kim, & Odlaug, 2007). A double-blind, placebo-controlled trial demonstrated the efficacy and tolerability of *N*-acetyl cysteine in 50 patients with trichotillomania (Grant, Odlaug, & Kim, 2009), and a case report (Odlaug & Grant, 2007) suggests that *N*-acetyl cysteine may be beneficial for pathological skin picking.

The positive outcomes found in both the formal and the proposed ICDs when treated with certain medications (opioid antagonists and possibly glutamatergic agents) provide evidence that the dopamine and possibly glutamate systems affected by these drugs may play roles in the pathophysiology of many of these disorders.

Conclusions

Early evidence suggests that the proposed ICDs share clinical characteristics, comorbidity, and possibly pathophysiology with the formal ICDs. There may be substantial heterogeneity within each ICD category. For example, perhaps only some pathological gamblers with strong urges share neurobiological underpinnings with some compulsively sexual individuals with strong urges. Disorder subtypes may be associated with neurobiological processes that overlap for both formal and proposed ICDs, whereas other subtypes may have a different etiology that is unrelated to the comorbid condition. Future research findings from genetic and brain imaging studies may help to identify key subgroup variables and serve as a methodology for identifying common biological substrates associated with both the proposed and formal ICDs.

Understanding the relationship of these ICDs will help refine views of the psychopathology and taxonomy of these conditions. In addition, by unraveling the shared and unique etiologies of these disorders, research may be better able to develop and test treatment and prevention strategies.

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The Compulsive-Impulsive Spectrum and Behavioral Addictions

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Abstract

Several models of psychopathology place constructs of compulsion and impulsivity in diametric opposition. There are, however, a number of other models for conceptualizing the relationship between compulsive and impulsive psychopathology. Here we discuss some of the symptomatic overlap and distinctions between compulsive and impulsive disorders (addressing also the notion of behavioral addiction), review some of the underlying psychobiological mechanisms that may account for these overlaps and distinctions, and briefly consider the implications of this phenomenological and psychobiological work for management. Understanding the overlaps and distinctions between the compulsive and impulsive disorders may be useful for both research and clinical purposes. A range of other approaches to impulsivity may, however, also be useful. Both compulsion and impulsivity are multidimensional constructs; further work is needed to delineate fully the nature of these dimensions and their underlying psychobiology.

Keywords: compulsion, impulsivity, behavioral addiction, obsessive-compulsive disorder, impulse control disorders

Introduction

Several models of psychopathology place constructs of compulsion and impulsivity in diametric opposition. Classical psychoanalytic theory, for example, has long contrasted patients with obsessive-compulsive neurosis and perfectionism to those with hysterical neurosis and hyperemotionality. A range of mechanisms have been posited to underlie such contrasts, ranging from the unconscious dynamics posited by psychoanalytic theory, through the more cognitive-behavioral processes of contemporary psychology, and on to the neurocircuitry and neurotransmitter mechanisms of clinical neuroscience (McElroy et al., 1994; Stein & Hollander, 1993b).

There are, however, a number of other models for conceptualizing the relationship between compulsive and impulsive psychopathology. One hypothesis, for example, has been that both

compulsion and impulsivity are characterized by impulse dyscontrol; thus, both compulsive disorders (such as obsessive-compulsive disorder) and impulsive disorders (such as pathological gambling) are characterized by repetitive thoughts and/or behaviors, while other conditions (such as Tourette's disorder) are characterized by a complex range of compulsive and impulsive symptoms (Grant & Potenza, 2006b; Stein & Hollander, 1993a).

In this chapter, we discuss some of the symptomatic overlap and distinctions between compulsive and impulsive disorders (addressing also the notion of *behavioral addiction*), review some of the underlying psychobiological mechanisms that may account for these overlaps and distinctions, and briefly consider the implications of this phenomenological and psychobiological work for management. Finally, we briefly review a number of other clinical models of impulsivity.

Compulsive and Impulsive Disorders and Symptoms

Obsessive-compulsive disorder (OCD) is arguably the paradigmatic compulsive disorder. It is characterized by obsessions—recurrent and persistent thoughts or urges that are experienced as intrusive and unwanted—and compulsions—repetitive behaviors or mental acts that the person feels driven to perform in response to an obsession or according to rigidly applied rules (American Psychiatric Association, 1994). Obsessions typically lead to an increase in anxiety or distress, while compulsions are typically aimed at preventing or reducing anxiety or distress. Although once it was thought uncommon, recent data suggests that OCD is present in 1%–3% of the population (Ruscio et al., 2008) and that it contributes significantly to the burden of disease (Murray & Lopez, 1996).

In recent years, several disorders have been posited to lie on an obsessive-compulsive spectrum of disorders (OCS) by virtue of their phenomenological and psychobiological overlap with OCD (McElroy et al., 1994; Stein et al., 1993b). Tourette syndrome (TS), for example, has important overlaps with OCD phenomenology (many OCD patients have tics, and many TS patients have obsessions and compulsions) and psychobiology (OCD is increased in family members of TS probands, and TS is increased in family members of OCD probands; Stein, 2000). Similarly, disorders such as body dysmorphic disorder and hypochondriasis, which are associated with somatic preoccupations and consequent ritualistic behavior, appear to have considerable phenomenological overlap with OCD and possibly some overlap in underlying psychobiology (Castle & Phillips, 2006).

Nevertheless, there is ongoing debate about which disorders are appropriately described as OCS disorders, with some authors taking a more inclusive approach and others a more exclusive one (Bartz & Hollander, 2006; Storch et al., 2008). There is a particular lack of consensus, perhaps, about the relationship between OCD and the *behavioral addictions*, such as pathological gambling and sexual addiction (Mataix-Cols et al., 2007). On the one hand, such conditions may be characterized by preoccupations and repetitive behaviors (e.g., gambling). On the other hand, whereas OCD involves anxiety and avoidance of feared stimuli, such disorders are characterized by reward-seeking behavior (Grant & Potenza, 2004).

The DSM-IV section on impulse control disorders not elsewhere classified includes not only pathological

gambling, but also a number of other conditions such as trichotillomania, kleptomania, and intermittent explosive disorder. These disorders overlap with pathological gambling in that they are characterized by the failure to resist an impulse to perform a behavior that is harmful to the person or to others (American Psychiatric Association, 1994). DSM-IV does not address the concept of behavioral addiction, but it notes that many of the impulse control disorders are associated with an increasing sense of tension or arousal prior to the behavior and an experience of pleasure, gratification, or relief at the time of the behavior—features that are key to this construct.

To some extent, the operational criteria for compulsive and impulsive disorders put these conditions in opposition. Although compulsive disorders are found in different parts of the DSM-IV manual, OCD is an anxiety disorder, and repetitive actions in OCSs (e.g., body dysmorphic disorder, hypochondriasis) are done to prevent or decrease feelings of anxiety and distress. In contrast, the behavioral addictions involve feelings of pleasure and gratification. On the other hand, as demonstrated by the debate about which disorders belong on the OCS, both groups of disorders involve repetitive behaviors, and DSM-IV notes that obsessions can be “impulses” and that impulsive behavior may lead to a range of distressing feelings.

Clinical experience shows that many patients have a complex mix of both compulsive and impulsive symptoms. A patient with trichotillomania, for example, when hair pulling, may display repetitive and ritualistic behavior—searching for just the right hair, handling it, and pulling it in a very specific and sequenced way, bringing it to the lips, and biting off the root according to a particular pattern. At the same time, the patient’s behavior may be considered impulsive in the sense that he or she resists the urge to pull hair for the whole day, comes home and has an upsetting interaction with a family member, and then suddenly chooses to go to his or her room to engage in a bout of hair pulling. Similar combinations of compulsive and impulsive features are seen in disorders ranging from OCD to pathological gambling.

A number of studies have attempted to clarify the relationship between compulsive and impulsive disorders by exploring their comorbidity. Several findings emerge. First, within OCD, there is a group of patients with comorbid impulse control disorders or significant symptoms of impulsivity (Fontenelle et al., 2005; Grant et al., 2006; Matsunaga et al.,

2005; Stein et al., 1994; Summerfeldt et al., 2004; Thomsen & Jensen, 1991). Such impulsivity may be associated with increased OCD symptom severity and treatment refractoriness (Matsunaga et al., 2005). Second, a number of OCSs are similarly associated with impulsivity, with TS being a condition that exemplifies the coexistence of compulsive and impulsive disorders and symptoms (Ferrao et al., 2009). Cluster analysis of OCSs in OCD patients indicates that these fall into separate groups, including one in which several behavioral addictions (e.g., pathological gambling, sexual addiction) cluster (Lochner et al., 2005). Third, epidemiological and clinical studies of impulse control disorders indicate that these have high rates of comorbidity with other impulse control disorders, with relatively low rates of OCD (Grant et al., 2006; Moeller et al., 2001). A possible exception is trichotillomania, in which rates of OCD are higher than might be expected (Ferrao et al., 2009).

Taken together, these kinds of data perhaps provide some support both for the argument that compulsivity and impulsivity differ in important ways (e.g., only some patients with OCD have comorbid impulsivity, low rates of OCD in impulse control disorders) and for the contrasting view that compulsivity and impulsivity have important overlaps (e.g., when such comorbidity exists, it may have particular clinical significance). Still, several additional factors should be considered before reaching a final decision about how closely the symptoms of compulsive and impulsive disorders overlap. In particular, there is considerable heterogeneity within each of these conditions. Thus OCD is characterized by a number of different symptom dimensions, each of which may have a somewhat different psychobiology (Mataix-Cols et al., 2005). Patients with symmetry obsessions and compulsions, for example, have symptoms that may involve a certain degree of reward seeking (patients may describe a sense of pleasure once everything is “just right”) and where the treatment response may differ subtly (in particular, such patients may be less likely to respond to serotonin selective reuptake inhibitors; Stein et al., 2008).

Psychobiology of Compulsivity and Impulsivity

Data indicating that compulsive and impulsive disorders require different treatment approaches raise the question of whether they have contrasting underlying psychobiology. Once again, we will argue that there are both overlaps and distinctions.

In recent decades, a wealth of data from brain imaging studies has become available on the neurocircuitry of OCD and other anxiety disorders. In general, there seems to be a key differentiation between OCD—which involves frontostriatal circuitry (Rotge et al., 2009)—and other anxiety disorders—in which the amygdala and other limbic structures play a key role (Etkin & Wager, 2007). At the same time, there may also be some overlaps between OCD and other anxiety disorders—in particular, it is possible that fear extinction plays an important therapeutic role in both disorders—and that across disorders, such fear extinction may involve prefrontal circuitry succeeding in suppressing processes mediated by subcortical structures (whether involving the striatum or the amygdala; Milad et al., 2006; Stein, 2008).

There is relatively less information available on the neurocircuitry of a number of the putative OCS disorders. Preliminary information suggest that TS, body dysmorphic disorder, and trichotillomania each involves somewhat different neuronal circuits from those thought to play an important role in OCD. For example, TS appears to involve sensorimotor regions, body dysmorphic disorder may involve parietal and temporal regions, and trichotillomania may involve the cerebellum (Stein, 2008). Again, however, there may be some overlap across these conditions; for example, the striatum has been implicated in each of them. Further, it has been argued, on the basis of neuropsychological and imaging data, that failures in behavioral and cognitive inhibition are a key endophenotype for understanding OCD and related disorders (Chamberlain et al., 2005, 2006; Watkins et al., 2005).

Impulse control disorders and impulsivity may be mediated by a range of different brain structures and regions. First, damage to prefrontal regions, which play a key role in executive functions, may lead to behavioral disinhibition. The classic case of Phineas Gage exemplifies the behavioral impact of orbitofrontal damage, with subsequent impulsivity and related psychopathology (Damasio et al., 1994). Frontal hypoactivity or other abnormalities are also seen in a number of impulsive disorders, including psychopathy and substance use disorders (Berlin et al., 2005; Davidson et al., 2001; Moeller et al., 2001). Second, impulsivity may possibly be associated with increased activity in limbic regions, which govern reward-seeking behavior (Chambers et al., 2003). Patients with temporal lobe epilepsy may, for example, display hypersexual symptoms.

These kinds of findings may apply to a number of the behavioral addictions, including pathological gambling (Stein & Grant, 2005). Thus, patients with pathological gambling may have decreased activity in brain regions involved in impulse control (e.g., orbitofrontal cortex, anterior cingulate) compared with normal controls (Potenza et al., 2003). During reward paradigms, there is activation of the ventral striatum (which includes the nucleus accumbens; Knutson et al., 2001), and it may be hypothesized that there is hyperactivation of such structures in some patients with behavioral addiction.

The group of impulsive-addictive disorders that has, however, received the most attention from basic and clinical researchers focused on neurocircuitry is the substance abuse disorders. Animal studies have emphasized the role of frontostriatal circuitry in habitual behavior, including the development of repetitive drug-seeking behavior (Cardinal et al., 2004). Human studies have similarly noted that regions involved in substance use disorders include both orbitofrontal cortex as well as a range of subcortical structures, including the striatum (Chang et al., 2007; Goldstein & Volkow, 2002).

Taken together, then, it appears that while the neurocircuitry of OCD and of impulsivity differ significantly, there is also some evidence for the involvement of frontostriatal circuitry across compulsive and impulsive disorders. Parallel, functionally segregated corticostriatal projections from the orbitofrontal cortex (OFC) to the medial striatum (caudate nucleus) may underlie compulsive activity, while those from the anterior cingulate/ventromedial prefrontal cortex to the ventral striatum (nucleus accumbens shell) may drive impulsive activity (Fineberg et al., 2009). (Fineberg et al., 2010) Thus, compulsive and impulsive disorders appear to be mediated by overlapping as well as distinct neural substrates.

Several factors, however, need to be considered before drawing general conclusions about the neuroanatomy of impulsivity and compulsivity. First, there is a relative paucity of imaging studies using the same methodology, or studying the same paradigm, across disorders. Second, despite important advances in brain imaging methodologies, current datasets often remain open to various interpretations. The increased activity in frontal circuitry in OCD, for example, remains poorly understood. It may represent primary pathology in this region, or it may represent a compensatory response to striatal pathology. Each interpretation would entail a different view of the relationship between compulsive and impulsive disorders.

Management of Compulsivity and Impulsivity

As briefly alluded to earlier, the pharmacotherapy of compulsive and impulsive disorders may also demonstrate some overlap, but also key differences. A key principle of the pharmacotherapy of OCD is that serotonin reuptake inhibitors (SRIs) are more robust than noradrenergic reuptake inhibitors (NRIs). Indeed, one approach to defining the OCSs is to consider which disorders are more responsive to SRIs than NRIs; this would include body dysmorphic disorder, trichotillomania, and a number of other conditions (Rapoport & Wise, 1988; Stein, 2000). This principle is not entirely straightforward, though, in that SRIs may be effective for a range of conditions that seem to have little to do with OCD (e.g., premenstrual dysphoric disorder).

A second important principle is that low-dose antipsychotic agents are a useful option in the augmentation of SRIs in the management of treatment-refractory OCD patients (Bloch et al., 2006; Ipser et al., 2006). As noted earlier, many patients with OCD have comorbid tics, providing a line of evidence that OCD may involve not only the serotonin system, but also the dopamine system (Denys et al., 2004). These agents may also be useful in a number of OCSs, including TS and trichotillomania, but they do not appear to be uniformly useful; for example, there is relatively little evidence of their value in body dysmorphic disorder (Ipser et al., 2009).

A third important principle of the management of OCSs is that exposure and response prevention is effective in the management of OCD, while habit reversal therapy is effective in the management of a number of OCSs, including TS, trichotillomania, and skin-picking disorder (Stein et al., 2009). Although cognitive interventions have also been shown to be effective in some of the OCSs, the majority of the data from the field emphasize the value of these more behavioral approaches. Although there is surprisingly little evidence that combined pharmacotherapy and psychotherapy is better than either modality alone, there are data indicating that in OCD patients who fail to improve with either pharmacotherapy or psychotherapy, each may usefully be augmented with the other modality.

It is notable that studies on SRIs in the impulse control disorders have been somewhat inconsistent. For example, an early trial gave the OCS field significant impetus by suggesting that trichotillomania did indeed show a selective response to clomipramine (Swedo et al., 1989). However, subsequent

data on the pharmacotherapy of this disorder have not been as consistent in showing overlap with OCD (Bloch et al., 2007). Similarly, early positive findings on SRIs in pathological gambling were not replicated in subsequent placebo-controlled trials. Furthermore, clomipramine could not be shown to be more effective than desipramine in paraphilic disorders (Kruesi et al., 1992).

The pharmacotherapy of impulse control disorders remains an ongoing area of study, with several other agents receiving attention (Grant & Kim, 2006; Moeller et al., 2001). Opioid antagonists seem to be a promising class of agents for certain behavioral addictions, such as pathological gambling (Grant et al., 2006). In contrast, there has been little work on opioid agents in OCSs, although there are preliminary data indicating that morphine may be useful in the management of OCD (Koran et al., 2005) and that naltrexone is useful in trichotillomania. Nevertheless, effect sizes for pharmacotherapy tend to be relatively low in studies of impulsive-addictive conditions. In addition, based on anecdotal experience, many clinicians feel that pharmacotherapy for such disorders should be used only within the context of a working therapeutic alliance and should be combined with psychotherapeutic intervention.

Indeed, the principles of psychotherapy for impulse control disorders and behavioral addictions also appear to differ from those used in the OCSs. Cognitive therapy, focused on beliefs about gambling, for example, appears effective in pathological gambling. Similarly, psychotherapy for substance use disorders focuses on reinforcing abstinent behavior and restructuring belief systems. A range of evidence-based psychotherapies for the impulsive personality traits characteristic of borderline personality disorder is now available, exemplifying the multiple factors that need to be addressed when attempting to alter impulsive-addictive symptoms (e.g., emotional regulation, interpersonal relationships, underlying schemas).

Stein and colleagues have suggested an A-B-C pharmacotherapy approach to a number of disorders characterized by repetitive behavior (Stein et al., 2006). Affective dysregulation is likely to play a role in a number of compulsive and impulsive conditions; many habits and behavioral addictions are, for example, exacerbated at times of stress. Serotonin reuptake inhibitors may modulate the relevant neurocircuitry, improve emotional regulation, and so contribute to decreasing behavioral dyscontrol (Grant et al., 2003; Stein et al., 2007).

Behavioral addiction may also be important in maintaining repetitive behaviors. Antipsychotics, opioid agents, and other medications that act on reward circuitry may therefore be useful in attenuating the relevant processes and decreasing symptomatology in certain compulsive and impulsive disorders (Grant et al., 2006, 2007; Ipser et al., 2006). Cognitive control is important in modulating habits and behavioral addictions, and a range of medications may be useful in improving such executive control. In particular, glutamatergic agents may act by modulating top-down control of subcortical regions (Grant et al., 2009). This kind of framework may provide a heuristic approach to treatment planning. Nevertheless, it undoubtedly entails a theoretical oversimplification of the complex psychobiological processes that are at play in compulsive and impulsive conditions, and much further clinical trials work is needed to develop the empirical evidence base for making optimal pharmacotherapy choices in this area.

Other Models of Impulsivity

If constructs of compulsion-impulsivity and of behavioral addiction provide only a partial perspective on the phenomenology, psychobiology, and management of impulse control disorders, what other models of impulsivity are theoretically valid and clinically useful? In this final section of the chapter, we briefly mention a number of important approaches to impulsivity and their consequent clinical implications. The particular approaches mentioned have emerged primarily from clinical theory and practice, although there is also a range of important basic neuroscience work that has the potential to ultimately influence clinical practice (Dalley et al., 2007; Winstanley et al., 2004).

One approach to impulsivity emphasizes the possibility that many psychopathological symptoms characterized by impulsiveness reflect underlying mood disorders (Moeller et al., 2001). McElroy and colleagues (1996), for example, have argued that impulse control disorders and bipolar disorder share a number of features, including phenomenological similarities, early onset and an episodic course, high comorbidity with one another and similar comorbidity with other psychiatric disorders, elevated familial rates of mood disorder, possible abnormalities in monoamine neurotransmission, and responsiveness to mood stabilizers and antidepressants. They also note, however, that certain impulsive control disorders may be more closely related to OCD than is bipolar disorder. They therefore broaden the

compulsive-impulsive approach to hypothesize that impulsivity and related bipolarity (or mania) represent one end of a spectrum, while compulsion and related unipolarity (or depression) represent the opposite pole.

A second approach to impulsivity has emphasized the possibility that many psychopathological symptoms characterized by impulsiveness reflect underlying attention deficit hyperactivity disorder (ADHD; Schachar et al., 1993). Certainly, a key set of symptoms in ADHD are characterized by impulsivity. Furthermore, patients with ADHD may have comorbid impulse control disorders, and there is arguably some overlap in the underlying psychobiology of, and management approach to, ADHD and impulse control disorders (e.g., in terms of psychotherapy interventions). This view is useful insofar as ADHD is often underdiagnosed and undertreated, particularly in adult populations. At the same time, there also seem to be significant differences in the psychobiology and management of ADHD and impulse control disorders; in particular, psychostimulants are highly effective in the management of ADHD but have only a limited role in the treatment of impulse control disorders (Pliszka et al., 2006).

A third approach to impulsivity has emphasized the importance of underlying impulsive personality traits in accounting for impulse control disorders and behavioral addictions. This view has roots in early psychodynamic thinking, but it is also seen in a range of more contemporary cognitive-behavioral and psychobiological models that emphasize that impulsive symptoms reflect an underlying tendency to gratify urges without sufficient consideration of the consequences. Siever and colleagues, for example, have demonstrated that impulsive personality traits involve particular neurocircuits and molecules, which overlap with those responsible for a range of Axis I disorders with related symptomatology (New et al., 2009; Siever & Davis, 1991). This view may be particularly attractive to those who are skeptical about the syndromal status of a number of different impulse control disorders or who wish to view impulsiveness as a dimension of symptomatology that cuts across a range of different psychiatric disorders.

A fourth approach to impulsivity has emphasized environmental precipitants of impulsive psychopathology. Early adversity, for example, results in a range of subsequent psychopathology, including impulsive symptoms and traits (Stein et al., 2005). Stressors may act in a cumulative way, so that

early exposure to trauma, plus subsequent exposure to stressors, is particularly likely to lead to symptomatology. There is a range of research on the underlying mechanisms, at a neurocircuitry and neurotransmitter level, that are responsible for this phenomenon (Faure et al., 2006). Additional environmental precipitants of impulsive symptoms include exposure to substances (Moeller et al., 2001) and to infectious agents (Swedo et al., 1998). Further, there may well be cross-sensitization between different stressors, and the underlying mechanisms of such sensitization are becoming increasingly well understood (Koob & Kreek, 2007).

This discussion of different approaches to understanding impulsive symptomatology is not intended to be a comprehensive one. Furthermore, it is pertinent to understand that there is significant overlap across a number of these disorders. As noted earlier, key aspects of different models of impulsivity include the notion that there is insufficient top-down regulation with concomitant executive impairment; that there is increased bottom-up reward seeking with associated urges; and that various kinds of stressors may act to further increase the likelihood of psychobiological mechanisms such as frontal-limbic disconnection or monoaminergic sensitization, with consequent emotional dysregulation and impulsive symptomatology (Stein et al., 2006). Indeed, impulsivity is a heterogeneous construct (Evenden, 1999), and the clinical treatment of patients with impulsive symptoms requires a flexible, multimodal approach that addresses a range of different factors that may be operating within a particular individual.

Conclusion

The central argument of this chapter is that understanding the overlaps among and distinctions between the compulsive and impulsive disorders may be useful for both research and clinical purposes. A range of other approaches to impulsivity may, however, also be useful.

Further research is needed to fully understand the constructs of both compulsion and impulsivity. It seems clear that both are multidimensional constructs; further work is needed to delineate fully the nature of these dimensions and their underlying psychobiology (Stein & Lochner, 2006). A translational approach is likely to be key; animal models of both compulsive and impulsive phenomena have been developed, and further exploration is likely to influence and be influenced by clinical research. At the same time, a good deal of research may need to

focus on human subjects; there are aspects of human compulsivity and impulsivity that cannot be explored solely with animal models.

From a clinical perspective, it may be useful to consider the role of certain agents, such as the SRIs, in the treatment of both OCD and a range of impulse control disorders and behavioral addictions. Similarly, given the possibly important role for reward circuitry in both compulsive and impulsive disorders, there may be value to considering the role of dopaminergic and opioid agents in both of these groups of conditions. There is also considerable current interest in the possible role of glutamatergic agents in both compulsivity and impulsivity. At the same time, given the differences between these disorders, it is important to consider differences in the approach to the treatment of compulsive and impulsive disorders. Whereas OCD requires concurrent cognitive-behavioral therapy focusing on exposure and response prevention, impulse control disorders require alternative forms of psychotherapy.

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Aggression, Impulsivity, and Personality Disorders

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Abstract

Aggressivity and impulsivity are traits that are core features of the Cluster B personality disorders. Within these disorders, impulsive aggression leads to a significant amount of morbidity and mortality. This type of behavior is intrinsically linked to violence, suicide, and substance abuse. In this chapter, we will discuss the phenomenology of these traits, the neurobiology of impulsive aggression, and some potential treatment options. We will conclude with some thoughts on the future direction of research in this field.

Keywords: personality disorders, impulse control disorders, impulsive aggression neurobiology, serotonin, orbitofrontal cortex, TPH2, 5HT2A, treatment

Introduction

Aggressivity and impulsivity are ubiquitous personality traits found throughout societies and cultures across the world (Swanson et al., 1990). Unfortunately, they lead to a significant amount of morbidity and mortality. They are intrinsically linked to violence, suicide, and substance abuse. These traits are also responsible for a large portion of the morbidity and mortality associated with several personality disorders.

Aggression is defined as hostile, injurious, or destructive behavior. It may further be classified as self-directed versus other-directed, physical versus verbal, pathological versus functional, and planned versus impulsive. With the exception of functional aggression, each of these variations is seen to a greater degree in certain personality disorders.

Impulsivity does not have as clear a consensus definition. In the simplest model, impulsivity is a decreased capacity to inhibit behavioral responding. Others have viewed impulsivity as increased risk-taking behavior, nonplanning, disregard for future consequences, or a preference for immediate gratification. We will analyze the relations of each

of these concepts in combination with aggression to personality disorders.

Aggression and impulsivity are found separately within personality disorders; however, they also are often found to occur together. When individuals have a tendency to act aggressively in what we have defined as an impulsive manner, that is, with disregard for future consequences and a preference for immediate gratification, we label the behavior as *impulsive aggression*. This type of behavior is a well-studied phenomenon within personality disorders and has extremely important clinical significance. We view patients with a history of impulsive aggression as high-risk and devote significant resources in attempts to ensure their own safety and that of individuals within their environment. Therefore, a better understanding of impulsive aggression may be valuable to efforts intended to curb this potentially dangerous type of behavior. In this chapter, we will explore the phenomenology of these traits (acting both separately and together) in personality disorders, their clinical measurements, the proposed biological mechanisms underlying them, treatment strategies, and future directions in research.

Phenomenology

As described in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed, Text Revision; DSM-IV-TR), a personality disorder can be diagnosed when a person's behavior deviates from that of his or her own culture to the extent that there is functional impairment. The manual lists four areas in which a person's behavior can lead to clinically significant distress: (1) cognition,(2) affect, (3) interpersonal functioning, and (4) impulse control (APA, 2000). Two of these four areas must be present to give a diagnosis of personality disorder. However, significant deficits in one area will likely contribute to problems in one of the other spheres of behavior. For example, an individual who is suffering from extreme paranoia is more likely to react in an impulsive-aggressive manor. It has also been shown that individuals with cognitive disorders, such as patients with Alzheimer's disease, are more likely to exhibit aggressive behavior (Burns et al., 1990). Further, researchers have shown that when a demented individual exhibits depressive symptoms, he or she is more likely to act in a physically aggressive manor (Lyketsos et al., 1999). Susceptibility to aggression may also be enabled by an anxiety state, as in generalized anxiety disorder or panic disorder (Swann, 2003).

From the previous discussion, it follows that in individuals diagnosed with personality disorders, who by the nature of their illness are prone to cognitive distortions and emotional dysregulation, there should be a significant amount of impulsive aggression, which indeed has been found. The DSM-IV further divides the personality disorders into three clusters: Cluster A (odd or eccentric), Cluster B (dramatic, emotional, or erratic), and Cluster C (anxious or fearful). Of the clusters, Cluster B is the one most clearly associated with increased impulsivity and aggression. Both borderline personality disorder and antisocial personality disorder are associated with higher scores on various measures of impulsivity (Morey et al., 2002). As well, aggressive behavior is seen to a greater degree in this patient population (Blair, 2001). As a result, individuals in this cluster have higher rates of suicide, domestic violence, criminal behavior, and substance abuse. Borderline personality disorder patients have a higher rate of suicide attempts than those in all other diagnostic groups, and this is the only diagnosis in which suicidality is a core feature (Soloff et al., 2005). Thus, these behaviors can lead to serious clinical consequences, and understanding their pathophysiology may lead to improvements in clinical treatment.

It is difficult to predict which patients will react in impulsively and aggressively. Beyond a history of this type of behavior, researchers have devised both self-report and laboratory measures in order to diagnose and predict which patients are more likely to act in an impulsive and/or aggressive manor. These measures are used to conduct research on the behavioral traits as well as inform clinical decisions.

Self-Report Measures

The most famous and most widely used self-assessment for impulsiveness is the Barratt Impulsiveness Scale (Patton et al., 1995) developed by Ernest S. Barrat. It is a 30-item self-report instrument designed to assess the personality construct of impulsiveness. Barratt began working on the scale in the 1950s as a unidimensional construct assessing impulsivity as a linear trait. After further analysis, he concluded that impulsiveness was actually a multi-faceted characteristic composed of three subtraits. Thus, the BIS-10 was redesigned to measure these three subtraits. Barratt labeled these subtraits *cognitive impulsiveness*, *motor impulsiveness*, and *non-planning impulsiveness*. Although the scale can be analyzed using these three subtraits, the majority of studies report the total impulsiveness score.

If we are exploring the role of aggression and impulsivity in personality disorders, the next logical question is: How do individuals with personality disorders perform on this self-report? There are numerous reports in the literature of patients with borderline personality disorder and antisocial personality disorder having predictably high scores on this measure. Dougherty compared the scores of 14 hospitalized women with borderline personality disorder and 17 comparison individuals (Dougherty et al., 1999). As expected, he found that the patients with borderline personality disorder had higher scores for impulsivity. Thus, this self-report measure can be a valuable tool in assessing impulsivity in personality disorders.

Laboratory Measures

While self-report measures are useful in the assessment of impulsivity and aggression, some researchers argue that they require the individual to have significant insight and good ability to relate his or her own history, qualities that are often lacking in individuals with personality disorders. Aside from a clinical history of impulsive aggression, some investigators argue that laboratory behavioral measures of these traits are the best means to assess these characteristics. They further state that a laboratory

measure is better able to evaluate temporary alterations in an individual's behavior that are more reflective of impulsive aggression rather than having an individual describe long-standing characteristics of his or her personality (Dougherty et al., 2005). To this end, many laboratory paradigms have been created that attempt to simulate real-life impulsivity and aggression. We will discuss several of them and their relevance to personality disorders.

The Point Subtraction Aggression Paradigm (PSAP) is a laboratory test for aggression that can elicit aggressive responses in individuals susceptible to aggression and therefore assay the predisposition to aggression that may not always manifest itself in the individual's natural environment—for example, when such an individual isolates himself or herself or avoids provocation. The PSAP relies on an assessment of aggressive responses in a computerized game in which subjects believe that they are playing in a fictitious network of individuals. When subjects find that points are being taken from them by someone they presume to be another player (generated by computer), those predisposed to aggression may "retaliate" by taking points from the perceived other subject. The number of aggressive responses correlates with aggressive behavior and has been validated in violent parolees (Cherek et al., 1997). Numerous reports have shown higher scores on the PSAP for individuals with personality disorders. In fact, one study found that higher scores on the PSAP, and therefore aggressive behaviors, were correlated more with Cluster B traits than with testosterone levels in weightlifters (Perry et al., 2003).

The go/no-go task measures the capacity to inhibit responses, which is clearly relevant to the impulsivity aspect of impulsive aggression. There are various modifications of the go/no-go task, but the basic premise is that a subject is presented with two different images, one that he or she is meant to respond to and one to which he or she is meant to withhold a response. It is the ability to withhold a response that has been correlated with impulsivity. And indeed, it has been shown in numerous reports that individuals with Cluster B traits have a harder time withholding responses compared to normal controls (Ruchsow et al., 2008).

The TIME paradigm has also played an important role in studying the impulsivity of personality disorders. The test is fairly straightforward: The examiner asks the subject to estimate a specific amount of time. The subject's response is then compared to the actual time. Individuals who act impulsively tend to overestimate the actual time (Barrat &

Patto, 1983). Given our definition of impulsivity, this is somewhat intuitive in that, at its core, impulsiveness involves acting too quickly. This is true of borderline personality disorder patients as well. In studies, these patients have been found to overestimate time when compared to normal controls (Berlin & Rolls, 2004). Besides their having a faster internal clock, this result may also be explained by their inability to wait as time passes.

By collecting these measures, researchers have been able to identify individuals who have a tendency toward impulsive aggression. Further, they have been able to study these traits within personality disorders. Once the traits have been identified in personality disorders, scientists can study the phenotypes of these individuals through different lenses in order to better characterize them by relating them to neurobiology and genetics, as discussed below.

Neuroanatomy

Frontal Lobe

We have known for a long time that damage to certain areas of the brain results in impulsive-aggressive behavior. Specifically, damage to the prefrontal cortex removes frontal control from subcortical impulses. This phenomenon was well illustrated in the case of Phineas Gage, who went from a dependable, stable railroad worker to an angry, irritable man after a tamping rod penetrated his skull and entered his orbitofrontal cortex (Damasio et al., 1994). Further, it has been shown that damage to the ventromedial prefrontal cortex results in severe disruption of emotional control (Anderson et al., 2006).

Temporal Lobe

The temporal lobe has also been implicated in impulsive and aggressive behavior. Tumors of the temporal lobe have been reported to increase aggressive behavior (Tonkonogy & Geller, 1992). Temporal lobe epilepsy has been shown to be associated with subacute postictal aggressive behaviors (Ito et al., 2007). Further, the famous Geschwind syndrome associated with seizures in the left temporal lobe is accompanied by a variety of impulsive behaviors (Waxman & Geschwind, 1975).

Neuroimaging Data

With the knowledge that damage to specific areas leads to impulsive-aggressive behavior, investigators set out to explore whether inherent structural differences in the brain may account for differences

in phenotypic behaviors. In fact, scientists have shown reduction in prefrontal gray matter in individuals with antisocial personality disorder (Raine et al., 2000). As well, significant volume reductions have been demonstrated in the left orbitofrontal cortex and the right anterior cingulate gyrus in patients with borderline personality disorder, most markedly in Brodmann's area 24 (Hazlett et al., 2005).

More recently, neuroimaging techniques like positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have allowed us to examine the dynamic function of the brain and to assess functional activity in various normal and pathological behaviors. When these techniques are applied to individuals with personality disorders, we are able to correlate regional brain activity, or lack thereof, with pathological behavior in personality disorders. An inverse relationship has also been reported between the history of impulsive-aggressive behavior and glucose metabolism in the orbitofrontal cortex and right temporal cortex, with reductions of metabolism in prefrontal Brodmann's areas 46 and 6 in patients with borderline personality disorder (Goyer et al., 1994). Using the previously described PSAP, patients with borderline personality disorder who were characterized by anger dyscontrol showed diminished responses to provocation in the medial frontal cortex and the anterior frontal cortex relative to comparison subjects but greater responses in the orbital frontal cortex. This result may be explained by their desire to dampen aggressive responses (New et al., 2006).

Another relatively new imaging technique that may help us elucidate the underlying anatomical abnormalities seen in personality disorders is diffusion tensor imaging (DTI). This technique is simply a reformatting of the standard MRI using specialized software (Le Bihan et al., 2001). The basic premise behind the technique is that water in intact myelinated nerves should diffuse uniformly in one direction. However, when nerves are damaged or misaligned, the water is free to diffuse in all directions. By measuring the diffusion of water, we can extrapolate the integrity of white matter in various regions of interest. This technique has been applied to borderline personality disorder and associated impulsivity. One study that employed DTI found that women with borderline personality disorder and self-injurious behavior exhibited decreased white matter microstructural integrity in brain regions that included components of orbitofrontal circuitry (Grant et al., 2007). Given its relative ease

of administration and favorable side-effect profile, we will likely see more researchers using DTI.

Limbic System

The other critical abnormality implicated in impulsive aggression is hyperactivity of the limbic system, including structures such as the amygdala, in response to negative or provocative stimuli, particularly anger-provoking stimuli. This aggressive diathesis can be conceptualized in terms of an imbalance between the "top-down" control or "brakes" provided by the orbitofrontal cortex and anterior cingulate cortex, which are involved in calibration of behavior to social cues and predicting expectancies of reward and punishment (Blair, 2004), modulating or suppressing aggressive behavior with negative consequences, and excessive "bottom-up" "drives" triggered or signaled by limbic regions such as the amygdala and insula (Figure 6.1).

An imbalance between limbic drives and prefrontal control mechanisms may be important in the impulsive aggression we see in personality disorders. Genetic and physiological susceptibilities, in conjunction with an experiential history of aggression, may serve to condition the responses to emotional provocation in the direction of aggression. For example, fMRI studies suggest that patients with borderline personality disorder, who often experience anger and aggression dyscontrol, are particularly sensitive to faces with angry expressions (Best et al., 2002; Coccato et al., 2007b). Activation of these systems in the face of diminished top-down regulation can lead to disinhibited anger and aggression. Enhanced responses of the amygdala have been reported in patients with borderline personality disorder toward negatively valenced pictures (Herpetz et al., 2001), faces with neutral, positive, and negative emotions (Donegan et al., 2003), and traumatic scenes (Schmahl et al., 2004) relative to healthy comparison subjects.

As we learn more about the neuroanatomical correlates of impulsive aggression within personality disorders, our understanding of the physiological basis of this behavior grows as well. With further understanding comes the promise of improved treatments. One area where there has been some steps toward clear treatment has been with neurotransmitters, which are the focus of the next section.

Neurotransmitters

With advances in the neurosciences, the significant role neurotransmitters play in controlling various behaviors is becoming more evident. For review, a

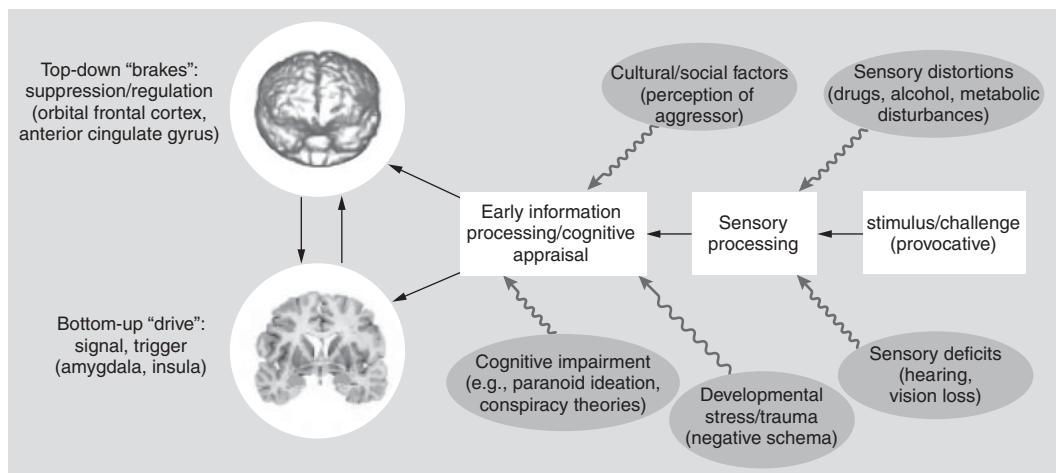


Fig. 6.1 Limbic system.

Source: Adapted/modified with permission from S. J. DeArmond et al. (1989) and Oxford University Press.

neurotransmitter is a chemical that is synthesized on the presynaptic side of the synapse and is present in sufficient quantity in the presynaptic neuron to affect the postsynaptic neuron; there are postsynaptic receptors for it, and a mechanism for inactivation is present. Researchers and clinicians have devised several ways to evaluate the role of neurotransmitters in psychiatric pathology. Measuring cerebrospinal fluid (CSF) levels of given neurotransmitters, evaluating clinical behavior in response to neurotransmitter probes, and radiolabeled imaging of neurotransmitter binding sites are some of the ways investigators have studied the role of neurotransmitters in behaviors associated with personality disorders.

Serotonin

Serotonin is the best studied of the neurotransmitters in terms of regulating impulsive and aggressive behavior. It has been postulated that it helps to regulate areas in the orbitofrontal cortex that act to suppress aggressive behaviors. Therefore, lower serotonin levels would lead to increased impulsive-aggressive behavior in patients with personality disorders. This theory has been validated by studies showing that treatment with selective serotonin reuptake inhibitors (SSRIs) leads to a reduction in impulsive-aggressive behavior in personality-disordered subjects (Coccaro & Kavoussi, 1997). Further studies have shown decreased levels of the serotonin metabolite 5-hydroxyindolacetic acid (5-HIAA) in aggressive personality disorder patients and in individuals who have committed violent suicide (Asberg et al., 1976).

We can also assess serotonergic activity by measuring the response of hormones, such as prolactin, to serotonergic releasing agents, including

d,l-fenfluramine. Blunted prolactin responses are associated with suicide attempts in personality disorder patients. Further, the same lack of response is associated with impulsive aggression in borderline personality disorder and specifically in those aspects of borderline personality disorder that reflect impulsive aggression (Coccaro et al., 1989). Other imaging studies have shown decreased activation in the orbital and ventral medial prefrontal cortex in response to *d,l*-fenfuramine administration in impulsive-aggressive personality disorder patients (Siever et al., 1999).

The serotonin receptor subtypes have been carefully studied, and it has been shown that the various receptors have differing effects on impulsive aggression. Antagonists of (5-HT2A) receptors reduce impulsivity in animal models (Winstanley, 2004), and atypical neuroleptics with prominent 5-HT2A antagonism have antiaggressive efficacy in clinical populations (Krakowski et al., 2006). Agonists, on the other hand, reduce impulsivity at the (5-HT2C) receptor (Winstanley, 2004), suggesting that the two receptor subtypes may have complementary roles in the regulation of aggression consistent with their reciprocal modulation in animal model systems (Bubar & Cunningham, 2006).

Positron emission tomography imaging studies have been used to study aspects of the serotonergic system, including the serotonin transporter and the 5-HT2A receptor and their relation to impulsive aggression in personality disorders. Serotonin transporter activity was found to be reduced in the cingulate cortex in aggressive patients diagnosed with a personality disorder relative to comparison subjects (Frankle et al., 2005). Receptor binding of 5-HT2A,

which can be measured by [¹¹C]MDL100907 or altanserin, is significantly increased in physically aggressive patients with personality disorders (Siever et al., 2006) and in female borderline personality disorder patients (Soloff et al., 2007), consistent with postmortem (Arango et al., 1990) and platelet studies (Pandey et al., 1995), suggesting increased 5-HT_{2A} receptor binding associated with suicide. These studies are consistent with 5-HT_{2A} antagonist reducing impulsivity, which may have further clinical implications (Meyer et al., 2008).

Catecholamines

We are also learning more about the role of catecholamines in impulsive aggression related to personality disorders. Specifically, dopamine and norepinephrine may enhance the likelihood of other-directed aggression. Elevated dopamine levels have been seen in aggressive behavior (De Almeida et al., 2005). Whereas with serotonin a lack of transmitter has been associated with impulsive aggression, a widely accepted theory is that impulsive aggression in personality disorders is related to an excess of dopamine and other catecholamines. Further proof for this theory may be seen in the decrease in impulsive aggression found in response to dopamine-blocking agents. Clinically, haloperidol and other antipsychotics, whose presumed mechanism of action is related to their ability to block the dopamine receptor, have been used successfully to treat aggression in various pathologies, from Alzheimer's disease to schizophrenia (Krakowski et al., 2008; Lonergan et al., 2002). In terms of personality disorders, there has also been some successful treatment of impulsive aggression with dopamine blockade. An Italian group reported a significant decrease in impulsive aggression in 15 patients with borderline personality disorder treated with risperidone (Rocca et al., 2002). We will further discuss the use of medication in the treatment of impulsive aggression in the Treatment section.

Glutamate/GABA

Other neurotransmitters have been theorized to play a role in impulsive aggression. Imbalance in glutamatergic/gabaminergic activity may contribute to hyperactivity of subcortical limbic regions (Hrabovszky et al., 2005). Gamma-aminobutyric acid (GABA) is the chief inhibitory neurotransmitter of the central nervous system, and GABA agonism tends to have an overall calming effect on healthy individuals. Gamma-aminobutyric acid type A (GABA[A]) receptor modulators may enhance aggression (Fish et al., 2002), and tiagabine,

a GABA reuptake inhibitor, decreases aggression, possibly by suppressing reactions to aversive stimuli (Lieving et al., 2008). Other antiepileptic medications that act agonistically have been shown to decrease impulsive aggression in personality disorders (Bellino et al., 2008; Hollander et al., 2005). Thus, there are indications that reduced activity at GABA receptors likely contributes to some of the aggression seen in personality disorders.

However, there have been reports that elevated levels of GABA in the CSF are correlated with impulsivity in individuals with personality disorders (Lee et al., 2009). Further, benzodiazepines that act agonistically on the GABA receptor have been shown to increase impulsive behavior in borderline personality disorder. This phenomenon has often been attributed to the potentially disinhibiting affect of benzodiazepines (Cowdry & Gardner, 1988). The fact that GABA levels have been both positively and negatively correlated with impulsive aggression in personality disorders points to a complex interaction between the neurotransmitter and the behavior. There is likely a regional effect that is related to this seemingly paradoxical occurrence. As well, it may be related to receptor subtype. Regardless, there is an obvious need for continued investigation of the role GABA plays in impulsive aggression in personality disorders.

While GABA is the chief inhibitory neurotransmitter of the brain, glutamate is the most abundant excitatory neurotransmitter. Glutamatergic enhancement has been shown to increase aggression in mice (Lumley et al., 2004), and higher levels of glutamine have been found in aggressive bulls than in nonaggressive bulls (Munoz-Blanco & Porras Castillo, 1987). These facts have led researchers to theorize that raising the glutamate level is the imbalance in the GABA/glutamatergic systems that contributes to aggression. While there are some medications that modulate the glutamate system, like riluzole for amyotrophic lateral sclerosis or memantine for Alzheimer's disease, few studies have looked at the role of glutamate manipulation in personality disorders. Given the theorized role of glutamate in impulsive aggression, this may be an area of future research.

Over the past several decades, the market has been flooded with new compounds that can be used to effectively treat patients with mental illness by correcting presumed abnormalities in neurotransmitter levels or function. Clinicians have used these medications safely in a variety of conditions from schizophrenia to social phobia. Thus, identification

of specific neurotransmitter systems or second messenger disturbances that underlie impulsive aggression in personality disorders can play an important role in the development of pharmacological approaches to treatment for this pathology.

Neuromodulators

Vasopressin

Besides studying neurotransmitters, researchers have explored the role other neuromodulators may play in the impulsive aggression within personality disorders. Vasopressin is implicated in aggression. A positive correlation has been reported between CSF vasopressin concentrations and the life history of aggression in personality disorder patients (Coccaro et al., 1998) even when controlling for serotonergic activity. This parallels findings in animal studies in which higher densities of anterior hypothalamic neurons containing vasopressin were associated with increased aggressive behavior (Gobrogge et al., 2007). Vasopressin 1b receptor knockout mice conversely show decreases in aggression (Wersinger et al., 2007). Further, increases in serotonergic activity can reduce concentrations of vasopressin centrally (Ferris & Delville, 1994).

Oxytocin

Oxytocin has been well studied in terms of its relation to affiliative behavior and trust (Kosfeld et al., 2005; Winslow & Insel, 2000; Zak et al., 2007). Although its absence may not lead directly to impulsive aggression, earlier in the chapter we stated that significant pathology in one sphere of behavior may contribute to impulsive aggression. If someone is extremely distrustful or even paranoid, he or she may be more likely to act aggressively. It has been theorized that early traumatic events, like those seen in borderline personality disorder, may lead to decreased oxytocin levels (Bartz & Hollander, 2006; Teicher et al., 2002). Further, oxytocin reduces amygdala activity in humans (Kirsch et al., 2005). Thus, deficits in oxytocin can contribute to the hostility, fear, and mistrust seen in persons with personality disorders that may provide the preconditions for the emergence of aggression. We have, in fact, seen this in animals; oxytocin knockout mice display exaggerated aggressive behavior (Ragnauth et al., 2005).

Opioids

Opiates have been related to aggression, particularly self-directed aggression. Reduced CSF endogenous opioid concentrations have been associated with self-injurious behaviors in patients with borderline

personality disorder (Sher & Stanley, 2009), consistent with the clinical observation of the propensity of borderline personality disorder patients to seek relief with opiate pain medications. Reduced opioids may be associated with increased separation/abandonment distress and rejection sensitivity that may heighten the likelihood of aggressive behavior (Macdonald & Leary, 2005; Panksepp, 2003). Reduced presynaptic opiate activity may upregulate postsynaptic μ -opioid receptors, and thus dramatic relief of pain may result when opiates are released in the context of the self-injurious behavior seen in borderline personality disorder.

Testosterone

While numerous reports have suggested correlations between plasma testosterone concentration and aggression, these relationships have not always been replicated and have been extensively reviewed (Archer, 1991). However, some correlations have been found between testosterone levels and impulsive aggression in personality disorders. High concentrations of testosterone have been reported in populations diagnosed with personality disorders (Coccaro et al., 2007a). Further, one Scandinavian study found that personality-disordered criminals with multiple offenses had higher serum testosterone levels than other criminal controls (Rasanen et al., 1999). On the other hand, cortisol concentrations have generally been found to be low in individuals with high aggression, including antisocial criminal offenders (Coccaro & Siever, 1995).

Thus, from serotonin receptors to opioid analogues, researchers have taken some significant steps toward a better understanding of the molecules and proteins that underlie impulsive and aggressive behavior. We know from molecular biology that these substrates are the products of enzymatic reactions catalyzed by proteins encoded from genetic material. Therefore, the next logical question is: What role do the genes related to these neurotransmitters and receptors play in this symptom cluster within personality disorders? This issue will be the focus of the next section.

Genetics

Within the scientific community, it is fairly well accepted that multiple genes, in combination with other factors, contribute to the development of a personality disorder (Gabbard, 2005). Some of the genes that are thought to be related to impulsive aggression are also thought to be related to the development of personality disorders. For example,

variations within the gene that codes for monoamine oxidase A (MAO-A) have been associated with aggression in rhesus monkeys as well as in human subjects with borderline personality disorder. Therefore, we will begin our discussion of the genetics of impulsive aggression in personality disorders with a brief survey of what is currently known about the genes involved in impulsive aggression as a symptom across pathologies; then we will examine the speculative role of similar genes in personality disorders.

A number of candidate genes have been explored in relation to impulsive aggression or disorders characterized by high aggression. These include serotonin-related genes, catecholamine-related genes, and other neuromodulator-related genes. For example, the 5-HT2A TYR 452 allele has been associated with childhood-onset aggression (Mik et al., 2007). An allele for low MAO-A activity has been reported to be associated with aggression in a Dutch pedigree (Brunner et al., 1993). Subjects with low activity of the MAO-A gene display more aggression and exhibit significant volume reductions in the bilateral amygdala, anterior cingulate cortex, and subgenual anterior cingulate cortex (Meyer-Lindenberg et al., 2006). Activity of MAO-A interacts with rearing to influence aggressive behavior in rhesus monkeys (Newman et al., 2005) and humans (Caspi et al., 2002). Serotonin transporter (5-HTT) polymorphisms are also associated with aggression in some populations (Davidge et al., 2004; Patkar et al., 2002). The tryptophan hydroxylase-1 (TPH1) allele has been associated with aggression in some (New et al., 1998; Nielsen et al., 1994) but not all (Gelernter et al., 1998) studies.

In terms of personality disorders, a number of genes in the serotonergic and catecholaminergic systems, and their relation to the development of a subsequent personality disorder, have been explored. For example, DRD2 and DRD4 gene variants interact to predict adolescent conduct disorder and adolescent antisocial behavior (Beaver et al., 2007; Congdon et al., 2008). These allelic variances may interact with the environment so that, for example, individuals with low MAO-A activity, when exposed to childhood maltreatment, are more likely to exhibit antisocial behavior as adults, while severe childhood maltreatment has a more modest effect on individuals with high MAO-A activity (Caspi et al., 2002). Further, an allele of MAO-A has also been associated with borderline personality disorder (Ni et al., 2007). Differences in allele frequency of a single nucleotide polymorphism, rs165599, in the

catecholamine-*O*-methyltransferase gene (Schifman et al., 2002) have been associated with increased physical aggression and aggressive responses on the PSAP in patients with personality disorders (Flory, 2007). Alleles in the TPH2 gene, which controls serotonin synthesis in the brain (Zhang et al., 2004; Zhou et al., 2005), have been associated with aggression and borderline personality disorder in personality disorder patients (unpublished data of Siever et al., 2007; unpublished data of Kennedy et al., 2007) and are related to emotional instability in healthy volunteers (Gutknecht et al., 2007).

From this survey, it is clear that we have gained some clues to the potential genetic underpinnings of impulsive aggression within personality disorders. Many of the genetic aberrations of neurotransmitter production or receptors found within personality disorders can be predicted based on the previously described dysfunction within these systems. However, these findings are just the tip of the iceberg. As science continues to work on the genetics of personality disorders, undoubtedly other genes and genetic mutations will be linked to these symptom clusters.

Treatment

As described earlier, patients who have a tendency to behave in an impulsive and/or aggressive manner have higher rates of adverse outcomes (i.e., suicide attempts, drug abuse, violent crime). For this reason, it is imperative to identify these individuals and treat them appropriately. However, to complicate the picture, patients with personality disorders who act impulsively and aggressively are extremely hard to treat. There is no clear Food and Drug Administration (FDA)-approved treatment for these patients. Many of their symptoms are refractory to conventional psychopharmacological and psychotherapeutic treatments. They are often noncompliant with prescribed treatments. Nonpsychiatric clinicians often misdiagnose these patients. Further, they often lack access to appropriate care. Despite these hurdles to treatment, there have been efforts to produce successful treatment strategies. We will review some of the more important scientific contributions to the treatment of impulsivity and aggressiveness within personality disorders.

Neurobiological Implications for the Pharmacotherapy of Impulsive Aggression in Personality Disorders

While there is no clear FDA-approved medication for use in treating impulsive aggression in personality

disorders, there have been studies that seem promising for several agents. When viewed in the biological framework previously laid out in this chapter, much of the success of these agents actually seems fairly intuitive. In terms of practical treatment, impulsive aggression may be treated similarly across pathologies; however, in this chapter, we will focus on the treatment within personality disorders.

Antidepressants

From our discussion of the serotonin system and its relation to impulsive aggression, one could theorize that medications that raise the overall serotonin levels, like SSRIs decrease impulsive aggression. Indeed, this has been shown in several studies. Open-label and randomized trials have shown that both fluoxetine and sertraline treatment have led to a significant decrease in impulsivity and aggressive behavior in personality disorders (Coccaro et al., 1990; Kavoussi et al., 1994). We also have mentioned that this population has a high incidence of suicide (Soloff et al., 2005). Therefore, when targeting this symptom cluster, an agent with low lethality in overdose is attractive to both researchers and clinicians. As a class, the SSRIs have been reported to be relatively safe in overdose (Barbey & Roose, 1998), which makes them desirable treatment options. Further, SSRIs are well tolerated by patients, and many of these medications have become quite affordable secondary to patent expiration.

The use of older agents, which also act on the serotonin system, for the treatment of impulsive aggression has also been investigated. Tricyclic antidepressants that raise the serotonin level have been reported to have a positive effect on impulsive aggression; however, due to their lethality in overdose, they are not a preferred treatment option (Bellino et al., 2008). Similarly, MAO inhibitors, like phenelzine, have also shown some evidence of decreasing impulsive aggression in personality disorders (Cornelius et al., 1993); however, their significant side effect profile and difficulty of administration make them less favorable than the SSRIs. Therefore, at this point, SSRIs, like fluoxetine and sertraline, which work on the serotonin system, appear to have the most support in the literature, in terms of efficacy and safety, for use in the treatment of impulsive aggression.

Anticonvulsants

In the model we have set forth, serotonin is responsible for enhancing the “frontal brakes” on emotions

derived from limbic irritability. Another target for controlling impulsive aggression is the aggressive drive itself. Medications like valproic acid, carbamazepine, and tiagabine, which have classically been used to treat seizures and act through the GABA system, are thought to have this capability. It has been theorized that they are able to stabilize limbic irritability. Studies have shown that these medications are effective in reducing impulsive aggression in personality disorders. Divalporex has been found to reduce aggressive behavior in patients in patients with Cluster B personality disorders in several clinical trials (Hollander et al., 2003; Kavoussi & Coccaro, 1998). Carbamazepine acts on voltage-gated sodium channels; however, some of its anticonvulsant properties also may be derived from its effects on the GABA system, which may explain its efficacy in personality disorders (Granger et al., 1995). One review found that patients with borderline personality disorder who had a history of extensive behavioral dyscontrol responded best to carbamazepine in terms of reduced physically violence (Hori, 1998). Tiagabine is an anticonvulsant medication that blocks the reuptake of GABA. In one study of 10 convicted criminals, tiagabine administration led to significant improvement in PSAP scores (Lieving et al., 2008).

An antiepileptic medication that has shown promise in the treatment of impulsive aggression in personality disorders is lamotrigine. As it is a relatively new medication, its exact mechanism of action is less well understood, and it may derive its effect less from GABA enhancement than from overall limbic stabilization. One double-blind, placebo-controlled trial in 24 women with borderline personality disorder showed a significant improvement in aggressive symptoms in the lamotrigine group (Tritt, et al., 2005). Further, it has been FDA approved for the treatment of bipolar depression. However, lamotrigine’s clinical use is limited by its potential to induce a life-threatening rash, Stevens-Johnson syndrome (Hirsch et al., 2004). The other antiepileptic medications previously described each have the potential for significant side effects (Walia et al., 2004). Valproic acid can cause hair loss, weight gain, agranulocytosis, hepatotoxicity, and pancreatic toxicity. Among other serious side effects, carbamazepine has been associated with aplastic anemia (Handoko et al., 2006). Thus, although these medications do show some efficacy in treating impulsive aggression, their use should be tailored to the appropriate patient due to their side effects.

Antipsychotics

We discussed the association between elevated dopamine levels and impulsive aggression within personality disorders. Some of the best-studied psychotropic medications are those that block dopamine. Researchers have investigated the potential use of these agents to target this symptom cluster. Early studies focused on the typical antipsychotics. One trial found haloperidol to be superior to amitriptyline and placebo for treating impulsivity in borderline personality disorder (Soloff et al., 1986). Another trial that used flupentixol, a long-acting depot formulation of a typical antipsychotic, found a decrease in suicidal behaviors in personality disorder patients. However, although these medications showed some efficacy in studies, they were not tolerated well by many patients due their significant extrapyramidal side effects. This poor tolerance leads to noncompliance and high treatment dropout rates (Cornelius, et al., 1993; Goldberg et al., 1986).

More recently, researchers have been exploring the efficacy of the atypical antipsychotics for the treatment of impulsive aggression. In addition to having a decreased risk of extrapyramidal side effects, many of these agents are unique in their antagonistic activity of the 5-HT2A receptors as well as the D2 receptors. Given the association between decreased 5-HT2A activity and impulsive aggression, scientists have theorized that this expanded receptor-blocking profile conveys added efficacy in the treatment of this symptom cluster. Several studies with risperidone and olanzapine have been successful in treating patients with personality disorders. One study of 60 outpatients found that when olanzapine was added to the treatment of patients with borderline personality disorder, there was a significant decrease in aggressive behavior (Soler et al., 2005). Similarly, an 8-week open trial of risperidone showed improvement in this symptom cluster (Rocca et al., 2002). As the literature continues to report positive outcomes with medications from this class, their use as first-line agents in the treatment of impulsive aggression in personality disorders appears to be promising.

Opioid Antagonists

Opioid antagonists have also been proven to decrease impulsive behavior in patients with personality disorders. Several studies have shown that the use of naltrexone, an agent that blocks the opioid receptor, decreases self-injurious and parasuicidal behaviors in patients with borderline personality disorder (McGee, 1997; Roth et al., 1996; Sonne et al., 1996).

In our discussion of the opiate system, we described how the act of cutting or self-mutilating likely results in the release of endogenous opioids in personality-disordered patients. Therefore, blocking this response may lead to extinction of these previously enforcing behaviors. However, although opioid antagonists may lead to a decrease in impulsive-aggressive behavior, they do not correct the underlying defect in the opiate system of reduced endogenous opioid concentrations.

While an exhaustive review of the current literature on the pharmacology of impulsive aggression is beyond the scope of this chapter, this section has provided an overview of the results of some pharmaceutical treatment strategies that were in line with the framework of the biology of impulsive aggression in personality disorders we have described. With a basic understanding of the biological aspects of this behavior, researchers and clinicians have attempted to use agents to produce a predictable change in patients' biochemistry and behavior. With a better understanding of the neurobiology of impulsive aggression, scientists can create novel therapeutic agents to target these symptoms.

Nonpharmacological Treatment

We have discussed some of the success in the treatment of impulsive aggression seen with the use of pharmacological agents; however, there has also been notable success with the use of other treatment modalities. An in-depth analysis of these alternatives is not in line with the remainder of the chapter, but any discussion of the treatment of this symptom cluster should mention some of these options.

Two psychotherapeutic techniques, dialectical behavior therapy (DBT) and cognitive behavior therapy (CBT), have been shown to be effective in the treatment of impulsive aggression. Although DBT has roots in traditional CBT, it can be distinguished by its emphasis on alternative coping mechanisms and skills training to replace self-injurious behaviors, which has a direct effect on reducing impulsive behavior. Several controlled studies have shown that DBT can decrease parasuicidal behavior in patients (Bohus et al., 2004; Linehan, 2006; Verheul et al., 2003). Recently, Linehan randomized 101 women with borderline personality disorder to either DBT or treatment by experts and found that the DBT group was half as likely to attempt suicide and had fewer emergency room visits (Linehan, 2006).

Cognitive behavior therapy has been used to treat symptoms like emotional dyscontrol and

stress-related paranoid ideation in personality disorders. In one study of patients with antisocial personality disorder and cocaine abuse, Messina found that the use of CBT led to a significant decrease in drug use (Messina, 2003). Beyond individual psychotherapy, other types of psychosocial interventions, like partial hospitalization or intensive group therapies, may also play a role in effectively treating this symptom cluster. It is likely that the most effective treatment strategy for an individual with these symptoms will be some combination of psychopharmacological, psychotherapeutic, and psychosocial interventions (Paris, 2009). Thus, when treating impulsive aggression in personality disorders, a clinician should consider both pharmacological and nonpharmacological treatment modalities.

Future Research

While we have discussed a great deal of information that is known about the biology of impulsivity and aggressivity in personality disorders, gaps in our knowledge still remain. There are significant deficiencies in our understanding of the exact neuroanatomical circuitry, knowledge of how to predict impulsive aggression, and selection of the optimal treatment strategy. Future research in these areas should work to fill in these gaps.

A more detailed understanding of the interconnections of impulsive aggression with global cognition should be sought. We have described how extreme fear, or paranoia, makes an individual more likely to behave in aggressive manor. The role of the amygdala in fear (LeDoux, 2007) is well studied and the orbitofrontal cortex, as we have described, plays a modulatory role in regulating negative emotion; however, the connection between the amygdala and impulsive aggression is not well delineated. A better understanding of the commonalities and differences between the regions and circuits involved in these different paradigms, which have many overlapping and synergistic clinical features, needs to be better defined. We may see more DTI or other neuroimaging studies analyzing the connectivity between these systems in the years to come.

We have described some of the paradigms that are used to predict impulsive aggression in patients. These paradigms are not perfect, and clinicians are often unable to predict accurately which individuals will act in this manner (Powell et al., 2000). Efforts are needed to create paradigms and measures that are better able to reliably identify patients at risk for this type of behavior.

We have discussed some of the available treatment options for impulsive aggression in personality disorders; however, there is still no FDA-approved medication or optimal treatment regimen. Further research should be conducted on novel pharmacological agents targeting this symptom cluster. We have described the role of the serotonin system in impulsive aggression and how 5-HT2A antagonism reduces this behavior in animal models, but such antagonists have yet to be studied in clinical populations. Pilot data suggest reductions in 5-HT2A binding with treatment with the SSRI fluoxetine, which may parallel reductions in aggression symptoms (unpublished data of Siever et al., 2007) implicating the increased 5-HT2A receptor sensitivity associated with aggression and its reduction with efficacious treatment.

In our discussion of opioid antagonists, we alluded to the fact that their successful reduction in parasuicidal behavior still leaves the patient with an overall deficit in endogenous opioids. Buprenorphine, an agent that is both a partial agonist and an antagonist of the opiate system, may be able to address this issue. Studies should be conducted on the efficacy of buprenorphine and other agents with opiate agonistic qualities in treating this symptom cluster.

Oxytocin is another agent that warrants further investigation. We have described how decreased oxytocin levels lead to distrust and aggression. Treatment with oxytocin may foster improved socialization and have a positive impact on impulsive aggression. In sum, we have made significant progress in our understanding of the mechanism and treatment of impulsive aggression in personality disorders; however, there remains a need for continued research in this area.

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Impulsivity and Co-occurring Psychiatric Disorders

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Abstract

Dual diagnosis, the co-occurrence of substance abuse and another psychiatric disorder, is common. There is evidence to suggest that impulsivity may serve as a common substrate for these comorbidities, despite behavioral and biochemical differences between disorders. This chapter describes common neurobiological and behavioral findings between individuals with high impulsivity and those with dual diagnosis. Specifically, we focus on the co-occurrence of substance abuse with schizophrenia, eating disorders, attention-deficit/hyperactivity disorder, antisocial and borderline personality disorders, and bipolar disorder. For each type of dual diagnosis, we review literature that provides empirical evidence for the presence of impulsivity and treatment recommendations. In this context, we propose a “bottom-up” conceptualization, in which clusters of co-occurring phenotypes are used to formulate diagnostic and clinical plans; such an approach may produce more homogeneous diagnostic groups than exist in the current system. Further, a bottom-up approach may reveal that dual-diagnosis disorders represent distinct groups that share a common factor of impulsivity.

Keywords: anorexia, antisocial personality disorder, attention-deficit/hyperactivity disorder, bipolar disorder, borderline personality disorder, bulimia, comorbidity, dual diagnosis, eating disorders, impulsivity, schizophrenia, substance abuse, substance use

In most psychiatric conditions, comorbidity is the norm rather than the exception; of the 46.4% of participants in the National Comorbidity Survey Replication (NCS-R) that met DSM-IV criteria for psychiatric illness over their lifetime, nearly 60% met criteria for two or more disorders (Kessler et al., 2005). Achenbach and Rescorla (2006) suggest at least three possible explanations for this phenomenon: (1) one disorder is the result of the other, (2) they both stem from the same risk factor, or (3) the disorders are not truly separate entities “but are manifestations of the same underlying condition” (p. 152). This final explanation leaves open the possibility that comorbidity is an artifact of DSM nosology; two disorders may occur together more often than expected by chance not only because

they are linked by shared etiology, but also because they are reflections of a common functional impairment.

To make sense of the high rate of co-occurring psychiatric diagnoses, one must consider the categorical structure of the DSM and similar manuals. The authors of the DSM have applied a nosological model of diagnosis, or a “top-down” classification scheme (Achenbach & Rescorla, 2006), which begins with a concept of a disorder and leads to criteria to fit that category. This method stands in contrast to a dimensional “bottom-up” strategy, which begins with quantitative data for functional problems and then applies statistics to elucidate patterns of co-occurrence among functional impairments. One possible application of the bottom-up approach

is to analyze clusters of symptoms from the NCS data to create an empirically supported diagnostic classification system (Insel & Fenton, 2005). Others have posited unifying personality traits that underlie commonly observed co-occurring behaviors in an attempt to simplify conceptions of patients with overlapping symptomology (e.g., Krueger, 1999). In this context, it is more useful to think of mental illness arising from a constellation of problem behaviors rather than as a set of discrete diagnoses.

If one were to adapt a bottom-up explanation for comorbidity, the next challenge would be to identify the underlying factors that unite seemingly disparate impairments. One possible factor that has been suggested to explain the prevalence of certain comorbidities is impulsivity. As Brewer and Potenza (2008) have summarized, "impulsivity is a complex, multifaceted construct" (p. 65) that, for example, has been divided into four components—urgency, lack of premeditation, lack of perseverance, and sensation seeking (Whiteside & Lynam, 2001)—or three elements—in the Barratt Impulsivity Scale: cognition, motor, and planning (Patton et al., 1995); in the Eysenck impulsivity scale: venturesomeness, impulsiveness, and empathy domains (Eysenck & Eysenck, 1978)—depending on the theoretical orientation of the researchers. As such, impulsivity seems a likely candidate to unify certain patterns of maladaptive behavior, in large part because it encompasses many key neurocognitive pathways.

In particular, substance abuse and concomitant psychiatric illness (often referred to as *dual diagnosis*) have received a great deal of attention in the literature, in large part because of the clinical implications for treatment (Mueser et al., 1998). Although impulsive behaviors are included among the diagnostic criteria for substance use disorders (SUDs), impulsivity is not cited consistently as an underlying feature of dual diagnosis. Current theories that seek to explain the prevalence of dual diagnosis include supersensitivity to drugs of abuse (Mueser et al., 1998), underlying personality disorders (Kokkevi et al., 1998), and self-medication for primary psychiatric symptoms in some patients (Goswami et al., 2004). Without disregarding these hypotheses, impulsivity may exist as a complementary or alternative explanation for dual diagnosis. Given its role in many forms of psychiatric illness, it is likely that impulsivity underlies a number of dual diagnoses and frequently may help to explain the co-occurrence of psychiatric illness with substance abuse. We begin by discussing the role of impulsivity

in SUDs and follow with a discussion of the putative role impulsivity may play in disorders that often co-occur with SUDs.

SUDs and Impulsivity

Initiation, continuation, and addiction to substances represent the stages of substance abuse and may be related to impulsivity to different degrees. Genetic studies reveal that elevated impulsivity and risk taking are major risk factors for substance use initiation but may have less impact on continuation and addiction (Kreek et al., 2005). These latter phases may be influenced less by impulsivity and more by dopaminergic (Hyman et al., 2006) and glutamatergic (Kalivas & Volkow, 2005) neurocircuitry involved in reward-related learning. Therefore, we will discuss the role of impulsivity in SUD as it relates to the initiation of these disorders, and focus less on addiction and relapse.

Results of the NCS-R show that 14.6% of respondents met criteria for a SUD in their lifetime (Kessler et al., 2005). Patients with SUDs and other psychiatric illnesses often require intensive treatment, have poor physical and psychiatric outcomes, and have low adaptive skills, all of which challenge treatment effectiveness (Chambers et al., 2003; Hawkins, 2009). A detailed description of current theories linking SUDs and impulsivity can be found elsewhere (see, for instance, the chapter in this volume by Stein and Harvey for a review of impulsivity and behavioral addictions); here we emphasize the bidirectional relationship between impulsivity and substance abuse.

First, a biological predisposition to impulsivity can potentially be a risk factor for substance abuse. Addiction and impulsivity are thought to result from dysregulated function of corticolimbic circuitry, with a pro-motivational drive contributed by dopaminergic (DA-ergic) projections from the ventral tegmental area (VTA) to the nucleus accumbens (NAcc) and involving GABAergic and glutamatergic processes (Everitt & Wolf, 2002; Koob et al., 1998). Relatively diminished frontal cortical control over pro-motivational limbic circuitry has been proposed as leading to impulsive behaviors and substance abuse and dependence (Chambers et al., 2003; Everitt & Robbins, 2005; Jentsch & Taylor, 1999). Specifically, ventral prefrontal cortical-to-ventral-striatal circuits have been implicated in seemingly impulsive thoughts and behaviors, with more dorsal corticostriatal circuits implicated in habitual or compulsive behaviors (Brewer & Potenza, 2008; Everitt & Robbins, 2005; Torregrossa

et al., 2008). Other brain regions implicated in motivated behaviors and the development of SUDs include the amygdala and related circuits that provide affective information, the hippocampus and related circuits that provide contextual memory information, and the hypothalamus and septum that provide homeostatic information related to thirst, hunger, and sex drive (Chambers et al., 2003; Field et al., 2005). In addition to dysregulation of DA, genetic polymorphisms in the serotonin transporter and monoamine oxidase A have also been linked to substance abuse and impulsive behavior (Verdejo-García et al., 2008). Findings from functional, genetic, and neurochemical studies of impulsivity and SUDs represent converging evidence of the role of impulsivity in the development of SUDs.

Behavioral data also suggest that patients with high impulsivity are prone to substance abuse. Research on the etiology of SUDs has focused on the role of craving and compulsion (O'Brien et al., 1998); studies have also supported impulsivity as a supplementary or alternative contributory factor in substance abuse. Neuropsychological research has provided evidence that both rash, spontaneous impulsiveness and impaired reward sensitivity may act as risk factors for substance abuse (Dawe & Loxton, 2004). As reviewed elsewhere (Moeller & Dougherty, 2002), strong evidence for such relationships come from studies of patients with diagnoses of attention-deficit/hyperactivity disorder (ADHD), conduct disorder (CD), and oppositional-defiant disorder (ODD), all three of which include aspects of impulsivity or related constructs in their diagnostic criteria (Kreek et al., 2005). Data indicate an association between comorbid ADHD-CD/ODD and later development of substance abuse, which suggests impulsivity may serve as a risk factor for SUDs; further, studying the relationship between childhood impulsivity and SUDs later in life may help to produce biological explanations of the role of impulsivity and SUD (Ivanov et al., 2007). On a molecular level, the same genetic polymorphisms that may predispose to impulsivity (e.g., genes that code for serotonergic, dopaminergic, and noradrenergic receptors) may also predispose to substance abuse (Moeller & Dougherty, 2002).

Second, substance use and abuse may exacerbate impulsivity. There is evidence that alcohol, cocaine, and methylene-dioxymethamphetamine (MDMA; Milstein et al., 2008), as well as nicotine (Kumari & Postma, 2005), may generate or exacerbate attentional deficits and impulsive behaviors in humans. With these two potential relationships in mind, one

can imagine a mechanism in which a predisposition to impulsivity acts as a risk factor for substance abuse, and the resultant substance use and abuse leads to an increase in impulsivity (or an impairment in decision making that may manifestly appear as impulsivity). In reviewing the role of impulsivity in diagnoses often paired with SUDs, we will focus on this mechanism and suggest possible points of behavioral and pharmacological intervention that may target the problematic common functional impairment of impulsivity.

Implications of Impulsivity in Subtypes of Dual Diagnosis

As a common contributor to dual diagnoses, impulsivity may manifest itself differently because of its interaction with specific genetic, social, and environmental factors. Therefore, dual diagnosis does not exist as a single phenomenological entity; patients are categorized by their predominant symptoms (by primary psychiatric diagnoses) and concomitant substance abuse. Here we review evidence for a role for impulsivity in clinically relevant subtypes of dual diagnoses, with an emphasis in each case on the semipermeable boundary between primary psychiatric diagnosis and substance abuse.

Schizophrenia and Substance Abuse

The prevalence of co-occurring substance abuse in schizophrenia is second only to personality disorders (Regier et al., 1990), and poses formidable treatment issues because of the severe effects of combined schizophrenia and substance abuse (Barrowclough et al., 2007; Kavanagh et al., 2002). The most common drugs of abuse include nicotine, alcohol, and cannabis (see Volkow, 2009, for a summary of prevalence rates in previous studies). Among the theories used to explain this type of dual diagnosis, the concept of self-medication, or negative reinforcement, has received support; according to this theory, for patients with schizophrenia, drugs of abuse act to reduce the side effects of medication or of disease symptoms (for review, see Blanchard et al., 2000). In addition, evidence from epidemiological studies indicates that cannabis use in adolescence may be a risk factor for adult-onset schizophrenia; however, such reports are hampered by methodological issues such as reliance on self-reports of drug use (Arseneault et al., 2004; Coulston et al., 2007) as well as few systematic details on whether or not prodromal symptoms of schizophrenia either predated or emerged at the same time as marijuana use (Hall, 2006). More recent thought has led to a positive

reinforcement model, which views substance abuse as a primary symptom of the psychiatric disease. In this model, the same corticobasalganglionic dysregulation that is thought to underlie the negative and positive symptoms of schizophrenia may also act as a risk factor for substance abuse.

The main evidence for substance abuse as a primary symptom of schizophrenia comes from comparing the neural circuitry of schizophrenia and substance abuse. Chambers, Krystal, and Self (2001) present a model that posits altered mesolimbic DA reward circuitry at the intersection of schizophrenia and substance abuse; namely, dysregulated DA-ergic and glutamatergic afferent signaling to the NAcc from the prefrontal cortex (PFC), VTA, and hippocampus results in a chronic decrease in inhibitory GABA-ergic signaling from the NAcc. In the context of environmental moderators, this mechanism may lead to impulsive behaviors characteristic of SUDs and positive symptoms of schizophrenia.

Behavioral studies also lend support to the theory of substance abuse as a primary feature in schizophrenia. Patients with substance abuse and schizophrenia report higher levels of impulsivity, sensation seeking (Dervaux et al., 2001), and suicidality (Gutfayand et al., 2001) than non-substance-abusing cohorts. Studies of male patients with schizophrenia have found an association between current alcohol (Kim, et al., 2007) and caffeine and nicotine (Van Ammers et al., 1997) use and novelty seeking. These studies are hampered by small sample sizes and reliance on retrospective self-report of drug use. With respect to *nonsubstance addictions*, a recent study found that in a sample of 337 individuals with schizophrenia or schizoaffective disorder, pathological gambling was acknowledged by 10% of patients and gambling problems by an additional 10% (Desai & Potenza, 2009). It is possible that abnormalities in decision making (e.g., as assessed by performance on gambling tasks) may contribute to this co-occurrence, although this hypothesis requires formal testing (Yip et al., 2009). With respect to co-occurring schizophrenia and alcohol abuse, Kim et al. (2007) found that those individuals with alcohol abuse demonstrated higher novelty seeking than the non-alcohol-abusing cohort using the Temperament and Character Inventory (TCI; Cloninger, 1994). However, the groups did not differ significantly in the reward dependence, harm avoidance, or persistence subscales of the TCI. This finding may indicate that only certain expressions of impulsivity (in this case, novelty seeking) represent risk factors for substance abuse in schizophrenia.

Treatment of co-occurring schizophrenia and SUDs requires both psychopharmacological and behavioral approaches. A recent review of medication treatments for this population yielded only a few controlled studies; the remainder were case series and open trials (Wobrock & Soyka, 2009). Nevertheless, extant data point to the efficacy of second-generation antipsychotics (SGAs; clozapine, in particular) in reducing positive and—to a lesser extent—negative symptoms, as well as craving and drug consumption (Smelson et al., 2008; Wobrock & Soyka, 2009). Second-generation antipsychotics such as clozapine may reduce craving through modulation of noradrenergic and DA receptors, which, in turn, normalizes mesocorticolimbic brain circuitry that regulates impulse control and reward sensitivity (Noordsy & Green, 2003). Supplementary agents that target craving in schizophrenia include certain antidepressants (especially tricyclic antidepressants such as desipramine and imipramine); the opioid receptor antagonist naltrexone; and disulfiram, which prevents the breakdown of acetaldehyde, producing an aversive reaction to alcohol consumption (Wobrock & Soyka, 2009). Behavioral treatments that target impulsivity include combined motivational interviewing (MI; Miller & Rollnick, 2002) and cognitive-behavioral therapy (CBT; Beck et al., 1979) that help clients understand the role of substance abuse in their lifestyle, devise strategies to avoid and handle high-risk situations, and cope with cravings (Barrowclough et al., 2007). Common issues that arise in this dual diagnosis population include low motivation, cognitive limitations, low self-efficacy, and limited interpersonal skills, which may be addressed in flexible, goal-oriented approaches such as motivation-based dual diagnosis treatment and dual recovery therapy (Ziedonis et al., 2004).

Eating Disorders and Substance Abuse

Eating disorders, including anorexia nervosa [AN], bulimia nervosa [BN], and binge-eating disorder [BED], are associated with specific patterns of disordered consumption and elimination of food (APA, 2000). Lifetime prevalence of eating disorders in the NCS-R ranges from 0.6% (AN) to 2.8% (BED), and comorbidity with SUDs ranges from 23.3% (BED) to 36.8% (BN) (Hudson et al., 2007). A current understanding of the neurobiology of eating disorders lends support to the association between impulsivity and substance abuse. Brain imaging studies of patients with AN and BN have shown abnormal functioning of frontal and

temporal lobes both during illness and following recovery, which may impair decision making and impulse control (Kaye, 2008). In addition, abnormalities in 5-hydroxytryptamine (5-HT) and DA in both AN and BN have been linked to impulsive binging and purging behaviors (Fava et al., 1990; Kaye, 2008; Monteleone et al., 2000).

Neuropsychological and personality research has revealed nuanced differences in subtypes of eating disorders. For instance, greater rates of reward sensitivity and impulsiveness have been found in BN and BED compared to AN (Bushnell et al., 1996; Cassin & von Ranson, 2005; Engel et al., 2005; Fahy & Eisler, 1993; Rosval et al., 2006). In addition, BN and BED are more often associated with substance misuse than AN (Bulik et al., 2004; Gadalla & Piran, 2007; Holderness et al., 1994). Given these findings, some have suggested that impulsivity in BN and BED may be a risk factor for substance abuse.

In a review of the literature, Dawe and Loxton (2004) found evidence for an association between impulsivity (and weaker evidence for impaired reward sensitivity) and substance abuse in women with binging and purging behavior. These findings may reflect a subtype of women with BED, distinct from those with concomitant mood disorders. For instance, Peterson et al. (2005) found that women with BED and SUD were characterized by greater impulsivity and binge eating than those with mood disorders. Further, a meta-analysis of impulsivity in BN (Fischer et al., 2008) revealed four subtypes of impulsivity, with negative urgency ("the tendency to act rashly when experiencing a negative mood" [p. 1414]) having the highest effect size; this finding may help to explain why binging/purging and substance abuse may each be responses to distress. Impulsivity in BN or BED may also be associated with risky behaviors other than substance abuse; women with BN or BED also exhibit high rates of suicidality (Forcano et al., 2009) and self-injurious behaviors (Ahrén-Moonga et al., 2008).

There are few empirically supported treatments for patients with both eating disorders and drug and alcohol problems, and fewer that target impulsivity. Most treatment studies for eating disorders exclude those with co-occurring substance abuse (Coelho et al., 2007). Coelho et al. recommend treating both disorders together to avoid the exacerbation of one set of symptoms while treating the other; for instance, Katzman et al. (1991) describe a group of women with BN and opiate addiction whose eating disorder symptoms worsened during inpatient

detoxification. Cohelho et al. (2007) describe outpatient (Mitchell et al., 1997) and inpatient (Lacey, 1995) abstinence-based, CBT approaches that have demonstrated success in decreasing substance abuse and eating disorder symptoms. Evidence with pharmacological approaches shows that treatment that combines the opiate antagonist naltrexone and CBT may be effective in reducing eating disorder and substance abuse symptoms in adolescents (Conason & Sher, 2006) and adults (O'Malley et al., 2007). There is also evidence that fluoxetine combined with CBT may decrease severity of BN (Berkman et al., 2006), but this finding has not been assessed in dual diagnosis individuals. Future research into treatment for individuals with eating disorders and SUDs should involve larger sample sizes and consistent acquisition of substance abuse and eating disorder severity measures.

Attention-deficit/Hyperactivity Disorder and Substance Abuse

The DSM-IV divides attention-deficit/hyperactivity disorder (ADHD) into three subdiagnoses: predominantly inattentive type; predominantly hyperactive-impulsive (HI) type; and combined type. The first is characterized by forgetfulness, disorganization, carelessness, and lack of attention. The second, in contrast, is characterized by motoric hyperactivity and rash impulsivity (blurtng out answers, difficulty waiting for one's turn, and interrupting others; APA, 2000). Although estimates of prevalence vary, a recent population-based study based on caregiver reports revealed that 8.7% of children aged 8–15 met criteria for ADHD, over half (57.2%) of whom met criteria for the HI or combined type (Froehlich et al., 2007).

To meet diagnostic criteria for ADHD, one must manifest symptoms before age 7, a point that underscores the developmental implications (and complexity) of understanding the connection between ADHD and SUD. Further complicating this picture is the frequency with which CD and/or ODD co-occur with ADHD; nearly half of the children diagnosed with ADHD also are diagnosed with CD or ODD (Jensen et al., 1997; Szatmari et al., 1989), although adults are more likely to have "pure" ADHD (McGough et al., 2005). Like ADHD, CD and ODD represent externalizing disorders, characterized by impulsivity and impaired behavioral control. Based on patterns of disease symptoms in families and twins, some have suggested a distinct genetic subtype of ADHD/CD (Biederman et al., 1992; Thapar et al., 2001).

Substance abuse is common among those with ADHD, although numbers vary when other comorbidities are considered. Although those diagnosed with the HI type or ADHD with ODD/CD are necessarily or typically impulsive, it is not clear that substance abuse is more prevalent in these populations than in the inattentive subtype: While some have found that the HI type predicts substance abuse (Elkins et al., 2007), others have found that a diagnosis of inattentive type in childhood is a stronger predictor of substance abuse in adolescence than HI or the combined type (Molina & Pelham, 2003). Still others have found that only early CD symptoms, regardless of the ADHD diagnosis, are associated with later substance abuse (Lynskey & Fergusson, 1995). These data indicate that ADHD is heterogeneous (Faraone & Biederman, 1998) and may act in synchrony with CD to confer a risk of substance abuse (Flory & Lynam, 2003). Additionally, any association between ADHD and SUDs may be confounded by alternate subtypes. For example, based on reports of comorbidity from 15 years of research, Jenson et al. (1997) suggest alternates to the HI and inattentive subtypes, namely, an aggressive and anxious subtype.

The pharmacological treatment of ADHD provides evidence for a neurobiological link among ADHD, SUDs, and impulsivity. Studies of humans with ADHD and animal models of the disorder have underlined the role of catecholamine regulation of corticostriatal pathways, especially the role of DA (Brennan & Arnsten, 2008). This understanding has led to widespread use in the treatment of ADHD of psychostimulants, drugs that have been found to be effective in reducing impulsive and inattentive symptoms in children with ADHD only or ADHD with ODD/CD (Jensen et al., 2001). Although some studies have found that early psychostimulant use predicts adult substance abuse (Lambert & Hartsough, 1998), other studies with larger sample sizes have not replicated this connection in adolescents (Barkley et al., 2003) or in adults (Faraone et al., 2007). Further, early psychostimulant use in children with ADHD may confer protection against substance abuse later in life, perhaps by reducing maladaptive, socially isolating behaviors in childhood and thereby reducing the motivation for substance abuse later in life (Katusic et al., 2005; Wilens et al., 2003). Nonetheless, because of the potential for abuse of methylphenidate, Kollins (2008) suggests using instead long-acting medications such as atomoxetine or bupropion that may be just as effective in reducing impulsive behaviors

associated with co-occurring ADHD and SUD but carry less risk for abuse.

Taken together, these phenomenological and treatment studies support the theory that impulsivity may explain the co-occurrence of ADHD and SUDs based on at least two findings: (1) substance abuse rates are elevated in ADHD with comorbid CD, which is characterized in part by rash impulsivity; (2) use of amphetamines, which in part regulate DA-ergic reward and impulse-inhibition circuitry, is associated with decreased rates of substance abuse over time. Therefore, other behavioral and pharmacological treatments that target impulsivity in ADHD may also reduce the risk of later substance abuse.

Antisocial and Borderline Personality Disorders and Substance Abuse

Co-occurrence of Cluster B personality disorders and substance abuse is common; data from the NCS-R reveal that 40.5% of those with antisocial personality disorder (ASPD) and 38.2% of those with borderline personality disorder (BPD) also meet 12-month DSM-IV criteria for SUDs. Likewise, of those with SUDs in the past 12 months, 3.2% and 7.2% meet criteria for ASPD and BPD, respectively (Lenzenweger et al., 2007). Both disorders are also associated with high rates of criminality and violent behaviors, which pose not only a psychiatric but also a societal challenge (Crocker et al., 2005; Fonagy et al., 1997; Johnson et al., 2000). Individuals with personality disorders and SUDs “are less responsive to traditional treatment, require more intensive therapeutic interventions and have poorer long-term prognoses than individuals with other disorders” (Crouse et al., 2007, p. 309). Trait impulsivity is a feature of Cluster B personalities, as studies of individuals with BPD (Chapman et al., 2008; Soloff et al., 2003) and ASPD (Hesselbrock & Hesselbrock, 1992; Swann et al., 2009) have found elevated impulsivity compared to controls.

Although impulsivity is represented in the diagnostic criteria for BPD, it is only an optional diagnostic criterion for ASPD (APA, 2000). Nevertheless, impulsivity is associated with ASPD and co-occurring SUDs. In a meta-analysis of reports of ASPD and SUDs, Ruiz et al. (2008) mapped subcategories of the five-factor model (FFM) of personality (Neuroticism, Extraversion, Openness, Agreeableness, and Conscientiousness; Costa & McCrae, 1992) to the Urgency, Lack of Premeditation, Lack of Perseverance, and Sensation Seeking (UPPS) model (Whiteside & Lynam, 2001;

Whiteside et al., 2005); thus, FFM high impulsivity, low deliberation, low self-discipline, and high excitement seeking mapped to UPPS. Antisocial personality disorder, SUDs, and co-occurring ASPD and SUDs were all associated with elevated impulsivity as measured by these domains.

Impulsivity has also been implicated in co-occurring BPD with SUDs. Bornovalova et al. (2005) reviewed evidence for alternate models of co-occurring BPD and SUDs, including a synergistic relationship between genetic predisposition to impulsivity and environmental factors that lead to decreased endogenous serotonin that influences substance abuse in those with BPD. This developmental model for co-occurring BPD and SUDs is supported by similarities between BPD and another developmental disorder, ADHD (Davids & Gastpar, 2005; Philipsen, 2006); both disorders may involve a serotonergic component (Norden, 1989). Philipsen (2006) has extended the similarities between BPD and ADHD to suggest that these may be different manifestations of the same underlying disorder.

Although we have discussed BPD and ASPD as separate entities, there is a significant diagnostic overlap between the two (Becker et al., 2005). In fact, in a sample of individuals with BPD, Feske et al. (2006) found that the relationship between BPD and SUDs decreased when ASPD was added to the model, suggesting that ASPD may act as a moderator in the relationship between BPD and SUDs. Because of this overlap between BPD and ASPD, some studies of personality disorders and SUDs do not distinguish between the Cluster B disorders. For example, Dom et al. (2006) compared levels of impulsivity in adult alcoholics with and without Cluster B personality disorders using self-report (Barratt impulsiveness and sensation-seeking scales) and behavioral measures (go/no-go, Stroop color-word, and delay-discounting tasks); they found that alcoholics with Cluster B personality disorders reported significantly higher levels of impulsivity on all the self-report subscales and diminished response inhibition on the go/no-go task. However, from these results, it is not possible to distinguish between the influences of BPD and ASPD on impulsivity.

Empirically supported treatments for BPD and co-occurring BPD and SUDs provide evidence for the role impulsivity may play in Cluster B personality disorders. Dialectical behavioral therapy (DBT) is arguably the most widely applied form of behavioral treatment for BPD, and has been adapted for co-occurring BPD and SUDs (Linehan et al., 1999).

Dialectical behavioral therapy is designed as an intensive outpatient process that focuses on five core difficulties in patients with BPD: emotional, interpersonal, behavioral, cognitive, and self-dysregulation (Linehan, 1993). The cognitive domain includes impulsive, self-harming behaviors; clinicians working with patients with co-occurring BPD and SUDs identify maladaptive coping mechanisms such as alcohol and drug use, and encourage alternative thought patterns and behaviors to counteract their impulsive actions (Crouse et al., 2007). Dialectical behavior therapy has been shown to reduce self-harm and other impulsive behaviors; however, cognitive, behavioral, and psychodynamic modalities have also shown promising results (Paris, 2009). One psychodynamic technique, mentalization-based therapy (MBT), seeks to reduce impulsive behaviors by exploring “inconsistencies in the relationship patterns of patients with borderline personality disorder and identification of unconscious factors that interfere with the possibility of change” (Bateman & Fonagy, 1999, pp. 1568–1569); participants in MBT continued to show decreased levels of suicidality compared to a treatment-as-usual group 8 years after initial treatment (Bateman & Fonagy, 2008).

Evidence for effective pharmacotherapy of BPD and ASPD is minimal and often contradictory, due in large part to the heterogeneity of the disorders (Paris, 2009; Verheul & Herbrink, 2007; Verheul & Van Den Brink, 2004). Nonetheless, results of a meta-review of clinical trials in BPD suggest that antipsychotics may be effective in reducing impulsivity and aggression (Nosè et al., 2006), and more recent work has reviewed evidence that the atypical antipsychotic olanzapine may reduce impulsivity in BPD (Abraham & Calabrese, 2008; Bellino et al., 2008).

Bipolar Disorder and Substance Abuse

The DSM-IV (APA, 2000) divides bipolar disorder (BD) into two primary types, characterized by manic or mixed episodes (BD I) and depression with at least one hypomanic episode (BD II). Results of the NCS-R reveal that approximately 4.4% of respondents met DSM-IV criteria for BD I or II, 42.3% of whom also met criteria for SUDs (Merikangas et al., 2007). Adults with co-occurring BD and SUDs experience greater disease severity than adults with BD without SUDs, as measured by lifetime psychiatric hospitalizations (Cassidy et al., 2001).

Impulsivity is an important component of BD and moderates the co-occurrence of BD with SUD

sas well as suicidality (Dougherty et al., 2004; Swann et al., 2005), impulse-control disorders, obsessive-compulsive disorder, and aggressive behavior (Najt et al., 2007). Trait impulsivity is a feature of individuals with BD, regardless of mood severity (Peluso et al., 2007; Swann et al., 2003). Trait impulsivity appears elevated in individuals with co-occurring BD and SUDs compared to those with either disorder alone. Adults with co-occurring BD and SUDs exhibit greater impulsivity (Sublette et al., 2009; Swann et al., 2004) and more risk taking (Holmes et al., 2009) than individuals with BD alone, regardless of mood severity or stage (i.e., intra- or intermanic episode). Compared to adolescents with BD, those with co-occurring BD and SUD had greater rates of suicide attempts (Goldstein et al., 2005), a higher 12-month prevalence of engagement with the police, and, in females, a higher prevalence of pregnancy and abortions (Goldstein et al., 2008).

Given the high frequency of suicide attempts in individuals with co-occurring BD and SUDs, it is tempting to hypothesize that intoxication increases the risk of suicidality; however, research with this population suggests that the role of substance use is less than that of trait impulsivity (Dalton et al., 2003; Sublette et al., 2009; Swann et al., 2005). Thus, one would expect successful treatment for co-occurring BD and SUDs to target impulsivity and/or aggression. Because of the severe mood alterations associated with BD and co-occurring BD and SUDs, psychological interventions such as individual and group counseling and motivational interviewing are typically augmented with pharmacotherapy (Whicher & Abou-Saleh, 2007). Lithium, arguably the best-studied mood stabilizer used in the treatment of BD (Thase & Sachs, 2000), has been shown to decrease positive urine drug screens and increase global functioning in adolescents with BD (Geller et al., 1998). Similarly, valproate, another mood stabilizer, decreased mood lability and alcohol consumption in adults with BD (Salloum et al., 2005). Although speculative, these mood stabilizers may decrease BD and SUD severity because they decrease impulsivity. This view may help explain why quetiapine, an atypical antipsychotic, improves depression ratings in adults with BD but not alcohol consumption in adults with co-occurring BD and SUDs (Brown et al., 2008), and the anticonvulsant carbamazepine has been shown to decrease positive urine screens for cocaine in adults with BD and cocaine abuse but not to significantly alter mood ratings (Brady et al., 2002). That is, the mechanism

of these latter two agents may not target the underlying functional domain of impulsivity, and therefore may alter mood or substance use, but not both.

Conclusions

Limitations and Future Directions

One of the major difficulties that researchers encounter when studying the role of impulsivity in dual diagnosis is the inherent multiplicity of factors of which impulsivity is composed (Evenden, 1999). This complexity makes a simple association between dual diagnosis and impulsivity challenging; however, we may arrive at a more nuanced understanding of this association by measuring impulsivity more systematically. For instance, many of the studies mentioned above measure trait impulsivity using the BIS, but there is little overlap in measures of state impulsivity. It would be helpful to establish age-appropriate batteries of impulsivity that, for instance, divide along lines of state versus trait (Wingrove & Bond, 1997) or motivational versus inhibitory control (Jentsch & Taylor, 1999). This method would increase power in comparing results from specific measures across studies. Furthermore, most studies of individuals with dual diagnosis do not address impulsivity, either because their findings are negative or because impulsivity was not assessed; adding a measure of impulsivity, in most cases, constitutes a minor addition to a protocol that would add considerably to the state of the field if adapted widely. Additionally, systematically dissecting impulsivity through a battery of self-report and behavioral measures may help elucidate the precise relationships between components of impulsivity and clinical features of dual diagnosis.

Another layer of complexity in studying dual diagnosis is the presence of more than two disorders, that is, a SUD and one or more other psychiatric disorder. As discussed above, findings from the NCS-R reveal that 17.3% of respondents met DSM-IV criteria for three or more disorders (Kessler et al., 2005); this number may be even higher in those with SUDs (Regier et al., 1990). For instance, eating disorders and SUDs are often associated with personality disorders (Thompson-Brenner et al., 2008). Further, in a conventional Kraepelinian diagnostic framework, it is difficult to parse features of a SUD from those of the so-called primary diagnosis or its treatment, as is frequently the case in co-occurring BPD and SUDs (Krishnan, 2005). Therefore, future studies may consider multiple diagnoses that co-occur with substance abuse, which

may yield clusters of psychiatric features based on functional impairments.

In addition, because the average onset for most psychiatric diagnoses is in childhood or adolescence (Kessler et al., 2005), future research should seek to understand how developmental changes interact with psychopathology, substance abuse, and impulsivity. This is a difficult task, given the extensive shifts in the cognitive and emotional functioning of even typically developing adolescents (Hawkins, 2009; Paus et al., 2008); however, this developmental work would be helpful in understanding the developmental aspects of the relationship between substance abuse and other psychopathology. That is, characterizing children with nonsubstance psychopathology before the onset of substance abuse may help to determine how substance abuse influences the course of typical and atypical development and what features precede substance abuse in children with psychopathology.

One method that would further elucidate the role of impulsivity would be to define and examine putative endophenotypes as they relate to impulsivity and co-occurring disorders (Gottesman & Gould, 2003). Impulsivity exists on a spectrum that can be quantified in family members of dually diagnosed individuals; this approach could yield important data regarding genetic predispositions to the negative sequelae of impulsivity and improve early intervention strategies for those at high risk for dual diagnosis.

Treatment Implications

Overall, the data regarding the role of impulsivity in dual diagnosis suggest treating the functional impairment rather than focusing solely on the disorder. As an analogy, there are treatments specific to heart disease and diabetes, but there are treatments for the functional impairment of high cholesterol that are managed similarly regardless of the formal diagnosis. Likewise, clinicians may address impulsivity or poor decision making and consequence appraisal rather than, for instance, ADHD or a SUD. The treatment strategies reviewed in each section above give support to this concept. For example, in substance-abusing individuals with eating disorders, treating the eating disorder or the SUD alone may lead to exacerbation of the other set of symptoms; an integrated treatment plan that focused on underlying impairments may concurrently decrease the severity of both substance abuse and disordered eating (Coelho et al., 2007).

In this framework, pharmacological treatments for dual diagnosis might target neurotransmitter

systems known to contribute to impulsivity. For instance, serotonergic abnormalities have been linked with impulsivity across disorders (Potenza, 2008), and recent research has specifically pointed to the role serotonin plays in affective aspects of impulsivity, such as reward-related tasks (Chamberlain et al., 2006). However, recent work has also implicated DA, noradrenaline, glutamate, and cannabinoid neurotransmission, findings that suggest novel therapeutic approaches (Pattij & Vanderschuren, 2008). By designing interventions for individuals with dual diagnosis that target the neurobiological substrates of impulsivity, clinicians may address multiple related problem behaviors simultaneously.

In summary, one model of treatment for dual diagnosis would involve (1) establishing a dual diagnosis, (2) assessing for impulsivity, and (3) providing impulsivity-targeted treatment that uses behavioral and psychopharmacological options that have shown efficacy in reducing impulsivity. At the very least, evaluations of dually diagnosed patients should include assessments of impulsivity and treatment plans should include efforts to reduce symptoms associated with impaired impulse control.

Summary

Throughout this chapter, we have attempted to conceptualize dual diagnoses within a framework in which impulsivity represents a main underlying construct that contributes to the experiencing of co-occurring disorders. The prevalence of dual diagnosis suggests that DSM (and DSM-like) categories have substantial overlap with respect to the targeted psychiatric populations, allowing one to challenge current psychiatric epistemology. A bottom-up system of categorization might lead to more homogeneous groups based on unifying factors, such as impulsivity. In turn, research into treatment for these groups would ideally be more productive because it could focus on specific substrates of a given functional impairment. Within this framework, impulsivity is not just a trait-dependent feature of dual diagnosis, but represents a unifying domain that could explain the co-occurrence of substance abuse and other psychiatric impairments.

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Epidemiology and Phenomenology of Pathological Gambling

Luke Clark

Abstract

Pathological gambling is an impulse control disorder (ICD) characterized by loss of control over gambling behavior. This chapter will describe the illness profile of pathological gambling. As well as summarizing the epidemiological data on the prevalence of pathological gambling and its associated comorbidities, I will also consider (1) the classificatory overlap between pathological gambling, the substance use disorders, and obsessive-compulsive disorder; (2) the emerging evidence for dimensional rather than categorical models of disordered gambling; and (3) some of the sources of heterogeneity among pathological gamblers, including the differences between common games. In the second part of the chapter, I will review several sets of psychological and neurobiological factors that are implicated in the etiology of pathological gambling, including the role of physiological arousal ("excitement"), conditioning influences, cognitive distortions, personality trait variables, and neuropsychological and neuroimaging markers. These mechanisms are often complementary, and a biopsychosocial theory of gambling will incorporate multiple levels of explanation.

Keywords: gambling, cognition, neuropsychology, risk-taking, impulsivity

Introduction

Pathological gambling is defined in the current DSM-IV-TR (American Psychiatric Association, 2000) as a persistent pattern of maladaptive gambling behavior. To meet a diagnosis, the individual must display at least 5 of 10 specified symptoms, which are summarized as follows: a preoccupation with gambling, the need to gamble with increasing sums of money, restlessness or irritability when stopping gambling, loss of control over gambling, chasing losses, lying to significant others about gambling, gambling as a means of escape, jeopardizing work or personal relationships due to gambling, illegal activity to support gambling, and relying on others to relieve gambling debts. The diagnosis was introduced in the DSM-III in 1980, and has undergone revision in the subsequent editions of DSM that is likely to continue with DSM-V. The term *pathological gambling* is preferred over the previous

term *compulsive gambling*, as the latter term carries the connotation of an ego-dystonic disorder that is not evident in many severe gamblers (Blaszczynski & Nower, 2002; Moran, 1970). The term *problem gambler* is also used widely to refer to individuals with symptoms of disordered gambling who do not fulfill the DSM requirement of five symptoms. The literatures on problem gambling and pathological gambling are blurred. The present discussion will incorporate findings from both, as well as data on gambling as a recreational activity in the general population.

In the DSM-IV-TR, pathological gambling is grouped within the impulse control disorders (ICDs), but its nosological status remains controversial (Potenza, 2006). The similarities between pathological gambling and the substance use disorders have been apparent since its classification, and the symptoms themselves were partly modeled on

those for substance dependence (Rosenthal, 1989). If pathological gamblers cease gambling suddenly, signs of withdrawal are reported, including psychological symptoms like restlessness and irritability, but also physiological symptoms like nausea and heart palpitations (Rosenthal & Lesieur, 1992; Wray & Dickerson, 1981). The development of tolerance (i.e., gambling with increasing amounts of money over time) and gambling cravings (Tavares et al., 2005) are also apparent, and along with withdrawal, these are often considered the hallmarks of an addiction syndrome. Other commonalities with substance use disorders will be noted in the following sections, and pathological gambling may ultimately be classified alongside these disorders. Other researchers have aligned pathological gambling with obsessive-compulsive disorder (OCD), placing the disorder on an impulsive-compulsive spectrum (Hollander & Wong, 1995; McElroy et al., 1994).

The aims of this chapter are to describe the illness characteristics of pathological and problem gambling and to consider some of the etiological influences that have been linked to the development of gambling problems. The first section will cover the epidemiology of the illness, including its prevalence, pattern of associated disorders, and natural course. The second section will cover some of the psychological factors implicated in pathological gambling. While myriad approaches that have been taken to this issue, I will focus on psychological and neurobiological mechanisms (see Orford, 2001; Walker, 1992a for fuller descriptions).

Epidemiology of Pathological Gambling

Prevalence

Gambling is a common recreational pastime in much of the world, and community surveys across several countries indicate that the majority of people engage in at least occasional gambling. In the 2007 British Gambling Prevalence Survey, 68% reported gambling in the past year (Wardle et al., 2007), a figure that has remained stable over the past decade (Sproston et al., 2000), and in the U.S. National Comorbidity Survey Replication, 78% reported lifetime gambling (Kessler et al., 2008). As a common behavior, gambling then becomes dysfunctional in a minority. The lifetime prevalence for DSM pathological gambling is estimated at 1%–2%, based on a meta-analysis of 120 North American studies up to 1997 (Shaffer et al., 1997), as well as specific surveys from Canada (Bland et al., 1993), the United States (Cunningham-Williams et al., 1998; Gerstein et al., 1999; Welte et al., 2002) and the United Kingdom

(Wardle et al., 2007). If we take a more liberal cutoff for problem gambling (e.g., at least three DSM symptoms), the lifetime prevalence estimates increase to around 2%–5% (Shaffer et al., 1997).

These rates of problem and pathological gambling are elevated in certain demographic sectors. With respect to gender, the rate of pathological gambling is greater in males than females (Shaffer et al., 1997). For example, a Canadian prevalence study reported a threefold higher rate in males (Bland et al., 1993). Males also account for a greater proportion of treatment-seeking cases, although the phenomenon of *telescoping* has been reported (as for substance use disorders), in which females may progress from initial recreational gambling to problematic levels of use more rapidly than males (Grant & Kim, 2002).

With respect to age, rates of pathological gambling are higher in adolescents (3%–8%) and college students (4%–14%; Ladouceur et al., 1994; Shaffer et al., 1999), and diminish in adults over age 60 (Welte et al., 2001). The typical age for the development of problem gambling is in the 20s, and pathological gamblers report initiation of recreational gambling at a younger age than nonproblem gamblers (Kessler et al., 2008), a trend that is also evident in substance use disorders.

Within clinical and community samples, problem gambling is reliably associated with a range of adverse psychosocial outcomes; for example, these individuals experience higher rates of divorce and marital separation (Cunningham-Williams et al., 1998), unemployment (Bland et al., 1993), incarceration (Potenza et al., 2006), and suicidal behavior (Bland et al., 1993). Adolescent problem gamblers report more delinquency and academic problems (Stinchfield, 2000). It is also important to acknowledge the environmental context: rates of problem gambling covary closely with gambling availability. This can be seen in terms of spatial geography, as rates of problem gambling are higher in those living within 10 miles of a casino (Welte et al., 2004b), and also temporally, in studies comparing gambling behavior before and after the introduction of new varieties of gambling, such as the British National Lottery (Grun & McKeigue, 2000).

Comorbidity in Pathological Gambling

Pathological gambling co-occurs with a broad range of other psychiatric diagnoses, including substance use disorders, personality disorders, mood disorders, and anxiety disorders (e.g., Cunningham-Williams

et al., 1998; Kessler et al., 2008; Petry et al., 2005). In the National Co-morbidity Survey Replication, a U.S.-based community survey of 3435 individuals, 0.6% of subjects met criteria for lifetime pathological gambling (2.3% reported at least one core symptom), but of these, 96% met criteria for at least one other lifetime diagnosis and 49% had been treated for another mental illness (Kessler et al., 2008). Thus, comorbidities are the rule rather than the exception, and to exclude individuals with coexisting diagnoses from research studies runs a risk of creating overselected and unrepresentative samples. The St Louis Epidemiologic Catchment Area (ECA) study observed a similar pattern of comorbidities in problem gamblers, who were defined as gamblers with at least one clear symptom of gambling-related harm (Cunningham-Williams et al., 1998). In this community survey ($n = 3000$), antisocial personality disorder was the most frequent coexisting diagnosis, followed by alcohol abuse/dependence and nicotine abuse/dependence.

These links with substance use disorders are particularly well established, and support the etiological overlap between problem gambling and drug addiction. In individuals seeking treatment for gambling problems, the rates of substance use disorder are highly elevated (around 50%; Black & Moyer, 1998); in those seeking treatment for substance-related problems, the rates of pathological gambling are inflated above population norms (Lesieur & Heineman, 1988). Treatment-seeking problem gamblers who were daily cigarette smokers also reported more severe gambling problems and higher gambling expenditure than treatment-seeking gamblers who did not smoke (Petry & Oncken, 2002).

The temporal ordering of pathological gambling versus its comorbidities (i.e., which condition manifested first) has been less widely studied, and there is an acknowledged paucity of longitudinal data in the field (see the section "Measuring Gambling Involvement"). To take the example of depression, pathological gambling could precipitate depressive symptoms (e.g., due to debt or interpersonal conflict); conversely, pathological gambling may arise secondary to depression as an attempt to alleviate low mood or escape from reality. The available data based on retrospective reports (Cunningham-Williams et al., 1998; Kessler et al., 2008) indicate that both directions can occur for most of the major comorbidities. However, for mood, anxiety, and substance use disorders, the most common pattern was that pathological gambling emerged *later* than the other disorder, and in

the case of other ICDs, the data were exclusively in this direction (Kessler et al., 2008). Data in adolescents using path analytic modeling also indicate that depression is more commonly a predictor than a consequence of gambling behavior (Gupta & Derevensky, 1998), and these data are compatible with theoretical models of gambling vulnerabilities (Blaszcynski & Nower, 2002).

Epidemiological links with OCD have also received some attention following the assertion that pathological gambling may lie on an impulsive-compulsive spectrum (Hollander & Wong, 1995; McElroy et al., 1994). At the present time, comorbidity data do not provide clear support for the proposal. In the St. Louis ECA study, which outlined many of the major comorbid conditions, there was no significant relationship between problem gambling and OCD (Cunningham-Williams et al., 1998). While subclinical OCD traits are somewhat elevated in groups with pathological gambling (see the section "Personality Variables in Pathological Gambling"), a study of 293 cases with primary OCD failed to find any elevation in rates of pathological gambling above population norms (Grant et al., 2006), and a similar lack of association was observed in a study of family members of OCD probands (Bienvenu et al., 2000). In contrast, other ICDs are elevated in groups of pathological gamblers: a study of 40 pathological gamblers in treatment found a rate of other ICDs of 35% (compared to 3% in controls), of which compulsive buying and compulsive sexual behavior were the most common (Specker et al., 1995). This study also found that attention deficit/hyperactivity disorder (ADHD) was elevated in the gamblers, and recent longitudinal work has shown that persistent ADHD symptoms in childhood and adolescence predicted subsequent gambling problems, compared to subjects without ADHD or with nonpersistent ADHD restricted to childhood (Breyer et al., 2009). Thus, the emerging consensus from this line of inquiry is that pathological gambling is a condition associated with substantial psychiatric comorbidity, with mood, anxiety, and personality disorders being highly prevalent. In particular, pathological gambling is aligned with other forms of mental illness characterized by impulsivity, namely, the other ICDs, ADHD, and the substance use disorders.

Natural Course of Pathological Gambling

Repeated failed attempts to control gambling is a diagnostic feature of pathological gambling and implies that this is a chronic, relapsing disorder.

In support of this notion, Shaffer and Hall (2002) described a prospective study of casino employees with excessive gambling, who showed a fluctuating course of improvement, relapse, and remission that closely resembled the course of a second group of subjects with excessive alcohol consumption. In one of the first prospective studies of gambling behavior, Winters et al. (2002) interviewed adolescents about gambling and other risky behaviors at ages 16, 17, and 23. Rates of problem gambling remained stable over the three waves, but several variables at waves 1 and 2 were predictive of wave 3 problem gambling, including male gender, substance abuse, and parental gambling history. There is also preliminary evidence of a “hopping” phenomenon seen in polydrug users, where as consumption of one drug declines (e.g., through treatment), consumption of another drug escalates (Hser et al., 1990). Blume (1994) describes this phenomenon in a pathological gambler with comorbid drug use.

Recent work, however, has indicated more variability in the trajectories of those with gambling problems and a heterogeneous prognosis. In a large survey of individuals identified with lifetime pathological gambling in a community survey, 36%–39% of them did not report any gambling problems in the past year, despite only 7%–12% having received treatment for gambling problems (Slutske, 2006). While some of these individuals may have experienced remission of their gambling problem as a result of treatment for other illnesses (Afifi et al., 2006), it is likely that a subset of problem gamblers experience natural recovery. This conclusion has been echoed in a longitudinal study showing that young adults frequently move in and out of gambling problems (Slutske et al., 2003). Vitaro et al. (2001) found that gambling participation at age 16 was a better predictor of gambling problems 1 year later than problem gambling itself at age 16. A meta-analysis of five longitudinal studies found no evidence for the persistence of severe problem gamblings and no evidence that mild problem gamblers were more at risk of progression to pathological gambling than nonproblem gamblers (LaPlante et al., 2008).

Measuring Gambling Involvement

In addition to the DSM interview, a number of other psychometric instruments have been devised to assess the severity of gambling problems. The South Oaks Gambling Screen (SOGS; Lesieur & Blume 1987) is a widely used 20-item self-report questionnaire, with cutoff scores of 3 for problem gambling and 5 for “probable Pathological Gambling.” The SOGS

scores are highly correlated with the endorsement of DSM-IV criteria for pathological gambling, and the screen has good classification accuracy in clinical samples (Stinchfield, 2002). However, in general population samples, the SOGS may overestimate pathological gambling, with an estimated 50% false-positive rate (Ladouceur et al., 2000; Stinchfield, 2002). Researchers are beginning to favor the interviewer-rated Canadian Problem Gambling Index (Ferris & Wynne, 2001) as an alternative. In studies that aim to detect fluctuations in symptom severity, the Addiction Severity Index–Gambling version (ASI-G; Petry, 2003) is widely used.

Psychometric studies of these scales yield either unidimensional or two-factor solutions. Studies using the SOGS and the DSM criteria have indicated a single dimension of gambling severity, which in the case of the DSM criteria accounted for 45%–68% of the variance (Stinchfield, 2002; Strong & Kahler, 2007). However, a secondary analysis of the 2000 British Gambling Prevalence Survey, which also employed the DSM and SOGS, indicated two separable factors aligned with gambling dependency (withdrawal symptoms, tolerance) and gambling-related harms (e.g., lying, illegal acts; Orford et al., 2003).

Other studies have utilized community surveys to assess the broader spectrum of disordered gambling. Toce-Gerstein et al. (2003) looked at the prevalence of each DSM symptom in individuals manifesting 1–2 symptoms (at-risk gamblers), 3–4 symptoms (problem gamblers), 5–8 symptoms (pathological gamblers), and 8–10 symptoms (severe pathological gamblers). Whereas the loss-chasing symptom was present across all four levels of severity (including 51% of at-risk gamblers), other symptoms emerged with regularity only in the more severely affected groups. Symptoms of dependence (withdrawal, tolerance) were reliably observed only in the groups defined as pathological gamblers (at least five symptoms) and illegal acts to support gambling only in the most severely affected group (at least eight symptoms). Although cross-sectional in nature, these findings suggest that a *trajectory* of gambling-related problems may exist. More broadly, it is evident that substantial symptomatology and gambling-related harms are present in individuals who do not fulfill the full DSM criteria for pathological gambling.

Sources of Heterogeneity in Gambling

The discussion up to this point has considered gambling as a single homogeneous behavior, but

this is inevitably a gross oversimplification. There are major psychological differences between different gambling games, which will cause certain games to appeal more to certain types of individuals. Moreover, there may be a variety of motivations driving the development of pathological gambling, and it may be unwise to seek a common etiological mechanism that describes all cases (Blaszczynski & Nower, 2002).

To first consider the various forms of gambling, these vary widely across cultures, and some forms can be culturally specific for example, Japanese Pachinko, a hybrid of a slot machine and pinball. That said, the most common varieties of gambling in most Western countries are as follows: lottery, scratch cards, slot machines, casino games (e.g., roulette, craps), card games (e.g., poker), and horse racing (and other forms of sports betting). A fundamental question is whether these games are differentially associated with the risk of developing gambling problems. This has proved a difficult question to address, in part because of differences in the social acceptability of different games and the tendency of gamblers to play multiple games, but it has been tackled in two basic ways. The psychological approach asks whether certain features of gambling games (*structural characteristics*, such as a rapid rate of play) are associated with greater gambling tendencies (e.g., persistent play in the laboratory). The epidemiological approach looks within large surveys at the prevalence of problem gambling among players of different games.

One important psychological distinction within these common varieties of gambling is between games of pure chance (e.g., lottery, roulette, slot machines) and games with some objective skill component (e.g., blackjack, sports betting). Other psychological parameters that vary among games and that could be associated with the *addictive potential* of the game include the wager size (e.g., small in slot machines, potentially very large in sports betting), the jackpot size (e.g., reasonably modest in slot machines, very large in the lottery), the delay between wagering and the outcome (e.g., short for slot machines and scratch cards, longer for horse racing and lotteries), and the potential for continuous play (not an option in lotteries; Griffiths, 1993a). Established principles of learning theory (see the section “Conditioning Influences”) would predict that rapid forms of gambling (namely, slot machines and scratch cards), with short delays between wager and outcome and the potential for continuous play, would be associated with the

highest levels of problematic behavior. Varieties with longer delays and discontinuous play (primarily state lotteries) would be expected to show the lowest levels of problem gambling. There is some evidence in line with these predictions; for example, in treatment-seeking pathological gamblers, the use of gambling machines was associated with more DSM symptoms (Morgan et al., 1996) and a rapid onset of problem gambling (Breen & Zimmerman, 2002). Wood and Griffiths (1998) also reported a high prevalence of pathological gambling (6%) among British adolescent scratch card players. Meanwhile, although the vast majority of problem gamblers report playing the lottery, it is unusual to see problem gamblers for whom the lottery is their preferred, or most problematic, form of gambling.

Epidemiological studies have sought to assess the prevalence of problem gambling among recreational players of various games in large cohorts. In some studies, the engagement with multiple games can be statistically controlled (Welte et al., 2009). These studies generally fail to support the “rapid rate” hypothesis, finding the highest risks of problem gambling associated with casino games, card games, and sports betting (Cox et al., 2000; Welte et al., 2004a, 2009). For example, in an analysis of data from a telephone survey of U.S. youths aged 14–21, in subjects reporting any past-year gambling ($n = 1,535$), the number of SOGS symptoms was most strongly associated with engagement in card games and casino gambling (Welte et al., 2009). The use of gambling machines (outside a casino) was not associated with a significant increase in gambling symptoms. Of course, it is an open question whether card games and casino gambling are more addictive or whether players with developing problems are drawn to these more advanced forms of gambling. Nonetheless, it is notable from these epidemiological data that a degree of skill involvement (possibly fueling cognitive distortions such as the illusion of control; see the section “Cognitive Distortions in Gambling”) is more strongly linked to problem gambling than the potential for rapid play.

Many gambling games can now be accessed through the Internet. The prevalence of Internet gambling is reported in recent surveys as relatively low compared to the established forms of gambling (Wardle et al., 2007; Welte et al., 2009). For example, in the 2007 British Gambling Prevalence Survey, 3%–4% of respondents reported online gambling in the past year, compared to 20% using scratch cards, 17% betting on horse racing, and 14% using slot machines (Wardle et al., 2007).

These rates appear higher in certain sectors; for example, 6% of an American sample of college students reported Internet gambling on a weekly basis (Petry & Weinstock, 2007). The youth survey by Welte et al. (2009) found that Internet gambling was among the least common forms of gambling (involving only 2% in the past year), but a dramatic 65% of respondents who gambled online reported at least one SOGS symptom. This might suggest a highly addictive form of gambling; however, it was also noted that the Internet gamblers tended to engage in the greatest number of other gambling games (mean, 6.9) and reported the highest number of gambling episodes per year (mean, 387). After controlling for these variables, the risk associated with Internet gambling per se was not statistically significant. Nevertheless, there are some valid concerns about Internet gambling; the constant accessibility and privacy of the Internet may present particular threats to vulnerable individuals and to those who may be disinclined to visit traditional venues like bookmakers or casinos.

In terms of the motivational differences within groups of pathological gamblers, it has long been recognized that some gamblers appear understimulated and gamble as a way to achieve excitement, whereas other gamblers appear overstimulated and gamble as a form of distraction or to alleviate anxiety (see the section "Conditioning Influences" for a description of behavioral mechanisms). These subtypes may covary both with preferred games and with gender. There is some evidence that casino and sports-betting players may be high sensation seekers who are motivated by the thrill of play (i.e., the understimulated subtype), whereas the repetitive nature of slot machine play may serve primarily to distract and alleviate low mood (i.e., the overstimulated subtype; Clarke, 2005; Cocco et al., 1995). In a study of 131 treatment-seeking pathological gamblers, preferred forms of gambling among males were blackjack, cards, and sports betting, whereas preferred forms among females were slot machines and bingo (Grant & Kim, 2002).

In the influential Pathways Model of problem gambling, Blaszczynski and Nower (2002) attempt to accommodate this heterogeneity with three subgroups of gamblers, varying on a hierarchy of biopsychosocial influences. They propose a basic conditioning process (see the section "Conditioning Influences") across all problem gamblers, whereby the act of wagering and the stimuli in the gambling environment come to be associated with physiological arousal and a feeling of excitement (see the

section "Physiological Arousal during Gambling"). Over time, these conditioned responses may foster cognitive distortions and faulty beliefs about winning and probability (see the section "Cognitive Distortions in Gambling"). Under certain circumstances (e.g., a dramatic "big win" early in the gambling career), the conditioning pathway may be sufficient to create a problem gambler in the absence of any preexisting psychopathology (Pathway 1). However, two further pathways are postulated, whereby the conditioning processes are predisposed in individuals with "emotional vulnerability" (Pathway 2: depression or anxiety disorders, or biological vulnerabilities such as neurotransmitter abnormalities; see the section "Neurobiology of Problem Gambling") or individuals with "antisocial impulsive" tendencies (Pathway 3: features of trait impulsivity, ADHD, or antisocial personality disorder, coupled with neuropsychological evidence of executive dysfunction; see the section "Neuropsychological Function in Pathological Gambling"). While large-scale empirical support for these three subgroups is lacking, the Pathways Model provides a useful framework for considering the interplay between the various psychological and epidemiological risk factors, and highlights the variability in etiological processes among different individuals with pathological gambling.

Etiological Factors in Pathological Gambling

As we have seen, gambling is a widespread activity in the general population that becomes disordered in a subset of individuals. Pathological gambling represents the extreme end of disordered gambling. To psychologists and behavioral economists, the sheer existence of gambling holds an enduring fascination. In deciding to gamble, the gambler experiences a standard choice scenario where the available options are either to gamble or to not gamble (i.e., walk away). In the traditional approach to decision making, subjects calculate expected values for the two options by combining the various costs and benefits of the different outcomes (win gamble, lose gamble) with the probabilities that those outcomes will occur. Subjects should then choose the option with the greatest expected value. The expected value of the "walk away" decision is approximately zero. The expected value of the gamble decision is negative: with repeated play, the expected losses should invariably outweigh the expected gains. Gamblers show at least some awareness of this fact, evidenced by the popular expression "the house always wins."

So, why do gamblers continue to select the “gamble” option over the “walk away” option? Many accounts have been proposed, but these can be divided into two basic approaches (Ladouceur & Walker, 1996). The first approach holds that gambling offers something to the gambler over and above the basic monetary values of the wagers and the available wins. Decision-making researchers would say that gambling offers some additional *utility*. The excitement or thrill of playing is one obvious contender for this utility (e.g., Brown, 1986), and the evidence for gambling-induced arousal (a physiological correlate of subjective excitement) will be reviewed in the section “Physiological Arousal during Gambling.” Thus, according to this account, the decision whether to gamble or quit is not made on a purely financial basis, and while the monetary (objective) expected value is negative, the *subjective* expected value of gambling is positive, and therefore this option is selected.

The second approach argues that gamblers experience a range of cognitive distortions and biases that cause them to misperceive the expected value and overestimate their chances of winning. Thus, the decision whether to gamble or walk away could be made on a purely financial basis, and while the true expected value is negative, the experienced expected value of gambling is positive. Various features of gambling games and the gambling environment may fuel these cognitive distortions, such that the gamblers may appraise their chances of winning more realistically when they are not at a gambling venue (e.g., in a treatment facility). The evidence for cognitive distortions during gambling will be reviewed in the section “Cognitive Distortions in Gambling.”

Within these approaches, the risk of developing disordered gambling may be explained by individual differences in gambling-induced arousal or the susceptibility to gambling distortions. Within a *biopsychosocial* approach to pathological gambling, this interindividual variation may be explained by personality traits, variability in cognitive functions, or disruption of specific neural circuitry or neurotransmitter systems. (Indeed, these facets should not be viewed as mutually exclusive, but rather as different levels of explanation: a personality trait such as impulsivity may be associated with specific cognitive subprocesses and supported by localized neural circuitry and neurotransmitters.) Thus, the evidence for these characteristics in pathological gamblers will be reviewed in the sections “Personality Variables in Pathological Gambling,”

“Neuropsychological Function in Pathological Gambling,” and “Neurobiology of Problem Gambling.”

Physiological Arousal during Gambling

The seminal experiment in this area was performed by Anderson and Brown (1984): heart rate was recorded using ambulatory monitoring in 12 regular blackjack players during a period of real blackjack play in a casino. These regular players also attended a second session where they played blackjack in a psychological laboratory, as did a group of inexperienced student control subjects (who did not attend the casino session). Heart rate increased by an average of 25 beats per minute (bpm) in the regular players during the casino session compared to a preplay baseline period. The maximum increase observed, 58 bpm, is comparable to that of strenuous physical exercise. The heart rate increases in the casino environment were significantly higher than those observed in the laboratory (mean, 9 bpm in the regular players). In addition, the heart rate increase correlated with the average stake size and also with the personality trait of sensation seeking in the regular players in the casino but not in the other conditions. The Anderson and Brown experiment provides compelling evidence for cardiovascular arousal during casino gambling and is also cited as evidence for the need for ecological validity in gambling research. When gamblers were studied in naturalistic settings, the effects were not only stronger, but were qualitatively different compared to the laboratory condition (some caveats to this conclusion will be noted below).

Subsequent research has confirmed elevations in heart rate during other forms of gambling: slot machine play in amusement arcades (Coventry & Hudson, 2001; Griffiths, 1993b; Moodie & Finnigan, 2005), Pachinko in Japanese Pachinko centres (Shinohara et al., 1999), and in horse-race gambling in a betting shop (Coventry & Norman, 1997; Dickerson et al., 1987). Moreover, the effects are observed on other indices of physiological arousal, including skin conductance responses (Diskin et al., 2003; Sharpe et al., 1995) and plasma levels of the stress hormone cortisol (Meyer et al., 2000), and beta-endorphin (Shinohara et al., 1999).

Based on these findings, an appealing hypothesis is that pathological gamblers experience greater physiological arousal induced by gambling compared to nonproblem gamblers, and that this hyperarousal represents the additional utility of gambling

for these individuals and thus a risk factor for the illness. Sadly, the research findings pertaining to this elegant hypothesis are inconsistent. While several studies have reported greater increases in heart rate in regular or problem gamblers (Leary & Dickerson, 1985; Moodie & Finnigan, 2005; Sharpe et al., 1995), several other studies have failed to replicate the finding (Coventry & Norman, 1997; Diskin & Hodgins, 2003; Griffiths, 1993b). At least some of the inconsistency may be attributed to the varied approaches taken to classifying disordered gambling in these studies: a number of studies compared regular and irregular players without assessing actual symptoms of problem gambling. Griffiths (1993b), for example, compared adolescent slot machine players who reported playing more (regular) or less (irregular) than once per week. Both groups showed clear heart rate increases (mean, 22 bpm) during gambling, with no difference between groups. The heart rate returned to baseline more rapidly in the regular players, which Griffiths interpreted as evidence for tolerance in gambling. However, in the exemplary study by Sharpe et al. (1995), increased skin conductance levels were observed in response to a video of a gambling session, as well as a personalized gambling script, in problem gamblers, but no differences were observed between regular and irregular slot machine players.

In another of the stronger studies, Moodie and Finnigan (2005) compared heart rate increases in probable pathological gamblers (SOGS ≥ 5), infrequent gamblers (SOGS 0–7), and nongamblers (SOGS 0–2) during slot machine play in an amusement arcade. The problem gamblers were the only group to show a robust heart rate increase (mean, 8 bpm). However, this group tended to win more than the other groups during the session, and in an analysis collapsed across groups, subjects who won showed greater heart rate increases than subjects who did not win. This highlights one of the sacrifices of ecological validity and the limitation of studying gamblers in the natural environment: it is not possible to control for numerous play-related variables like win frequency. However, in a similar design comparing reasonably large groups of pathological gamblers (at least five DSM symptoms; $n = 30$) and regular, nonpathological gamblers (mean, one DSM symptom; $n = 34$), Diskin and Hodgins (2003) found no differences on multiple measures of arousal (heart rate, skin conductance levels, electromyographic activity) during real video lottery play in a laboratory setting. The pathological gamblers did report greater subjective excitement

during play and when thinking about winning, leading Diskin and Hodgins to suggest that the *perception* of arousal may be more closely related to disordered gambling than objective markers of physiological state.

Recent laboratory studies have begun to identify some of the psychological determinants of gambling-induced arousal. One key feature is the availability of genuine monetary rewards; when people gamble for tokens or hypothetical points, the heart rate response to gambling is greatly attenuated (Ladouceur et al., 2003; Wulfert et al., 2005). A recent study also shows that the *magnitude* of the heart rate response increases proportionally with the size of the available monetary reward (\$2, \$7, \$15; Wulfert et al. 2008). These findings are congruent with naturalistic studies that report greater heart rate increases in sessions where a win was experienced compared to equivalent sessions without a win (Coventry & Hudson, 2001; Moodie & Finnigan, 2005). Thus, laboratory studies of gambling behavior do not inherently lack ecological validity; rather, there are certain prerequisites (like the availability of monetary wins) that are important factors in maintaining ecological validity.

In summary, there is clear and incontrovertible evidence for elevations in physiological arousal during gambling play in naturalistic settings. Research attempting to link individual differences in gambling-induced arousal to disordered gambling has yielded inconsistent findings, with similar numbers of studies reporting increases in gambling-induced arousal in regular or problem gamblers to studies reporting no effect. These discrepancies may be expected based on the evidence for heterogeneity in problem gambling (see the section “Sources of Heterogeneity in Gambling”), and these sources of variability have not been explored thoroughly in relation to gambling arousal. It is thought that these indices of physiological arousal are related to subjective excitement as a form of reinforcement that maintains gambling behavior. However, the question of how gambling-induced arousal influences ongoing gambling behavior (e.g., the decisions to chase losses) has received very little empirical study (see Rockloff et al., 2007, for an exception). Increases in heart rate during slot machine play were correlated with a measure of gambling cognitive distortions (erroneous verbalizations; see below; Coulombe et al., 1992). However, it is unclear whether the state of arousal fosters gambling distortions or whether the presence of gambling distortions increases arousal; in reality, a bidirectional effect seems most likely.

Conditioning Influences

The development of disordered gambling can also be understood within the principles of learning theory, which distinguishes between Pavlovian (or classical) and instrumental (or operant) modes of conditioning. Both are relevant to gambling behavior, although textbook accounts emphasize the instrumental processes. Here, gambling is an instrumental response that the organism learns to associate with a reinforcing outcome (winning money). The reinforcement strengthens the learned association and increases the likelihood of repeating the response in the future. In theories of instrumental conditioning, the *schedule of reinforcement* is critical, and gambling is an example of a variable ratio (VR) schedule (or, more accurately, a random ratio schedule; Dickerson & O'Connor, 2006). In a VR schedule, reinforcement is delivered unpredictably on a proportion of responses, such that a win may be received twice in quick succession but then followed by many trials without reinforcement. For example, when betting on single numbers in roulette, wins are delivered on a VR38 schedule. Two important characteristics of the VR schedule, identified through studies of experimental animals, are that responding is difficult to establish but, once established, it is highly resistant to extinction. *Extinction* is the process by which responding diminishes when the reinforcement is withdrawn. This resistance to extinction may be analogous to the habitual, persistent responding seen in problem gamblers. With regard to the initial establishment of the gambling response, it is thought that the majority of problem gamblers experience some kind of significant win, either early in their gambling careers or shortly before their gambling becomes problematic (Custer, 1984). Empirical evidence for this *big win* hypothesis is rather limited and largely retrospective in nature. Some studies have tried to test the idea in the laboratory by delivering monetary wins early in a sequence of gambling trials and studying subsequent task persistence, but these studies have failed to provide evidence that early large wins foster persistent play (Kassinove & Schare, 2001; Weatherly et al., 2004). Of course, it is inherently difficult for researchers to emulate realistic gambling jackpots on a tight budget.

Monetary wins during gambling constitute an example of *positive reinforcement*, where the addition of a reinforcing stimulus increases the frequency of a response. Instrumental learning can also be driven by *negative reinforcement*: the removal of an unpleasant stimulus. This also has relevance to

gambling, as the alleviation of negative states including boredom, low mood, stress, and anxiety is often described by gamblers as an important motivation to play. In the study of gender differences by Grant and Kim (2002), female gamblers were more likely than male gamblers to report loneliness as a precipitant of gambling episodes.

Pavlovian conditioning is also relevant to gambling. This occurs when a stimulus acquires emotional significance through repeated pairing with an unconditioned stimulus that elicits an unconditioned response. In the case of gambling (and drug addictions), environmental cues (e.g., the sounds of coins falling in the slot machine) and gambling paraphernalia (e.g., playing cards, bookmaker logos) may become conditioned to the states of high arousal (elevated heart rate, a feeling of excitement) that occasionally follow monetary wins. These conditioned stimuli may then induce some degree of arousal in the absence of actual wins. For example, Loba et al. (2001) manipulated the sensory features of a slot machine game, including the speed of play and the presence/absence of sound cues. Subjective ratings of enjoyment and excitement were higher in the conditions with high sensory features, and these effects were most pronounced in probable pathological gamblers. In this manner, the learning theory approach to gambling is highly congruent with the psychophysiological approach described above, as the physiological arousal can be viewed as the expression of the learning process.

Cognitive Distortions in Gambling

The cognitive approach to gambling proposes that players misperceive, and more specifically *overestimate*, their chances of winning due to a variety of cognitive distortions and biases in the processing of the gambling scenario. A variety of distortions have been described (see Table 8.1), which generally pertain to the evaluation of the probability of winning, the misperception of randomness, and the belief that the game involves a greater element of skill than is warranted objectively. Several different distortions can often be detected in the same gambler, the types of distortion experienced can vary across preferred forms of gambling, and it is unlikely that any single distortion is present in all problem gamblers (Mitrovic & Brown, 2009; Toneatto et al., 1997).

Empirical support for the role of these distortions came from studies using the *think-aloud* procedure (see Gaboury & Ladouceur, 1989; Ladouceur & Walker, 1996, for review). Here, gamblers are

Table 8.1 Some Examples of Common Cognitive Distortions Associated with Gambling

Distortion	Description	Example
Gambler's Fallacy	Failure to appreciate the independence of turns	Belief that a win is due after a run of losses
The Near Miss	An outcome close to a win encourages further play	"I was really close last time; I'm going to play again."
Active Illusory Control	Belief in superstitious rituals or charms; preference for personal involvement or choice in games of chance	Blowing on dice to increase accuracy; belief in choosing certain lottery numbers.
Passive Illusory Control	Attributing outcomes to luck; reinterpreting outcomes in hindsight	"I knew I was going to lose that time; I wasn't concentrating properly."
Memory Biases	Preferentially recalling wins, ignoring losses	"I just keep thinking about how much money I won that time. That's got to happen again."

asked to play their preferred game in a naturalistic setting (e.g., a slot machine in an amusement arcade) for several minutes. During play, they are asked to verbalize all of the thoughts that occur to them, and it is emphasized that they should try to talk constantly and should not censure their thought content. The experimenter records the speech output and later encodes each statement as either an accurate, "rational" statement that reflects the true nature of randomness in the game (e.g., "It's a machine, we have no control over it, it's all luck") or an erroneous, "irrational" statement that reflects some faulty processing of probability, skill, or randomness ("I'm getting good at this game. I think I've mastered it"; Ladouceur & Walker 1996). The consistent finding with this paradigm is that in regular gamblers, roughly three-quarters of all gambling-relevant statements during play are classified as erroneous (Delfabbro & Winefield, 2000; Gaboury & Ladouceur, 1989; Griffiths, 1994; Walker, 1992b). These erroneous beliefs are most pronounced during actual gambling, and even occur at high levels in subjects who are basically aware that the game is determined by chance, from questionnaires administered before or after the procedure (Ladouceur & Walker, 1996).

The think-aloud procedure provides a sensitive composite index of cognitive distortions but has been criticized on several grounds. First, the distracting and intrusive nature of the verbalization procedure may change the thought content. Second, in

some gambling games, there are only a limited number of ways that a gambler can express accurate cognitions about the game. Third, one cannot infer that erroneous statements made in the think-aloud procedure reflect cognitions held with a high degree of conviction or reflect beliefs that are actually used to justify persistent gambling (Delfabbro, 2004; Dickerson & O'Connor, 2006).

One of the more prevalent gambling distortions, known as the *illusion of control*, is the tendency to infer skill involvement in games of chance. Craps players, for example, may blow on the dice or throw the dice with more forceful wrist movements when trying to roll high (Henslin, 1967). Studies by Langer (1975) identified a number of psychological variables that increase illusory control, including active involvement, a choice, and familiarity with the stimuli. The illusion of control can also be induced experimentally: in one of Langer's original studies (Langer, 1975, experiment 3), company employees were given the opportunity to buy a lottery ticket, and the experimenter was later asked to buy back their ticket. Those subjects who initially drew their ticket from a bag demanded more money (\$9) to exchange their ticket than a control group who were allocated a ticket at random (\$2). The other classic cognitive distortion is the *gambler's fallacy*: a failure to appreciate the statistical independence of different gambles. In games of chance, each outcome is generated independently of the past outcomes, creating a random, unpredictable sequence.

Humans are generally very poor at recognizing truly random sequences. For example, when evaluating binary sequences of heads and tails from a series of coin tosses, we typically expect a balanced number of each outcome, without long “streaks” of the same outcome. In gambling behavior, this can be manifested as the general belief that a win is somehow “due” after a prolonged run of losses, or when lottery players avoid certain sequences that they believe are non-random (Ayton & Fischer, 2004; Hardoon et al., 2001).

The *cognitive approach* predicts that the pathological gambler should show either a qualitative or quantitative difference in cognitive distortions compared to the nonproblem gambler. Given the high rate of erroneous verbalizations that occur in recreational players, this is not an easy hypothesis to test with the think-aloud method due to ceiling effects (e.g., Ladouceur, 2004). Nonetheless, Griffiths (1994) reported that regular slot machine gamblers (at least once per week) made more erroneous verbalizations on the think-aloud task than irregular players (less than once per month; see also Walker, 1992b). Delfabbro and Winefield (2000) reported that the frequency of erroneous statements was associated with the total amount wagered during the period of slot machine play. A study of university students also found more erroneous verbalizations during computerized games of roulette, blackjack, and a slot machine in subjects classified as probable pathological gamblers on the SOGS (Baboushkin et al., 2001).

With the development of psychometric scales to measure gambling distortions (e.g., the Gambling Beliefs Questionnaire; Steenbergh et al., 2002, and the Gambling-Related Cognitions Scale; Raylu & Oei, 2004), recent studies have begun to explore the association between gambling distortions and clinical measures of gambling pathology in much larger samples. Student gamblers displaying subthreshold problem gambling scored higher on a belief in luck scale compared to social, nonproblem gamblers (Wohl et al., 2007). In a study of the Vietnam Era Twin Registry ($n = 1354$), symptoms of pathological gambling on a DSM screening interview were positively correlated with cognitive distortion scores on a 12-item scale after controlling for other lifetime psychiatric diagnoses (Xian et al., 2008). Finally, a Canadian study of 11,562 individuals looked for the associations between cognitive distortions, risky gambling practices (either loss chasing, borrowing money, or betting more than one could afford), and gambling dependence (assessed as the need to

gamble with larger sums of money to achieve the same excitement), using item analyses from the Canadian Problem Gambling Index (Miller & Currie, 2008). The cognitive distortion score was seen to moderate the relationship between risky gambling practices and gambling expenditure, as well as the relationship between risky gambling practices and gambling dependence. Thus, in those individuals reporting at least one risky gambling practice, increased expenditure and dependence were *only* observed in those individuals who had cognitive distortions (Miller & Currie, 2008). To summarize, there is robust evidence that during gambling, players experience a distorted sense of skill, probability, and randomness such that they overestimate their chances of winning. While these distortions are reasonably common in recreational gamblers, there is accumulating evidence for at least a quantitative increase in the severity or frequency of erroneous thoughts in individuals with problem gambling.

Personality Variables in Pathological Gambling

Personality scales provide a relatively simple method of assessing individual differences in styles of thinking and feeling that may be associated with the risk of developing disordered gambling. It is therefore unsurprising that these measures have been studied extensively in the context of pathological gambling. In terms of the major “superfactors” of human personality (identified in the Eysenck 3-factor model and the Big Five model), pathological gamblers display higher levels of neuroticism, and the associated traits of harm avoidance and negative emotionality (Blaszczynski et al., 1986; Forbush et al., 2008; Roy et al., 1989). Given its diagnostic position among the ICDs, pathological gambling has been studied most widely in relation to a constellation of lower-order traits of impulsivity, sensation seeking and novelty seeking, which are themselves moderately intercorrelated. Numerous case-control studies have reported that scores on these scales (e.g., the Barratt Impulsivity Scale, the Eysenck Impulsivity-Venturesomeness-Empathy scale, the Zuckerman Sensation Seeking Scale) are elevated in pathological gamblers (Castellani & Rugle, 1995; Cunningham-Williams et al., 2005; Kim & Grant, 2001; Lawrence et al., 2009b; McCormick et al., 1987). The St Louis Area Personality, Health and Lifestyle Survey is the largest of these studies, where personality data on Cloninger’s Temperament and Character Inventory (TCI) were available from

913 respondents identified by random dialing and stratified by level of gambling involvement (Cunningham-Williams et al., 2005). The mean scores for novelty seeking were -9 in nongamblers, -2 in recreational gamblers, 24 in (subclinical) problem gamblers, and 105 in pathological gamblers ($p < .001$). Significant trends in the opposite direction were seen on the TCI traits of "self-directedness" (being reliable and purposeful) and "cooperativeness" (being tolerant and agreeable). There are some notable negative findings on impulsivity-type scales: Allcock and Grace (1988) found no differences between pathological gamblers and controls on the Barratt Impulsivity Scale, but in a very small sample, and Blaszczynski et al. (1986) found unexpected lower scores on the Zuckerman Sensation Seeking Scale, coupled with raised neuroticism, in 51 pathological gamblers. If some heterogeneity across different populations of gamblers is accepted (Blaszczynski & Nower, 2002), impulsivity scores have been shown to positively predict gambling severity (Krueger et al., 2005; Steel & Blaszczynski, 1998), the degree of physiological arousal during gambling (Anderson & Brown, 1984; Krueger et al., 2005), and short-term engagement with treatment (Leblond et al., 2003). Trait impulsivity has also been shown to mediate the relationship between depressive symptoms and problem gambling severity in a university sample (Clarke, 2006). Similar elevations and links with treatment outcome have been reported in substance use disorders (see Verdejo-Garcia et al., 2008, for review), and in healthy subjects, the level of trait impulsivity predicts the euphoric effect of stimulant drugs (Hutchison et al., 1999).

Two prospective studies form the most compelling evidence for trait impulsivity as a risk factor for the later development of disordered gambling. Vitaro et al. (1999) assessed 154 adolescents aged 12–14 on self-reported impulsivity (a 5-item version of the Eysenck scale) as well as on a cognitive test of response persistence, the Newman card-playing task. On this test, the subject must decide on each trial whether to play or quit the game. Play decisions cause a card to be turned over, which can either win or lose the subjects some points. The rate of reward is high at the start of the task (e.g., 70%), such that it is advantageous to play, but the rate of reward decreases over successive blocks of the task, such that there is an optimal point at which to quit the game. Play decisions beyond that point are maladaptive and provide a laboratory index of persistence that may be related to the clinical symptom of loss chasing. Indeed, the ecological validity of this

procedure was shown in a study by Breen and Zuckerman (1999) where self-reported impulsivity levels discriminated "chasers" (who lost all their points on the card-playing task) from "nonchasers" who quit while still in profit. In the Vitaro et al. study, the adolescents were followed up at age 17 and screened for problematic gambling behavior: baseline impulsivity and persistence on the card-playing task independently predicted gambling problems after controlling for initial gambling behavior, aggression, and anxiety.

A second prospective study by Slutske et al. (2005) used the Dunedin birth cohort, a group of 939 individuals born in Dunedin, New Zealand, in 1972. Subjects received a personality assessment with the Multidimensional Personality Questionnaire at age 18, and recreational use and misuse of gambling, nicotine, alcohol, and cannabis was assessed 3 years later, at age 21. Lower levels of "restraint" (high impulsivity) and higher levels of negative emotionality (neuroticism) were predictive of later gambling problems, consistent with the Vitaro et al. (1999) prospective findings. A second, equally important finding in this study was that the same personality profile was associated with the three forms of substance dependence even when the individuals with multiple risky behaviors (e.g., problem gambling and alcohol abuse) were excluded from the analysis (Slutske et al., 2005).

The trait of compulsivity has also received some attention in pathological gamblers, measured with subclinical scales of OCD symptoms such as the Padua Inventory. This has relevance to the possible conceptualization of pathological gambling on an impulsive-compulsive spectrum, although as reviewed above, epidemiological studies have failed to indicate significant comorbidity across these two conditions. Several studies have found significant increases on compulsivity scales in pathological gamblers (Blaszczynski, 1999; Frost et al., 2001). However, a direct comparison of personality measures in patients with pathological gambling and OCD revealed clear differences, with significantly higher novelty seeking (impulsivity) and lower harm avoidance (neuroticism) in the pathological gamblers (Kim & Grant, 2001). Any changes in compulsivity also seem to be outweighed by the group differences on impulsivity scales. For example, Blanco et al. (2009) studied the relationship between gambling symptoms, impulsivity, and compulsivity in 38 pathological gamblers participating in a clinical trial of paroxetine. Measurements were taken at baseline and after treatment. While the three scales

were intercorrelated at baseline, the correlation between gambling severity and compulsion did not survive partialing for impulsivity. Moreover, the reduction in gambling severity with treatment correlated with levels of impulsivity but not compulsion. These authors conclude that while both impulsive and compulsive features may be detectable in problem gamblers, the impulsive features predominate and are more closely associated with gambling severity (Blanco et al., 2009).

Neuropsychological Function in Pathological Gambling

Neuropsychological assessment aims to describe the profile of cognitive impairment and cognitive sparing in a disorder, using tasks that have been validated in patients with acquired focal brain injury. As such, neuropsychological findings provide an indirect method for localizing brain dysfunction. Typical domains of assessment might be attentional function (e.g., sustained attention), motor function (e.g., rapid reaction time), memory (e.g., list learning), visual perception (e.g., picture completion), and various forms of executive function. A number of neuropsychological studies in patients with pathological gambling have indicated basically intact performance in the domains of memory, motor function, visual perception, and sustained attention (see Goudriaan et al., 2004, for review; also see Goudriaan et al., 2006; Rugle & Melamed, 1993). Tasks of executive attention, like the Stroop test (where the subject must inhibit the tendency to read a color word in order to name the ink color the word is printed in) and the Wisconsin Card Sort Test (where the subject learns to sort cards according to color, shape, or number and occasionally has to switch the sorting rule), and executive measures of working memory function (e.g., digit span, self-ordered pointing), have yielded inconsistent results to date, with approximately equal numbers of papers reporting impaired and intact performance (Forbush et al., 2008; Goudriaan et al., 2006; Lawrence et al., 2009a; Leiserson & Pihl, 2007; Regard et al., 2003; Rugle & Melamed, 1993). For example, one of the stronger studies compared 49 pathological gamblers and 49 controls, and revealed deficient performance in the gamblers on measures of executive inhibition (Stop Signal Test), forward planning (Tower of London), and attentional shifting (Wisconsin Card Sort Test), coupled with intact working memory, motor speed, and visual retention (Goudriaan et al., 2006).

The heterogeneity across studies may be at least partially explained by illness severity. The subjects in the Goudriaan et al., (2006) study were currently engaged in treatment programs, whereas in our own study of problem gamblers recruited through the community, we observed no deficits in executive inhibition or working memory, although these domains were impaired in a second group with alcohol dependence (Lawrence et al., 2009a; 2009b; see Figure 8.1). There may be numerous other factors contributing to executive and attentional performance in pathological gambling. In the study by Regard et al. (2003), 52% of the problem gambling group reported some history of head injury. This can be easily overlooked in research studies using a single screening question ("Have you ever been knocked unconscious for more than 2 minutes?") but recent studies of substance users, with more rigorous interviewing for head injury, report similar high levels of past head injury (Bjork & Grant, 2009; Walker et al., 2007); for example, a 32% rate of head injury was reported among substance abuse patients in treatment programs (Walker et al., 2007). As described above, psychiatric comorbidities like depression and substance abuse tend to occur in the majority of individuals with pathological gambling. While a number of studies have excluded individuals with DSM diagnoses of lifetime substance abuse or dependence, studies have not typically controlled for nicotine usage, as well as subclinical levels of depression, ADHD, and OCD, which are often elevated in problem gamblers (Goudriaan et al., 2006; Lawrence et al., 2009b) and are known to influence cognitive ability in other disorders (e.g., Clark et al., 2002; Glass et al., 2009).

In contrast to the inconsistent findings in executive function, neuropsychological studies of risky and impulsive decision making have indicated a large and reliable deficit in problem and pathological gamblers. These impairments are thought to be underpinned by pathophysiology in the ventromedial aspect of the prefrontal cortex (vmPFC), as similar performance is observed in patients with focal lesions to this region due to stroke or tumor resection (Bechara et al., 2000; Clark et al., 2008). For example, in the Iowa Gambling Task (Bechara et al., 2000), the subject makes a series of 100 choices from four card decks (A, B, C, D), where each choice will either win or lose some hypothetical points. The four decks differ in their profitability: decks A and B are risky, associated with higher wins but occasional dramatic penalties so that the subject loses money over time. Decks C and D are

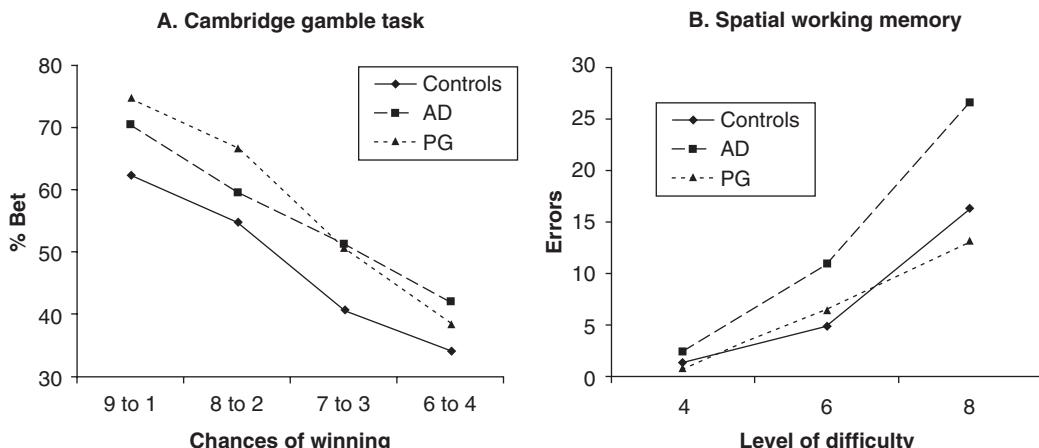


Fig. 8.1 Neurocognitive function in pathological gamblers (community recruited, SOGS ≥ 3), outpatients with alcohol dependence, and healthy controls. A: Pathological gamblers and alcohol-dependent individuals displayed increased betting behavior on the Cambridge Gamble Task. B: The alcohol-dependent group was selectively impaired on CANTAB Spatial Working Memory (<http://www.camcog.com>), a test of executive function, which likely reflects long-term harmful effects of alcohol consumption that are absent in pathological gamblers. Data redrawn from Lawrence et al. (2009a) with permission of the journal *Addiction*, the Society for the Study of Addiction, and Blackwell Publishing.

safe, associated with smaller wins but negligible losses so that the subject gradually accrues a profit. Healthy controls initially prefer the risky decks but rapidly adjust their responding to select from the safe decks, finishing the task in profit. Patients with vmPFC fail to develop an advantageous strategy on the task and typically finish in debt. A similar pattern of performance has been observed in several studies of pathological gamblers (Cavedini et al., 2002; Forbush et al., 2008; Goudriaan et al., 2005; Petry, 2001b; Roca et al., 2008). For example, Cavedini et al. (2002) compared 20 treatment-seeking individuals with DSM pathological gambling and 40 healthy controls and reported a highly significant difference in Iowa Gambling Task performance, such that the pathological gamblers actually increased their preference for the risky decks over the course of the task. This contrasted with intact performance in the Wisconsin Card Sort Test in the pathological gamblers.

The Iowa Gambling Task assesses decision making under uncertainty, as the probabilities of winning and losing on the four decks are never explicitly revealed to the subject. Recent studies have tested pathological gamblers on other decision-making tasks with explicit probability information (*decision making under risk*), such as the Game of Dice task (Brand et al., 2005) and the Cambridge Gamble Task (Lawrence et al., 2009a), and reported

similar impairments in performance. The pattern of elevated wagering seen on the Cambridge Gamble Task was also observed in a second group with alcohol dependence, who did not gamble (Lawrence et al., 2009a) (see Figure 8.1).

Decision making has also been examined in the time domain rather than the probability domain, using tests of delay discounting where the subject must choose between a small reward available immediately versus a larger reward available at some point in the future (e.g., “Would you prefer \$10 now or \$30 in 3 weeks’ time?”). By adjusting one parameter (e.g., the delay to the larger reward) across trials, one can calculate an *indifference point* for each subject where the two options are assumed to be valued equally. A preference for immediate rewards, and steeper discounting of delayed rewards, is a well-validated indicator of impulsivity that is reliably observed in substance use disorders (Bickel & Marsch, 2001; Reynolds, 2006). The same phenomenon of steeper delay discounting is reported in pathological gamblers (Dixon et al., 2003; Petry, 2001a; Petry & Casarella, 1999). The elegant study by Petry (2001a) directly tackled the issue of comorbidity by comparing groups with pathological gambling and substance use disorders ($n = 21$), pathological gambling alone ($n = 39$), and controls ($n = 26$). Steeper discounting of future rewards was observed in both groups with pathological gambling,

but there was an additive effect of substance abuse, with the steepest rate in the comorbid group. A study by Dixon et al. (2006) is also notable, as one of the few studies to compare a cognitive function across different environmental contexts. Twenty pathological gamblers completed a delay discounting task at their preferred gambling venue (an off-course betting facility) and in the laboratory. While the rate of discounting in the two contexts was highly correlated, the gambling context was associated with significantly steeper discounting of future rewards.

Recent studies have begun to directly contrast neuropsychological and personality-based methods of classifying subjects with problem gambling, although findings at this point are inconsistent and likely to depend heavily on the sensitivity and psychometric properties of the exact tests employed. A study by Goudriaan et al. (2008) was a follow-up to this group's case-control design, looking at predictors of treatment response in the pathological gamblers after 14 months. There were significant differences between responders and nonresponders (to the question "Do you think that you have a gambling problem again?") on baseline neuropsychological measures of response inhibition (stop-signal reaction time) and persistence (the card-playing test, described above) but no differences on personality measures of impulsivity. In contrast, however, Forbush et al. (2008) found that personality measures including impulsivity added significant incremental variance over a neuropsychological assessment in discriminating pathological gamblers and controls, but that neuropsychological variables (including measures of executive function and the Iowa Gambling Task) did not add significantly over personality measures.

Neurobiology of Problem Gambling

The indirect evidence from neuropsychological testing that pathological gamblers are impaired on measures of vmPFC function (reviewed above) have been substantiated by functional neuroimaging investigations in pathological gamblers, where brain activity is measured directly during performance of a cognitive task. In the first study of its kind, Potenza et al. (2003a) compared brain activity during performance of the Stroop test in pathological gamblers and healthy controls. While both groups displayed similar recruitment of dorsolateral PFC and anterior cingulate cortex, the gamblers showed reduced recruitment of the vmPFC region.

Subsequent studies have looked at simple decision-making tasks. An influential study by Reuter

et al. (2005) used a guessing task where subjects could win or lose 5 euros for correctly or incorrectly guessing the color (red or black) of an upturned playing card. The contrast of monetary wins minus monetary losses detected reliable recruitment of the ventral striatum and vmPFC (observable at the individual level), consistent with previous functional magnetic resonance imaging (fMRI) studies of monetary reinforcement (e.g., Breiter et al., 2001). These responses were attenuated in a group of pathological gamblers, and the fMRI response in these regions was inversely related to gambling severity measured with the SOGS. A study using an fMRI version of the Iowa Gambling Task also reported reduced vmPFC activity in problem gamblers with comorbid substance dependence compared to healthy controls, as well as in nongamblers with substance dependence (Tanabe et al., 2007). In the largest imaging study to date, de Ruiter et al. (2009) used a reversal learning task with monetary feedback in 19 pathological gamblers, 19 healthy controls, and 19 nongamblers with nicotine dependence. Receipt of monetary wins and losses were both associated with reduced signal in the lateral aspect of the ventral PFC in the pathological gamblers.

Two further fMRI studies have used the cue-induced craving paradigm to study the neural substrates of gambling urges in pathological gamblers. An early study by Potenza and colleagues (2003b) found reduced activation in the vmPFC region during presentation of personalized videotapes with gambling content in 10 male pathological gamblers compared to healthy controls. The pathological gambling group showed additional reduced activation of the striatum and thalamus. These findings corroborate the data with the Stroop test reported by the same group, as well as data in cocaine addicts using the same cue-induction procedure (Potenza, 2008). However, a subsequent cue-induction study comparing casino videos against nature videos found *increases* in brain activity in pathological gamblers in several regions, including the right dorsolateral PFC (Crockford et al., 2005). This finding is perhaps more compatible with the phenomenon that addicted individuals (either behavioral or substance addictions) show intense subjective reactions to the cue-induction procedure. The discrepant findings between these two fMRI studies may be attributable to the precise cue-induction method, the modeling of brain activity, the sensitivity of the scanning protocol to the ventral prefrontal region (where signal dropout often occurs), or patient characteristics.

The finding that emerges with some consistency from this handful of fMRI studies in pathological gamblers is one of impaired recruitment of the ventral PFC and, in some studies, the striatum (Potenza et al., 2003b; Reuter et al., 2005). The ventral PFC is thought to play a key role in impulse control and emotional decision making, but it is also recruited during basic reinforcement processing. As such, these findings are congruent with a broader *reward deficiency hypothesis*, which argues that the core motivation in disordered gambling may be to stimulate a developmentally underactive brain reward system. This hypothesis was developed in the context of substance addictions, where it has considerable empirical support (Bowirrat & Oscar-Berman, 2005). It is also compatible with the idea that at least a subgroup of problem gamblers are hypoaroused and motivated to seek intense emotional experiences (although failing to acknowledge the heterogeneity among gamblers in this respect, as discussed in the section “Sources of Heterogeneity in Gambling”).

At a neurochemical level, the transmitter dopamine is thought to play a pivotal role in brain reward processing, a conclusion based in part upon electrophysiological data recording from midbrain dopamine neurons during conditioning tasks in the monkey (Schultz, 2006). A number of studies have tested pathological gamblers on assays of dopamine transmission. In measuring markers of dopamine function in cerebrospinal fluid, Bergh et al. (1997) found decreases in dopamine coupled with increases in the metabolite homovanillic acid. Meyer et al. (2004) also reported greater increases in plasma dopamine (and noradrenaline) in pathological gamblers during a genuine casino session compared to a laboratory gambling session for points reward. Alterations in peripheral measures do not necessarily have a straightforward relationship to central transmission, however. Other evidence for dopamine involvement in disordered gambling comes from genetic studies looking at the prevalence of dopamine polymorphisms (Comings et al., 1996; Lobo & Kennedy, 2009), as well as pharmacological challenge studies showing increased urges to gamble, heightened processing of gambling information, and gambling-induced arousal in pathological gamblers following administration of dopaminergic drugs (amphetamine, haloperidol; Zack & Poulos, 2004, 2007).

A final line of evidence for dopaminergic involvement in problem gambling comes from studies of Parkinson’s disease, where the emergence of

pathological gambling is sometimes seen as part of a constellation of reward-seeking and impulse-control disorders, linked to the initiation of dopamine agonist medications (Dodd et al., 2005; Weintraub et al., 2006). Two drugs, pramipexole and ropinirole, have been highlighted in this linkage, and both have relatively high affinities for the dopamine D3 receptor, which is localized to the substantia nigra and limbic regions like the amygdala. While the phenomenon of medication-induced problem gambling is a provocative finding, its significance for neurobiological models of gambling behavior such as the reward deficiency hypothesis remains somewhat opaque. It links gambling to the *stimulation* of dopamine transmission, but in the context of a degenerative disorder of the dopamine system. To illustrate this complexity, the premorbid personalities of individuals who develop Parkinson’s disease are often conservative and *nonimpulsive* (Dagher & Robbins, 2009), but the emergence of pathological gambling as a side effect is associated with elevated trait impulsivity and sensation seeking, as well as earlier age of onset and familial or comorbid alcohol abuse (Voon et al., 2007).

In the first study to *directly* measure central dopamine function in problem gambling, using the D2-receptor radioligand [¹¹C]raclopride in a positron emission tomography (PET) scanning experiment, Steeves et al. (2009) compared patients with Parkinson’s disease with and without pathological gambling. In addition to a baseline scan to assess D2-receptor occupancy, a dynamic scan was acquired during performance of a gambling task involving an unpredictable monetary reward task, previously shown to elicit striatal dopamine release in healthy volunteers (Zald et al., 2004). Those patients with pathological gambling showed reduced dopamine receptor levels at baseline coupled with a greater displacement of raclopride, indicative of endogenous dopamine release, in response to the gambling task. However, while this combined effect may account for both the hypo- and hyperarousal aspects of gambling behavior, it is unclear how to reconcile the phasic component with the fMRI evidence of blunted activation of the mesolimbic reward system (Reuter et al., 2005). One problem in this literature, which is possibly intractable, is that the level of trial-by-trial monetary reward offered to gamblers in these experiments is trivial compared to the amounts these subjects may typically wager in their gambling sessions. This may have a pervasive effect on their engagement with the task. A related issue that applies primarily to

decision-making tasks is that gamblers' extensive experience in handling complex probabilities, coupled with the presence of distorted beliefs about chance and probability, may substantially alter their patterns of brain activation during such tasks in a manner that need not be indicative of a generalized endophenotypic marker for addiction.

Research into the neurochemical basis of pathological gambling has not focused exclusively on dopamine; other studies have assessed markers of serotonin, noradrenaline, opiate, and glutamate transmission in particular, with numerous lines of preliminary evidence for dysregulation (e.g., Grant et al., 2007; Nordin & Eklundh, 1999; Roy et al., 1988). These studies are reviewed in more detail in the chapter on treatment approaches.

Conclusion

Pathological gambling is a reasonably prevalent ICD seen in approximately 1%–2% of the population. It is clear that gambling-related harms often exist in gamblers who do not meet the full DSM criteria for pathological gambling, and if one accepts a more liberal threshold for disordered gambling, prevalence estimates rise accordingly to 2%–5%. As a psychiatric entity, pathological gambling co-occurs frequently with a range of other mental health problems including mood and anxiety disorders, others ICDs, and antisocial personality disorder, but the etiological overlap with the substance use disorders is particularly evident. The diagnostic entity of pathological gambling also comprises much heterogeneity, in terms of the plethora of psychological differences among the common gambling games, and also the differing motivational factors that appear to drive play in different pathological gamblers.

In reviewing the etiological processes that have been proposed in pathological gambling, I began by describing the basic paradox of gambling behavior: in all gambling games, the long-term expected value of gambling is negative. Some general appreciation of this state of affairs is indicated by the popular saying “the house always wins.” But if people truly understand the mathematics of the gambling situation, why is gambling such a widespread recreational activity in most cultures, and why does a significant minority across these cultures show excessive gambling? The two approaches that psychologists have taken to resolve this question posit that either there is some additional utility to gambling beyond mere financial considerations, or alternatively, that gamblers fail to perceive accurately their chances of winning. (These approaches are not mutually exclusive,

and both situations may occur.) The main candidate for the additional utility is the experience of some thrill or excitement during gambling, which is likely to be acquired through a combination of Pavlovian and instrumental learning processes. This subjective excitement is manifested in physiological arousal (e.g., heart rate increases). However, while there is unequivocal evidence for such arousal during gambling play in naturalistic settings, research has failed to unanimously show a change in the degree of arousal in individuals with disordered gambling. There is also considerable support for the second approach, with many studies showing that gamblers experience cognitive distortions in the way they process probability, randomness, and skill during gambling. These distortions may be restricted to (or accentuated within) the gambling context, and there is now reasonable evidence that these distortions are more frequent, or somehow more severe, in those with disordered gambling.

Three further sets of vulnerability factors were considered: (1) the evidence for changes in personality traits such as impulsivity, (2) the evidence for changes in cognitive and neuropsychological functions such as impulse control or risky decision making, and (3) the evidence for neurobiological alterations, for example in the integrity of the vmPFC or in dopamine neurotransmission. In each case, the research evidence is encouraging, although far from definitive. It is important to bear in mind that these vulnerability mechanisms represent different levels of explanation and are fully compatible with one another in principle. For example, a genetically mediated deficit in dopamine transmission may lead to abnormal development of the vmPFC region, with a concomitant influence on the individual's personality and circumscribed disruptions of cognitive function. Moreover, these vulnerability markers may moderate either the excitement and physiological arousal that are engendered by gambling or the susceptibility to cognitive distortions during gambling play.

Recent work has therefore begun the complex process of forging links between these levels of explanation. For example, work from my own group has used functional neuroimaging (fMRI) to study the brain's response to gambling near misses. Near misses are non-win outcomes, and in games of chance, they provide no useful information about future chances of success. Nevertheless, gamblers often report that near misses are significant events that encourage further play (Reid, 1986). Within a conditioning account of gambling, a near miss may

represent a conditioned reinforcer that the gambler has learned to be predictive of a winning outcome (Kassinove & Schare, 2001). Alternatively, near misses may fuel cognitive distortions in gambling by encouraging the belief that the gambler has acquired some skill at the game (i.e., the illusion of control). Congruent with this cognitive formulation, we found that near misses only increased the desire to continue gambling if they occurred on trials where there was active personal involvement in the game (Clark et al., 2009). Using a simplified slot machine task, near misses were observed to activate brain reward circuitry (ventral striatum, insula, medial PFC) that also responded to monetary wins. The neural response to near misses in the insula cortex (an area known to play a key role in emotional arousal and representing a physiological state) predicted that the subjective effects of near misses would encourage further play, and also predicted the score on a questionnaire measure of cognitive distortions (Clark et al., 2009). A related line of research by Rogers and colleagues (Campbell-Meiklejohn et al., 2008) has looked at the neural responses that underlie decisions to loss-chase. Decisions to chase were associated with greater activity within brain regions linked to reward processing (vmPFC), whereas decisions to quit were associated with greater activity in regions linked to conflict processing (dorsal anterior cingulate). These authors speculated that in pathological gambling, the balance between these two systems may be altered, predisposing to a tendency to chase. Both of these fMRI experiments were conducted in healthy volunteers with little evidence of disordered gambling, and future studies will aim to corroborate these data in groups of pathological gamblers.

In recent decades, concerns about ecological validity have stunted progress in this area. Certainly, behavior captured by studying gamblers in natural settings is invaluable, and we may be unable to ever elicit responses of this magnitude in the laboratory. However, neuroimaging studies of gambling are only possible in a research environment, using computerized tasks, and I would argue, moreover, that the complex interplay of psychological processes present in gambling games can only be dissected using simplified, and precisely controlled, laboratory models. A new generation of studies that combine physiological, behavioral, and subjective measures during gambling tasks promise to provide important developments in our scientific understanding of gambling behavior, and

our clinical understanding of how individuals move from healthy, controlled, recreational gambling to disordered behavior. Such an understanding will carry fundamental implications for treatment of pathological gambling and, ultimately, prevention.

Acknowledgments

Supported by research grants from the Economic and Social Research Council (U.K.), the Responsibility in Gambling Trust, and the Medical Research Council (U.K.), and completed within the Behavioural and Clinical Neuroscience Institute, which is funded by a consortium award from the Wellcome Trust and the Medical Research Council.

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Phenomenology and Epidemiology of Trichotillomania

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Abstract

Trichotillomania is a psychiatric disorder characterized by recurrent hair pulling, leading to hair loss and functional impairment. This chapter reviews the phenomenology and epidemiology of trichotillomania, and considers its relationship with putative obsessive-compulsive spectrum conditions and other body-focused repetitive behaviors. Salient animal models of the disorder, along with findings in human patients using neuroimaging and cognitive probes, are summarized. A brain-based model of trichotillomania is formulated, focusing on affect dysregulation, addiction, and impulse dyscontrol. Finally, the chapter flags cardinal questions for the attention of future clinical and research scrutiny.

Keywords: impulsivity, compulsivity, habit, obsessive-compulsive, cognition

Introduction

Trichotillomania (from the Greek *thrix*, for “hair”; *tillein*, “to pull”; and “mania”) is a psychiatric disorder characterized by repetitive hair pulling, leading to noticeable hair loss and significant functional impairment. Trichotillomania is currently classified as an impulse disorder according to the *Diagnostic and Statistical Manual* (DSM-IV-TR; APA, 2000). Diagnostic criteria emphasize growing tension before the act of pulling and relief, gratification, or pleasure afterward. However, many patients with clinically significant hair pulling do not endorse these criteria. Patients with trichotillomania are often highly secretive and go to great lengths to conceal bald patches—by, for example, using hair extensions, shaving, wigs, bandannas, and makeup. The embarrassment affiliated with the condition can cause patients to become socially withdrawn and shun close relationships. Often, patients with pathological hair pulling do not approach clinicians until the onset of more socially known and accepted Axis I comorbidities, such as major depressive disorder.

Unlike several other Axis I psychiatric disorders that have been surveyed on a relatively large scale, such as obsessive-compulsive disorder (OCD), there exist no populationwide studies of trichotillomania. As such, rates of occurrence in the background population—in particular countries—are not known. Based on a college student survey (~2500 participants) conducted in the United States, the lifetime prevalence of pathological hair pulling was estimated at 0.5%–3%, depending on how strictly the diagnostic criteria (DSM-IV) were applied (Christenson et al., 1991b). Of course, college students may not be representative of patients with the disorder at large. Duke and colleagues conducted a citywide questionnaire based survey of hair pulling in ~800 subjects from a southeastern college city in the United States (Duke et al., 2009). They reported prevalence rates of 0.6%–1.2%, again depending on the strictness of the applied criteria. These studies both reported higher rates of pathological hair pulling in females, and indeed, the overwhelming majority of recruits coming forward for clinical and

research trials have been females—usually five or more times as many females as males (Chamberlain et al., 2007c). The typical age of onset of pathological hair pulling is around 12–13 years. It is worth noting that hair pulling in young children may be regarded as a distinct, relatively benign clinical entity that tends to resolve of its own accord (Swedo & Rapoport 1991). Survey data suggest that most adult patients with trichotillomania have never received treatment and that sufferers consider treatments received to have been generally ineffective (Cohen et al., 1995).

This chapter considers trichotillomania from several perspectives, starting with historical accounts of hair pulling and moving on to present-day considerations. I review what is known of the epidemiology of hair pulling across species, the relationships between trichotillomania and other conditions (i.e., nosological issues), and findings from the neurosciences. I also describe a neurobiologically based model of the condition that may be useful and flag salient questions for future clinical and research work.

Historical Perspective

The act of pulling out one's hair, often in frustration, is recognized in common parlance today, and in fact the phenomenon itself has been recognized for many years, dating back to work attributed to Hippocrates (Christenson & Mansueto 1999). Thus, in *Epidemics I*, it was recommended that clinicians should examine whether a patient plucks out his own hair as part of the medical examination. References to plucking out one's own hair are found in diverse literature, including the Old Testament (Book of Ezra) and Shakespeare's *Romeo and Juliet*. The phenomenon of hair pulling is thus very much culturally bound. The first modern conception of trichotillomania as a stand-alone medical entity was formulated by the French dermatologist Hallopeau, who actually coined the term in the nineteenth century (Hallopeau, 1894). More recently, trichotillomania was incorporated into the *Diagnostic and Statistical Manual III Revised* (DSM-III-R) in 1987 and now stands as an impulse control disorder not elsewhere classified in the DSM-IV, alongside such conditions as pyromania, kleptomania, and intermittent explosive disorder (APA, 2000). Diagnostic criteria in the DSM-IV include repetitive hair pulling leading to noticeable hair loss and significant functional impairment; not due to another medical (e.g., dermatological) or psychiatric condition (e.g., delusions of parasitosis); tension before pulling or

when resisting; and pleasure, gratification, and/or relief when pulling out hair.

Etiology

The heritability of mental disorders is variable, with some loading more heavily on genetic influences (e.g., bipolar disorder) and others loading relatively on environmental factors (e.g., posttraumatic stress disorder). Virtually nothing is known of the role of genetic and environmental factors, and their interplay, in the manifestation of trichotillomania. Genetic influences are usually assessed, in the first instance, using twin studies. In a recent seminal Internet-based study conducted in 24 twin pairs of patients with trichotillomania, concordance rates for monozygotic and dizygotic twins were estimated at 38% and 0%, respectively. This, together with the finding that trichotillomania often runs in families, strongly suggests underlying genetic contributions to the manifestation of this debilitating, hidden condition. There have been almost no genetic studies of the disorder. The *SLTRK1* gene is involved in neural development, and specific mutations in this gene were identified in two patients with trichotillomania, while no such mutations were found in 2000 control subjects (Zuchner et al., 2006). These exciting results suggest that simple point mutations can, in some instances, result in complex phenotypes, that is, the development of trichotillomania. Interestingly, mutations in this gene have also been implicated in Tourette's syndrome (Abelson et al., 2005), another disorder characterized by inappropriate release of motoric "habits."

Phenomenological Considerations I: The Disorder Itself and Epiphénoména

Hair pulling in trichotillomania can affect any body site with hair, but the majority of sufferers pluck at their own hair from the scalp, eyebrows, and/or eyelashes. A minority of sufferers may pull hair from pets, relatives, or toys, but this is debated. At a given point in time, hair pullers tend to have two or three preferred sites, usually plucking hairs one at a time. Hair pulling often co-occurs with other putative "grooming" habits, notably skin picking and nail biting (Grant & Christenson, 2007). In milder forms, all these behaviors can be considered part of the grooming repertoire, which occur across different animal species. The difference between these normal grooming habits and the symptoms of—for example, trichotillomania—lies in the time spent engaging in the activities, the damage done, and the interference in one or more areas

of occupational functioning (APA, 2000). For trichotillomania itself, patients report reduced work productivity, disrupted family life, difficulty forming and maintaining close social relationships, and interference in sports through avoidance of such activities (Diefenbach et al., 2005). While trichotillomania benefits from a formal position in DSM-IV, pathological skin picking and pathological nail biting do not, although this situation may change with the upcoming DSM-V revision. In addition to the co-occurrence of other grooming disorders alongside trichotillomania, comorbidity with Axis I disorders should be regarded as the norm rather than the exception. In one study comprising outpatients with chronic pathological hair pulling, 82% met criteria for one or more past or present Axis I disorders besides trichotillomania, most frequently depression, OCD, other anxiety disorders, and substance abuse (Christenson et al., 1991a).

It has been proposed that hair pulling can be broadly categorized as *focused* (as opposed to *non-focused*) and/or *automatic* (versus *nonautomatic*; Flessner et al., 2009). Focused hair pulling has a compulsive tinge, with patients consciously pulling out particular hairs, often with rules about the preferred texture and site, engaging in rituals such as counting hairs, plucking at the bulb roots, or ordering them up. Automatic hair pulling occurs while sufferers are in a trance-like or dissociative state—such as when watching TV or driving—with patients describing suddenly moving out of this state and realizing, with regret, the damage done. It should be noted that most patients exhibit a combination of hair-pulling types in differing degrees. Flessner et al. conducted an Internet-based survey of ~1500 subjects and found that high levels of focused pulling and high levels of automatic pulling were both individually associated with more severe disease and functional impairment in general (Flessner et al., 2008). Elsewhere, Flessner and colleagues used a cross-sectional design (with ~1500 subjects) and found that focused pulling fluctuated considerably over time and was influenced by psychological distress and pubertal onset; they also found that automatic pulling was relatively stable temporally (Flessner et al., 2009).

Trichotillomania has been associated with several medical complications, although these are thought to be relatively rare. A significant proportion of patients with trichotillomania, some 15%, indulge in hair consumption, termed *trichophagia*, which in rare cases can result in gastrointestinal obstruction due to the development of a hair ball (*trichobezoar*).

In case reports, other physical consequences of trichotillomania have been reported, including repetitive stress injury (RSI), infections of pulled sites, and carpal tunnel syndrome (Enos & Plante 2001; Frey et al. 2005; O'Sullivan et al., 1996).

Similarities in the phenomenology of trichotillomania and OCD led to the notion that trichotillomania represents a candidate member of an “OC spectrum” of conditions, which are collectively associated with repetitive habits that are difficult to suppress (Bienvenu et al., 2000; Christenson & Crow, 1996; Hollander & Rosen, 2000; Stein et al., 1995; Swedo & Leonard, 1992). Indeed, the gold standard instrument for assessing OCD severity, the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), includes trichotillomania in its symptom checklist (Goodman et al., 1989a, 1989b), reflecting an early clinical realization that these two symptoms share a relationship. Obsessive-compulsive spectrum disorders may share overlap in terms not only of phenomenology, but also neurobiology (see later), comorbid expression, treatment response, and underlying etiology. First-degree relatives of patients with OCD have been found to be at increased risk of aberrant grooming behaviors (Bienvenu et al., 2000). There is likewise some evidence of increased rates of OCD in first-degree relatives of trichotillomania patients (Lenane et al., 1992).

Another proposal concerning how to conceptualize trichotillomania, suggested by Stein and colleagues (Stein et al., 2007, 2008), is that clinically debilitating, body-focused, repetitive behaviors be themselves placed into a single nosological category. Thus, hair pulling in trichotillomania would be considered alongside other repetitive behaviors such as body rocking, self-biting, and skin picking.

Phenomenological Considerations II: Animal Models

Animal models have been used with success to facilitate translational approaches to understanding a variety of psychiatric disorders. The relative specificity and overt behavioral nature of the symptoms of trichotillomania render the condition particularly suited to animal modeling. The symptoms of trichotillomania are thus accessible to animal modeling in a way that intrusive thoughts in OCD, or (to a lesser extent) complex rituals in OCD, are not. Many animal models of trichotillomania have been described over the years (see Moon-Fanelli et al., 1999). Animal models of trichotillomania can, in general terms, be labeled as either (1) ethological, involving spontaneously arising stereotyped behaviors

(e.g., fur chewing), such as during times of sensory deprivation or stress, or (2) laboratory-based, involving behavioral patterns induced by genetic and/or pharmacological manipulations.

Perhaps the most fruitful ethological model of trichotillomania to date is that of barbering (fur and whisker trimming) in mice (see Garner et al., 2004, for review). Barbering occurs spontaneously in a subgroup of mice, and involves plucking whiskers and/or body hair mostly from other mice but also, to a lesser extent, from themselves. Garner and colleagues have described similarities between barbering in mice and the human manifestation of trichotillomania. Notably, barbering mice tend to remove hair from cagemates in an idiosyncratic pattern; barbering is more common in female mice; barbering becomes increasingly common with advancing age; the youngest barber found in the research was pubescent; and marked differences in the prevalence of barbering were found between strains of mice, suggesting underlying genetic contributions.

Of the available laboratory-based models of trichotillomania, the two most prominent are perhaps the *Hox-B8* gene model developed by Greer and Capecchi (2002) and the *SAPAP3* mutant mice model developed by Welch and colleagues (2007). *Hoxb8* is a gene involved in early development, including development of the central nervous system. Mice with disruption of this gene exhibit pathological grooming behavior; they inappropriately remove hair, spend more time grooming than controls, initiate grooming sequences more often than controls, and excessively groom their wild-type cagemates (Greer & Capecchi 2002). *SAPAP3* is a scaffolding protein involved in neural development and is expressed in high proportions in the striatum. Mice with deletion of this gene exhibit compulsive grooming behavior leading to skin lesions; interestingly, these behaviors are ameliorated by treatment with the selective serotonin reuptake inhibitor fluoxetine—a first-line treatment for OCD (albeit not necessarily effective in treating human trichotillomania; see the chapter titled “The Assessment and Treatment of Trichillomania”). Furthermore, variation in the human *SAPAP3* gene was found to be nominally associated with one or more grooming disorders (genotypic relative risks: 1.6–3.3) in a study of ~400 families thoroughly phenotyped for OCD-related research purposes (Bienvenu et al.; 2009).

These animal models of trichotillomania represent a rich but largely untapped vein for exploring

the neurobiology of the disorder (see Moon-Fanelli et al., 1999). Several important questions remain to be addressed. Do animal models of trichotillomania respond to particular pharmacological treatments? What particular genetic polymorphisms are associated with barbering? Do animals exhibiting these grooming pathologies also manifest neurobiological dysfunction akin to that found in patients with trichotillomania, such as abnormal structure and function of cortico-subcortical circuitry and specific cognitive deficits?

Neuroscientific Study of Trichotillomania

Scant neuroscientific research has been undertaken in patients with trichotillomania (see Chamberlain et al., 2009, for detailed review). This section focuses on several key studies that have used neuroimaging and objective neurocognitive tests dependent on the integrity of frontostriatal circuitry.

Structural and functional brain imaging techniques have been deployed with success in the study of OCD, which has been linked to abnormalities of the orbitofrontal cortices, posterior parietal cortices, and basal ganglia (particularly the caudate; Menzies et al., 2008). By contrast, few imaging studies have been undertaken in patients with trichotillomania. Based on the finding of caudate structural abnormalities in OCD, and on the notion of possible overlap between the neurobiology of trichotillomania and OCD, Stein and colleagues explored caudate volumes in patients with trichotillomania using magnetic resonance imaging (MRI); however, they did not detect any abnormalities versus controls (Stein et al., 1997). In another study, patients with trichotillomania showed reduced left frontal gyrus and increased right cuneal cortex volumes versus controls (Grachev, 1997) and smaller left putamen volumes versus controls (O’Sullivan et al., 1997). Keuthen and colleagues, using parcellation techniques, identified reduced cerebellar volumes in trichotillomania patients compared to controls (Keuthen et al., 2006).

In addition to these structural imaging techniques, there have been a few functional imaging studies of the disorder. Using positron emission tomography (PET), Swedo and coworkers identified increased metabolic activity at rest in patients with trichotillomania across multiple brain regions (Swedo et al., 1991). The only published functional MRI (fMRI) study in patients to date found no significant differences in brain activation in patients with trichotillomania compared to controls during a sequence learning task (Rauch et al., 2007).

Most of the above-described studies relied on region-of-interest approaches, often not correcting for the vast swathe of multiple comparisons involved when considering the human brain. One recent trichotillomania study explored brain structure using MRI in conjunction with statistical techniques enabling distributed abnormalities in gray matter to be mapped across the whole brain. Such an approach is valuable for the study of psychiatric disorders, which are likely to involve distributed subtle developmental abnormalities across multiple regions rather than solitary overt point lesions. In comparison to matched controls, patients with trichotillomania showed abnormally elevated gray matter densities in bilateral frontal regions, the bilateral cingulate cortex, the left amygdalo-hippocampal formation, and the left putamen (Chamberlain et al., 2008; Figure 9.1). It should be noted that these represent average differences between the groups, and that follow-up work is needed to clarify whether these gray matter abnormalities reflect vulnerability markers or rather perhaps reflect changes due to the undertaking of the repetitive habit itself (neuronal rewiring). In the same sample of patients and controls it was also shown, using a technique known as *diffusion tensor imaging* (DTI), that the white matter tracts connecting salient brain regions exhibited lower integrity in patients than in controls (Chamberlain et al., unpublished findings).

Cognitive testing allows researchers and clinicians to probe the integrity of frontostriatal circuitry in a relatively convenient and cost-effective manner.

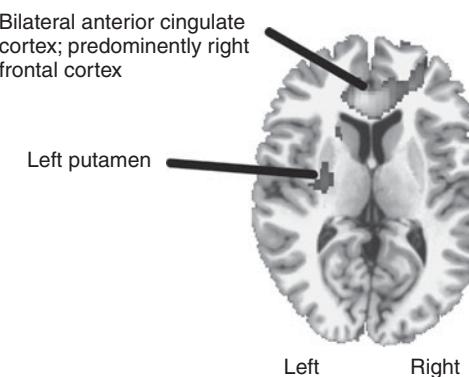


Fig. 9.1 Compared to controls, patients with trichotillomania showed abnormally increased gray matter density across multiple neural regions including the putamen, amygdalo-hippocampal formation, anterior cingulate, and frontal cortices. This figure shows a representative template brain slice with clusters of significantly abnormal brain structure superimposed, based on data reported in Chamberlain et al. (2008).

Given the evidence of structural brain abnormalities in patients with trichotillomania surveyed above, and the disinhibited nature of the symptoms, it is arguably to be expected that patients would also show deficits on cognitive tests tapping frontostriatal brain circuitry. The first cognitive study in the trichotillomania literature used the Stylus maze test, in which subjects learn to navigate across a peg board using a stylus. Patients with trichotillomania were impaired across multiple measures compared to the controls (Rettew et al., 1991). In a different study using a somewhat similar test, no deficits were found (Coetzer & Stein, 1999). Stanley and coworkers found evidence for impairments on several measures of divided attention in patients (Stanley et al., 1997).

Objective computerized tests offer potential advantages over traditional pen/paper cognitive tests in terms of translational modeling. Such tests were used to assess various aspects of cognition in patients with trichotillomania compared to patients with OCD and healthy controls. Patients with trichotillomania, in the absence of comorbidities such as depression, showed a relatively intact neurocognitive profile, exhibiting selective deficits on a test measuring the ability to suppress or inhibit impulsive responding (the stop-signal reaction time [SSRT] task; see Figure 9.2) (Chamberlain et al., 2006a, 2007b, 2006b). From separate translational research, it is known that inhibitory performance

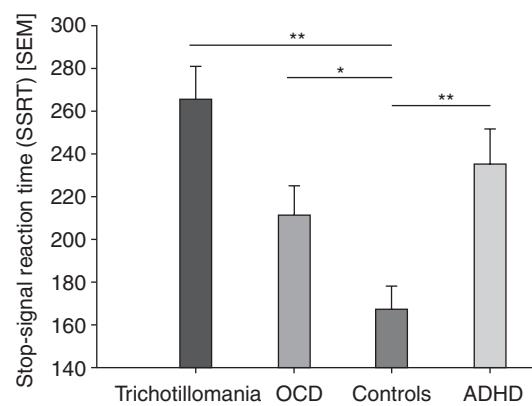


Fig. 9.2 Performance on the stop-signal reaction time (SSRT) task across key clinical groups. Left three bars: patients with trichotillomania showed significantly impaired SSRT versus patients with OCD and controls; OCD patients were impaired versus controls (Chamberlain et al., 2006b). Right-hand bar, for visual comparison, performance of adults with ADHD (Chamberlain et al., 2007a). * $p < 0.05$ impaired versus controls, ** $p < 0.01$ impaired versus controls. Figure reprinted from Chamberlain et al. (2009) with permission of Elsevier.

on this task is dependent on a right-lateralized neural network including the right frontal gyrus and its subconnections. It is also known that inhibitory control on this task is under probable noradrenergic control. These findings suggest a new treatment trial stream for trichotillomania using agents with putative noradrenergic properties (Chamberlain et al., 2009). In contrast to the relatively profound deficit manifested on the SSRT, patients with trichotillomania showed a relatively intact cognitive profile across other domains, while patients with OCD exhibited problems across several tasks, including those measuring cognitive flexibility (set-shifting and sequence strategy use tests). These findings, viewed together, suggest cognitive dysfunction in patients with trichotillomania to be relatively restricted compared to those with OCD.

Toward a Brain-Based Model of Trichotillomania

Clearly, trichotillomania is a heterogeneous disorder and—in contrast to other conditions, such as OCD—has received relatively little research attention. Any neurobiological model of the disorder must be viewed with these limitations in mind. Stein and colleagues have suggested an *ABC* model of trichotillomania emphasizing *Affect dysregulation, Behavioral addiction, and Cognitive dyscontrol* (Stein et al., 2006).

A role for affect dysregulation in the manifestation of trichotillomania is suggested by the high rates of comorbidity with anxiety and mood disorders; it may well be the case that individuals engage in hair pulling to distract themselves from distressing negative cognitions. As described previously, there is evidence from the brain imaging literature for gray density abnormalities of the amygdala in trichotillomania patients (Chamberlain et al., 2008). It is known that the amygdala is heavily implicated in emotional regulation in health, and in the context of other disorders such as depression and anxiety (Phelps & LeDoux, 2005). In one study that sought to characterize trichotillomania cue profiles, it was found that hair pulling was influenced by emotional states and sedentary activities (Christenson et al., 1993). Using self-report measures, another study showed that hair pulling was initially associated with decreases in boredom, sadness, anger, and tension, and with subsequent increases in guilt, sadness, and anger (Diefenbach et al., 2008).

In common with other conditions currently classified as impulse control disorders (e.g., compulsive

gambling), behavioral addiction may also play a role in trichotillomania. The impulse control disorders, such as trichotillomania, share overlapping features with substance dependence, including repetitive behaviors that are associated with strong urges, and a sense of gratification, relief, or pleasure during/after the pathological act (Brewer & Potenza 2008). Several neural regions shown to be structurally abnormal in the previously described trichotillomania brainwide study (Chamberlain et al., 2008) play a cardinal role in motivational drive and addiction. Notably, the amygdala is involved in assigning emotional significance to stimuli; the hippocampus is involved in mnemonic processing relevant to emotion; and the cingulate cortices monitor behavior and facilitate aspects of cognitive control (Brewer & Potenza, 2008). This “addiction” approach to understanding trichotillomania is not simply academic; it has led to novel treatment trials using agents targeting addictive circuitry (the nucleus accumbens and glutamatergic neurotransmission; Grant et al., 2009). See the chapter “The Assessment and Treatment of Trichillomania” for a discussion of treatment trials.

The repetitive symptoms of trichotillomania suggest—*prima facie*—underlying problems with behavioral regulation, that is, impulse dyscontrol. According to models of frontostriatal circuitry, the basal ganglia can be viewed as pattern generators driving motor behavior, while cortical regions serve to moderate (inhibit) these habits (Eblen & Graybiel, 1995; Graybiel, 1997, 1998; Graybiel & Rauch, 2000). As noted previously, trichotillomania patients in a whole-brain MRI study showed abnormally increased gray matter densities in the putamen and cortical regions, including the anterior cingulate. Furthermore, patients with trichotillomania showed impaired performance on an objective and well-validated neurocognitive measure of impulse control, the SSRT task (Chamberlain et al., 2006b). Several tiers of evidence implicate brain regions, notably the right frontal lobe and anterior cingulate cortex, in this ability to suppress motor responses (Aron et al., 2007; Rubia et al., 2003). The SSRT deficits in patients with trichotillomania are arguably consistent with abnormalities in one or more of these brain regions. The issue of brain dysfunction in patients with trichotillomania, for example during impulse control, could be addressed more directly by using a functional imaging version of cognitive tasks that has been shown to be sensitive to pathology in other neuropsychiatric disorders such as ADHD (Rubia et al., 2005).

Conclusion

Trichotillomania is a relatively neglected, often hidden, condition associated with repetitive hair pulling leading to noticeable hair loss, substantial functional impairment, and distress. Despite having an estimated lifetime prevalence of 0.5%–3%, little research funding and attention have been directed to elucidating the etiology, phenomenology, and neurobiology of this condition. Point genetic mutations have been implicated in the manifestation of trichotillomania in some individuals, and the only twin study of its kind to date implicated genetic factors in its etiology. Neurobiological factors implicated in the manifestation of trichotillomania include affect dysregulation, addiction, and cognitive dyscontrol. Given the heterogeneity of the disorder, it is probable that these three factors play different roles, depending on the particular patient being considered. Trichotillomania presents commonly with mood disorders, anxiety disorders, and conditions of the putative OC spectrum such as OCD, along with body-focused repetitive behaviors like nail biting and skin picking. Trichotillomania, by virtue of its overt motoric symptomatology, is suited for animal modeling, and several models have been explored, including barbering and induced *HoxB8* genetic mutations. It is hoped that multi-tiered translational approaches between disciplines will further our understanding of trichotillomania and engender improved treatments for this and related candidate OC spectrum disorders.

Future Research Directions

- What are the genetic predisposing factors to trichotillomania? In a typical case, are there many genes, each conferring a small effect, or a few substantive mediators (the “holy grail”), such as genes involved in neural development?
- Do patients with trichotillomania exhibit functional as well as structural brain abnormalities (e.g., using fMRI)?
 - What is the longitudinal course of trichotillomania in terms of symptoms, brain structure, and cognitive function?
 - To what extent do animal models of trichotillomania (e.g., barbering, *Hoxb8*, *SAPAP*) map onto findings in human patients with the disorder in terms of abnormal brain structure and function, cognition, and treatment response?
 - How should trichotillomania be categorized—as an impulse control disorder, an OCD, or a body-focused repetitive disorder?

- Can medications known to target other forms of behavioral addiction and impulse dyscontrol be shown to be useful in the treatment of trichotillomania?

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Phenomenology and Epidemiology of Kleptomania

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Abstract

Stealing behaviors have been reported since the eighteenth century. However, only in 1952 was the phenomenon included in the *Diagnostic and Statistical Manual of Mental Disorders*. While shoplifting and kleptomania share the same behavior, the motives behind each behavior differ significantly. Kleptomania is characterized as an uncontrollable urge to steal, accompanied by mounting stress before committing the theft, followed by relief, guilt, and shame. Kleptomania seems to be a more female-dominant disorder, and the exact causes remain unknown. The explanations are broad, stretching from stressful childhood and low self-esteem to head trauma. It seems to be chronic, with exacerbations and remissions. Treatments vary from psychological approaches suggesting psychotherapy to pharmacological interventions with various medications. A combination of the two had been found to be the best treatment strategy. With the right diagnosis and treatment, there is hope for bettering the quality of life of people suffering from kleptomania.

Keywords: kleptomania, stealing, shoplifting, pharmacotherapy, Cognitive Behavioral Therapy, SSRI, opioid receptors.

History

Stealing behavior and shoplifting date back centuries. In the eighteenth century, Franz Joseph Gall, a German neurophysiologist, pointed out that King Amadis of Sardnia habitually stole trifles from others (Polak & Dannon, 2001). The idea that some people might not be able to control their impulse to steal was first published in the medical literature in 1816 by Mattley and was named *Klopemanie* (which means “stealing madness” in Greek; Polak & Dannon, 2001).

This phenomenon was seriously examined in 1838 by the French physicians Jean-Etienne Esquirol and C. C. Marc, who coined the term *kleptomania* by which they aimed to describe shoplifting that is involuntary and irresistible.

By the end of the nineteenth century, explanations of the phenomenon referred to uterine diseases or premenstrual tension as possible causes of klepto-

mania, due to the perception that such behaviors only affected women. Later on, men were shown to suffer from kleptomania as well, and by the early twentieth century, the female reproductive system as the cause for this behavior was discarded along with virtually all clinical and research interest in the disorder (Grant & Odlaug, 2008).

DSM

In 1952, the first *Diagnostic and Statistical Manual of Mental Disorders* (DSM-I) included *kleptomania* as a supplementary term for *obsessive-compulsive reaction* rather than as a distinct diagnosis. However, kleptomania was ignored altogether in the DSM-II (1968). It reappeared only in the DSM-III (1980) and has been categorized ever since as an impulse control disorder not elsewhere classified (DSM-IV, 1994). The *ICD-10 Classification of Mental and Behavioral*

Diseases of the World Health Organization classifies it under the heading of habit and impulse disorders, together with pathological gambling, pyromania, and trichotillomania (ICD-10; World Health Organization, 1992). Both classification systems are based on recurrent failure to resist the impulse to steal, despite the ego-dystonic nature of the impulse and the awareness of the wrongfulness of the act.

The DSM-IV-TR (DSM-IV-TR, 2000) defines kleptomania by the following criteria:

1. Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value.
2. Increasing sense of tension immediately before committing the theft.
3. Pleasure, gratification or relief at the time of committing the theft.
4. The stealing is not committed to express anger or vengeance and is not in response to a delusion or hallucination.
5. The stealing is not better accounted for by another disorder.

The literature describes a typical person suffering from kleptomania as a married woman with recurrent, intrusive, and irresistible urges to steal (see Table 10.1; Grant & Kim, 2002a). This typical characterization possibly derives from the fact that current knowledge about kleptomania largely derives from case reports, series, and patients selected through the medical or legislative system (Grant & Kim, 2002a).

The prevalence of kleptomania in the U.S. general population today is unknown but has been estimated at 6 per 1000 people (Aboujaoude et al., 2004), which translates into 1.2 million people out of the 200 million American adults.

Comorbidities

Although currently included in the impulse control disorders chapter of the DSM, kleptomania as a distinct diagnostic entity has often been challenged. Many continue to see it as a symptom of other psychiatric disorders. In general, people suffering from kleptomania who seek treatment tend to have higher rates of personality disorders than do members of nonclinical populations (Grant, 2004), which only strengthen the belief that kleptomania is indeed just a symptom. However, a study from 2004 (Grant, 2004) suggests that kleptomania is a phenomenon independent of any particular personality disorder. In addition, the presence of a personality disorder does not appear to affect the overall severity of the

Table 10.1 Demographic Characteristics—Average of Data from Four Studies

Characteristics	Value
Gender	
Male (range)	29% (0–41.9)
Female (range)	71% (58.1–100)
Age	
Mean (SD)	40.5 (12.3)
Range	36.7–45.6
Marital status	
Married (range)	52.12% (40–63)
Single (range)	24.05% (16–27.5)
Widow/separated/divorced (range)	23.83% (21–26.7)
Employment status	
Employed	66.4%
Unemployed	33.6%

Source: Compiled from Aboujaoude et al. (2004), Dannon et al. (2004), Grant (2003), and Grant et al. (2007).

kleptomania symptoms. On the contrary, kleptomania should not be diagnosed if stealing appears secondary to antisocial personality disorder.

Additional infrequently reported symptoms included nail biting, bed wetting, sleepwalking, and altered mental states at the time of the stealing—including dissociation, absentmindedness, and mild confusion states due to physical illness or medication side effects (McElroy et al., 1991a). However, people suffering from kleptomania present a specific psychopathological profile that distinguishes patients with this disorder from patients with alcohol abuse or dependence and other psychiatric comparison patients (Baylé et al., 2003). Most people suffering from kleptomania appear to suffer from a current or past major mood disorder (see Table 10.2; Grant & Kim, 2002a).

McElroy (1991) states that the most striking finding of her study was that all patients suffering from kleptomania demonstrated substantial psychopathology in addition to kleptomania—most notably major mood disorder, but also anxiety disorders, alcohol and other psychoactive substance use, eating disorders, and other impulse control disorders (McElroy et al., 1991b).

Table 10.2 Comorbidity of Kleptomania and Other Psychiatric Disorders in 11 Patients

Disorder	%
Bipolar disorder	27
Recurrent depression	45
Generalized anxiety	9
Social phobia	9
Compulsive buying	18
Trichotillomania	9
Impulse control disorders not otherwise classified	9
Alcohol abuse or dependence	9
Nicotine use or dependence	36
Bulimia	18
Paraphilia	9
Histrionic personality disorder	18
Nonspecified personality disorder	36
Suicidal behavior	36

Source: Compiled from Baylé et al. (2003).

Kleptomania also appears to have many phenomenological similarities to *substance abuse and addiction* such as urges or cravings, tolerance, withdrawal, repeated successful attempts to cut back or stop, and impairments in areas of life functioning (Grant et al., 2009).

The possible association between kleptomania and *depression* was reported by Janet (1911) in the early twentieth century. The mood disorder may precede or appear concurrently with the onset of kleptomania. In severe cases, depression may lead to self-injurious behaviors and even suicide. Some individuals report relief from depression or manic symptoms after stealing (Dannon et al., 2004).

The average data findings of five studies (Aboujaoude et al., 2004; Dannon et al., 2004; Grant, 2003; Grant & Kim, 2002b; Grant et al., 2007) show the first five comorbidities most prevalent in patients with kleptomania (see Table 10.3).

Kleptomania versus Typical Shoplifting

Shoplifting and kleptomania might have resembling results (stolen items), but there is a fundamental difference between these two acts. While shoplifting is defined as taking something from

Table 10.3 Comorbidities as Recorded in Five Studies of Kleptomania

Comorbid Lifetime Disorders	Value
Major depressive disorder, % (range)	34.94% (26.7–45.5)
ADHD	9% (7.5–10.5)
Generalized anxiety disorder	7.1% (5–9.7)
Alcohol abuse	19.6% (7.5–33.3)
Obsessive-compulsive disorder	7.9% (5–11)

a store without paying for it, independent of intent or motivation, kleptomania is a psychiatric disorder characterized by the inability to resist recurrent impulses to steal objects that are not needed (Grant et al., 2009). The vast majority of shoplifters are described as amateurs with sporadic activity, with no known history of criminal activity, who steals for their own consumption rather than for resale. Rates of kleptomania among people who are arrested for shoplifting have ranged from 0.1% to 8%, and according to the DSM-IV-TR, fewer than 5% of shoplifters are identified as suffering from kleptomania (Grant, 2006). The distinction between shoplifters and people suffering from kleptomania may reside in evaluating the motivations for stealing (as reflected in the DSM-IV-TR criteria) instead of focusing on the behavior (Grant, 2004). In a 2002 study of 22 people suffering from kleptomania, all of them reported that they could afford the items they stole and that in the majority of cases, they did not understand why they stole the particular items they did (Grant & Kim, 2002b).

To conclude, shoplifters would usually steal something they need or want can cannot afford, or as an act of mischievous; people suffering from kleptomania would usually steal something they do not need or can afford, as a result of their inability to resist the urge to steal.

Stealing Behavior

The average age of onset of stealing behavior is typically during adolescence, although there are reports of this behavior starting as early as 4 years of age and as late as 77 years of age. Women usually present for evaluation at a younger age than men (Grant, 2006).

Individuals suffering from kleptomania report that the objects stolen usually are of little value and are affordable. After stealing the items, they usually

discard, hoard, secretly return, or give the items away. These individuals may avoid stealing when immediate arrest is likely, but the chances of apprehension are usually not fully taken into consideration. Although a sense of pleasure, gratification, or relief is experienced at the time of the theft, individuals describe a feeling of guilt, remorse, or depression afterward. These guilt feelings and depression are usually the result of the fundamental pain and humiliation of repeated arrests (Grant, 2006).

Legal Aspects

Many patients with kleptomania face legal difficulties due to their behavior. Studies have reported that 64% to 87% of people suffering from kleptomania have a history of being apprehended. In fact, one study found that patients reported a mean number of lifetime apprehensions of approximately three. Although most apprehensions do not result in jail time, early evidence suggests that 15% to 23% of people suffering from kleptomania have been jailed for shoplifting (Grant & Odlaug, 2008). People with kleptomania report suffering guilt due to their behavior and fear of being apprehended (Grant, 2004). Perhaps this is why, more often than not, individuals with this disorder do not approach psychiatric facilities voluntarily and are treated as recidivist offenders by the judicial system (Durst et al., 2001). Many people with kleptomania report that they did not come for treatment at an earlier date due to the fear that their treatment providers would notify the police (Grant, 2004).

The items usually stolen are diverse and include mostly household goods, groceries/food, clothing, tools/mechanical/games/toys/sport, toiletries, and books/music. The items are stolen mostly from stores, friends, and relatives (Grant & Kim, 2002b), and the act of thievery is usually carried out alone (Durst et al., 2001). Once a subject with kleptomania engages in a particular stealing activity, he or she tends not to steal from multiple locations (that is, steal from the same venue over and over again). Patients with kleptomania tend to be specific in what they steal and from whom (they would still the same kind of object from the same person). Usually, the type of item stolen does not change over the course of the illness, although the expense of particular items may increase. Patients reported that the increase in price was necessary to relieve the increasing sense of anticipatory tension they experienced shortly before thefts (Grant & Kim, 2002b). For some, the “rush” associated with the theft appears proportional to the monetary value of

the item. For others, the value of the stolen objects increases over time, suggestive of tolerance (Grant & Odlaug, 2008).

Etiology and Demographics

Previous research findings suggest that kleptomania is a rare condition (affecting 0.6% of the population).

Gender

Approximately 50% to 70% of reported kleptomania cases involve women. However, these data might be culturally influenced (Durst et al., 2001; Polak & Dannon, 2001).

Gender aspects of kleptomania have received little research focus. One study found that men with kleptomania are more likely to have a history of birth trauma. Men with kleptomania also appear less likely to suffer from co-occurring eating disorders, but they appear to have higher rates of co-occurring paraphilic behaviors (Grant & Odlaug, 2008). Goldman (1991) noted that men may develop more overtly antisocial behavior, whereas women develop other symptoms, such as hysterical behavior. Men and women with impulse control issues may also manifest these problems differently. Men seem to have a relative monopoly on the more aggressive acts of impulse such as pyromania, intermittent explosive disorder, and pathological gambling. Kleptomania and trichotillomania, the less overtly destructive impulse disorders, may be more common in women (Goldman, 1991).

The Course of the Disorder

Little is known about the course of the disorder, but it appears to be chronic, with exacerbations and remissions (Grant & Kim, 2002b).

Characteristics

Subjects with kleptomania score low on socialization, high on impulsivity, and high on novelty-seeking (Grant, 2004). Individuals who score high on novelty seeking are described as “curious, impulsive, quick-tempered and disorderly” (Grant & Kim, 2002a.). High novelty-seeking scores in kleptomania are consistent with the literature that has found high levels of impulsivity among kleptomaniacs. This impulsivity does not appear to be simply a consequence of another Axis I or II disorder. Subjects with kleptomania also have significantly higher harm-avoidance scores than control subjects, which is associated with “apprehension, shyness, pessimism and fatigue” (Grant & Kim, 2002a, p. 225).

Also, individuals with kleptomania often report a stressful childhood, marital conflicts, and lack of self-esteem. Thus, stealing may be a symptom of the personality difficulties that are often associated with these conflicts (Grant, 2004). According to psychoanalytic theories, kleptomania is the ego's defense against anxiety, suffering, child neglect, or an experience of emotional trauma (Allen, 1965). Kleptomania can also be traced back to a child's feelings of neglect concerning proofs of love. In stealing, the child finds a substitute for a lost pleasure and simultaneously takes revenge on those who caused his or her suffering (Goldman, 1991). In addition, stealing might be a symbolic expression of unwanted and unconscious conflicts and impulses, such as sexual impulses, castration anxiety, and penis envy (Goldman, 1991; McElroy et al., 1991a; Murray, 1992). Reports about sexual arousal or an orgasm during the theft, and reports about lack of psychosexual function in kleptomania, support theories that relate the phenomenon to the early stages of psychosexual development (Fenichel, 1945). In general, the condition is thought to be an expression of an underlying emotional disturbance rather than a form of neurosis itself (Grant, 2005).

Neurobiology

Other theories relate the onset of kleptomania to head trauma (Aizer et al., 2004), frontal lobe damage (Kozian, 2001), dementia (Mendez, 1988), and hypoglycemia secondary to insulinoma (Segal, 1976). According to Aizer et al. (2004), closed head injury is known to be associated with psychiatric disturbances including mood disturbances, apathy, emotional lability, and impulsive and aggressive behaviors.

Functional Impairment

Kleptomania causes severe suffering and significant impairment in the ability to function socially and occupationally. Many patients report intrusive thoughts and urges related to shoplifting that interfere with their ability to concentrate in their daily life activities. Others report missing work, often in the middle of the day, after leaving early to shoplift. It is not surprising that they also report significantly lower life satisfaction and poorer quality of life compared to a general nonclinical adult sample. Some patients have even considered suicide as a mean of avoiding shoplifting (Grant & Odlaug, 2008). Suicide attempts have been reported in 25% of people treated for kleptomania (Grant et al., 2009). Clinical observation has also suggested that

kleptomania may be associated with high levels of perceived stress. People who report high levels of perceived stress tend to view their lives as unpredictable and uncontrollable. Perceived stress in kleptomania appears to be a function of both the shame that follows each episode of stealing and the urges associated with the disorder. Because the urges may often have no identifiable trigger, patients with kleptomania live with a constant fear of perhaps being driven to perform behavior that they find reprehensible (Grant et al., 2003). Both the urges and the behavior are experienced as ego-alien, noncomprehensible, wrong, and dissonant with the basic personality of the perpetrator (Durst et al., 2001).

Diagnostic Obstacles

Even though kleptomania is associated with impaired functioning and a poor quality of life, there is some indication that both clinicians and researchers fail to screen for or diagnose the disorder. This failure may be due to the lack of DSM-based diagnostic instruments. In addition, kleptomania presents many difficult considerations concerning the differential diagnosis. According to Grant (2006), a diagnosis of kleptomania should only be made if all the inclusion criteria below are present (Grant et al., 2006):

1. There are recurrent impulses to steal items
2. There is recurrent failure to resist impulses
3. The stolen items are not needed for personal use or monetary value
4. There is an increasing sense of tension immediately before committing the theft
5. Pleasure, gratification, or relief is experienced at the time of committing the theft.

In addition, all of the following exclusion criteria exist (Grant et al., 2006):

1. The stealing is not committed to express anger or vengeance.
2. The stealing is not committed in response to a delusion or hallucination.
3. The stealing behavior not better accounted for by a manic episode.
4. The stealing behavior is not better accounted for by antisocial personality disorder.

Classifications

Lacassagne (1896) identified three subgroups of people suffering from kleptomania: (1) hoarders who accumulate stolen objects with no need or target; (2) impulsive and dysregulated individuals who act under the influence of irresistible sudden

impulses; and (3) organic or psychiatric patients with kleptomania as a symptom (Durst et al., 2001).

Dupouy (1905), on the other hand, distinguished among individuals with obsessive-compulsive urges whose acts are controlled by irresistible, “obsessive thoughts disorders,” those who act in an “automatic reflective manner,” and an intermediate group characterized by “morbid desire” (Durst et al., 2001).

Today, kleptomania is considered to be associated with one of the following groups, with the underlying assumption that medications that have benefited individuals with these disorders would be helpful in treating kleptomania.

The Affective Spectrum Model

Since a high comorbidity of affective disorders exists in people with kleptomania, the *affective spectrum* was proposed by Hudson and Pope (1990). They have found a relationship between kleptomania, obsessive-compulsive disorder, eating disorders, and panic disorders.

Lifetime rates of mood disorders in kleptomania range from 45% to 100%, and, individuals with kleptomania often report that their symptoms worsen when they are depressed. It has also been suggested that the act of shoplifting itself has an antidepressant effect. In fact, it is hypothesized that kleptomania might be a symptom of subclinical hypomania or mania (Grant, 2006).

This theory is based on (McElroy et al., 1996):

1. Phenomenological similarities between kleptomania and affective disorders, including harmful, dangerous, or pleasurable behaviors, impulsivity, and similar affective symptoms and dysregulation.
2. Both kleptomania and affective disorders onset in adolescence or early adulthood and are characterized with an episodic and/or chronic course.
3. There is a high comorbidity of kleptomania and affective disorders (or vice versa) and similar comorbidity with other psychiatric disorders.
4. With both kleptomania and affective disorders, there are elevated familial rates of mood disorders.
5. In both kleptomania and affective disorders, there are possible abnormalities in central serotonergic and noradrenergic neurotransmitters.
6. Both people affected by kleptomania and those affected by affective disorders response to well mood stabilizers and antidepressants.

The Impulse Control Spectrum Model

Grant (2003) suggested that impulsivity is a fundamental feature of kleptomania. He demonstrated that people meeting criteria for kleptomania are more likely than controls to suffer from another impulse control disorder, but they are not more likely to experience symptoms that match those of mood disorders or OCD (Grant et al., 2003).

In addition to the existing model, there is growing research interest in the attention deficit hyperactivity disorder (ADHD) model. The main symptoms of ADHD in adults are impulsiveness and inattention. One study found that 15% of individuals with kleptomania met the criteria for ADHD in their lifetime (Presta et al., 2002). However, there are no other published studies on the subject that can confirm or disprove this hypothesis (Grant, 2006).

The OCD Spectrum Model

Kleptomania is often compared to OCD since both disorders involve unwanted and irresistible acts. Compulsive stealing is impulsive and repetitive, resembling most cases of OCD as a struggle to oppose the drive and evoke anxiety and tension. Gratification or tension relief occurs during and/or after thievery, followed by regret, shame, depression, and guilt. Attempts at concealment are usual and are associated with anxiety, although despondency or guilt does not prevent continued stealing (Durst et al., 2001).

Hollander and Wong suggested that kleptomania should be considered as a subtype of OCD (Hollander and Wong, 1995). This conclusion is strengthened by evidence that some individuals with kleptomania report hoarding symptoms that resemble those of individuals with OCD (Grant, 2006). However, studies on the subject report inconsistent results, with some showing relatively high rates of co-occurrence (45%–60%) and others demonstrating low rates (0%–6.5%). When rates of kleptomania have been examined in subjects with OCD, relative co-occurrence has been found (2.2%–5.9%; Grant, 2006). Grant (2006) suggested that perhaps only some individuals with kleptomania share common features with individuals with OCD. Conceptualizing all individuals with kleptomania as similar may be too broad. Some subtypes of kleptomania may be more like OCD, whereas other subtypes may have more in common with addictive or mood disorders (Grant, 2006).

The Addiction Model

Kleptomania shares some distinct features with substance abuse disorders. Both disorders share an

urge to engage in a behavior with negative consequences. In addition, in both there is mounting tension unless the behavior is completed, and a rapid but temporary reduction of the urge follows the completion of the behavior. However, the urge returns after a few hours, days, or weeks. The behavior is ignited by unique external cues and secondary conditioning by external and internal cues like dysphoria or boredom. Both disorders induce hedonic feelings early in the addiction (Grant, 2006; Grant et al., 2009).

This suggestion for association between kleptomania and addictions is strengthened by the lifetime rates of substance abuse disorders among people with kleptomania, which range from 29% to 50%, according to Grant (2006). It is also common for people with kleptomania to have first-degree relatives with substance use disorders (Grant, 2006).

Psychological Models

From the 1920s to the 1950s, psychoanalytic theories dominated all discussions about kleptomania, and the psychoanalytic therapy was the treatment of choice. Starting in the 1960s, psychoanalysts showed less interest in kleptomania and the field of psychoanalysis lost most of its influence on the field of psychiatry, but psychoanalysis still left a mark on the interpretation of kleptomania. A current social approach suggests that kleptomania is a consequence of consumerism and the abundance of commodities in modern society. Psychoanalysts have interpreted the syndrome as a reflection of the unconscious ego defence against anxiety, forbidden instincts and wishes, unresolved conflicts or prohibited sexual drives, fear of castration and sexual arousal, sexual gratification, and orgasm during the act of thievery. Symbolic meaning has been attributed to the act itself, the object stolen, and the victim of the theft. Stealing may provide people, particularly youth, to appear like heroes to their peers. It has been suggested that kleptomania can have direct sexual significance and the thrill of secretly committing forbidden acts. Stealing has been observed to be the actual sex life of the person with kleptomania and indicates that his or her sex life is underdeveloped. It has also been suggested that kleptomania is analogous to a perversion and that the stolen objects have other sexual significance. These assumptions are highlighted by the fact that kleptomania and other impulse control disorders have been associated with fetishistic behavior (Goldman, 1991).

The psychoanalytic and dynamic approach provided a rationale for a long-term therapy as the main

treatment of choice for many decades. Kleptomania, like many other psychiatric disorders, was viewed through the psychodynamic lens rather than as a biomedical disorder (Durst et al., 2001). The efficacy of psychoanalytic and dynamic psychotherapy is unknown due to lack of controlled studies, but case reports reveal that some patients respond well to this type of therapy, sometimes in conjunction with medications, while others show no improvement (Grant, 2006).

Cognitive-behavioral therapy has recently replaced the psychoanalytic and dynamic approach in the treatment of kleptomania. A number of behavioral strategies have proven useful in the treatment of kleptomania, such as covert sensitization using aversive imagery of nausea and vomiting, aversion therapy, and systemic desensitization (Durst et al., 2001). Cognitive-behavioral therapy (CBT) suffers from a lack of controlled studies as well, but case reports reveal that some patients respond well to this type of therapy, especially in conjunction with medication (Grant, 2006).

Family History

Although studies on kleptomania have focused primarily on illness description, it is still unclear whether it is related to mood disorders, OCD, impulse control disorders, or addiction. A study on family history found that 35% of first-degree relatives of people suffering from kleptomania suffered from a mood disorder, 25% had OCD, and 15% had a substance use disorder (Grant, 2003). In a recent study, the data presented in Table 10.4 were derived (Grant, 2003).

Table 10.4 Family History as Compiled in Grant (2003)

Diagnosis	% of Relatives of Patients with Kleptomania	% of Comparison Relatives
Alcoholism	15.1	5.1
Depression	9.9	7.6
Bipolar disorder	1.3	0.8
Drug use disorder	2.0	3.4
Other psychiatric disorder	5.3	1.7
Any psychiatric disorder	28.3	11.0

Grant and Kim state that family history of psychiatric illness is a predictor of developing kleptomania within 1 year of beginning to steal (Grant & Kim, 2002b). The majority of patients appear to have significant family histories of psychiatric illnesses, substance use disorders, and kleptomania. The findings of high rates of mood and substance use disorders in first-degree relatives are consistent with the literature (Grant & Kim, 2002b). In one study, McElroy et al. (1992) stated that the most consistent finding has been high rates of mood and substance use disorders in the first-degree relatives of individuals with kleptomania. In that study, among 103 first-degree relatives of 20 subjects with kleptomania, 21% had a major mood disorder and 20% had alcohol or substance abuse disorder (McElroy et al., 1992).

Conclusions

Kleptomania seems to be more common than is generally believed, and the female population is more affected than the male population. Kleptomania may be a biologically nonhomogeneous disorder with various comorbid psychiatric diagnoses, also associated with legal, marital, and occupational issues.

Cognitive-behavioral interventions have been reported to be effective in certain cases. In other cases, treatment with a selective serotonin reuptake inhibitor may be effective and should be considered as a first-line treatment. Opioid receptor antagonists and/or mood stabilizers should be considered as second-line options. Further controlled trials of pharmacotherapy for kleptomania are needed to explore the effectiveness of drugs, the duration of treatment, and augmentation strategies, as well as to provide some understanding of the underlying etiopathology of this disorder.

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Pyromania: Phenomenology and Epidemiology

Michel Lejoyeux and Candice Germain

Abstract

Pyromania corresponds to fire setting not done for criminal reasons, for profit or sabotage, for monetary gain, as an expression of sociopolitical ideology (an act of terrorism or protest) or anger, or for revenge. Pyromania, in the sense of arson without a separate motive, is a rare phenomenon.

In the DSM-IV-TR, pyromania is classified as an impulse control disorder (ICD) not elsewhere classified. It is characterized by a failure to resist impulsive, repetitive, deliberate fire-setting urges that are unrelated to external reward.

The only study of the prevalence of fire setting derived from the National Epidemiological Survey on Alcohol and Related Conditions found a lifetime prevalence of 1% for fire setting in the U.S. population. The prevalence of pyromania in adult psychiatric inpatients was 3.4% ($n = 7$), and the lifetime prevalence was 5.9%.

Fire setting is significantly associated with a wide range of antisocial behaviors. Multivariate logistic regression analyses identified strong associations between lifetime alcohol and marijuana use disorders, conduct disorder, antisocial and obsessive-compulsive personality disorders, and a family history of antisocial behavior. Intentional illicit fire-setting behavior is associated with a broad array of antisocial behaviors and psychiatric comorbidities. The most prevalent psychiatric disorders among persons with a history of fire setting are any lifetime alcohol use disorder (71.7%), antisocial personality disorder (51.46%), marijuana use disorder (43.17%), and nicotine dependence (42.95%). A family history of antisocial behavior is also frequent (60%).

Keywords: pyromania, addiction, impulsivity, impulse control disorders, behavioral addiction, sensation seeking, alcohol use disorders

History of Psychiatric Attention to the Disorder

Fire setting and pyromania are serious and costly forms of antisocial behavior. Pyromania has been early identified as a psychiatric disorder. *Firesetting* is often used interchangeably with the legal term *arson*, which is the intentional setting of fire with the intent to defraud or damage (Vaughn et al., 2010). Fire setting is a broader behavioral phenotype with important forensic, psychiatric, and criminological implications. Psychiatric classifications kept the term *pyromania* to define an impulsive

behavior leading to fire setting without an identifiable motive other than taking pleasure in viewing the fire and its effects. The essential features are deliberate and purposeful fire setting on more than one occasion. The term *pyromania*—always considered as a psychiatric disorder—was introduced in 1833 by Henri Marc, a French psychiatrist, who described it as a form of “instinctive and impulsive monomania.” At that time, *monomania* meant an abnormal behavior, “a crime against nature, so monstrous and without reason, as to be explicable only through insanity, yet perpetrated by subjects

apparently in full possession of their sanity" (Marc, 1833). In 1845, Etienne Dominique Esquirol classified pyromania as a reasoning monomania caused by an instinctive desire to burn (Esquirol, 1845). According to Esquirol "the irresistible impulses show all of the features of passion elevated to the point of delirium". The word *pyromania* was transported from the French to the English medical vocabulary in the early nineteenth century.

Isaac Ray (1844), an American psychiatrist, addressed the question of pathological arson in his text on medical jurisprudence of insanity. He described pyromania as a "distinct form of insanity, annulling responsibility for the acts to which it leads". In the 1850s and 1860s, debates separated the authors who supported the idea that pyromania was a mental disorder and those who rejected this idea. President James Garfield's assassination in 1881 put into disfavor the use of the insanity plea to defend against criminal charges, and throughout the remainder of the nineteenth century, most American authorities rejected the concept of pyromania.

After Henri Marc in France, Wilhelm Griesinger, a German psychiatrist, stated, "Away with the term pyromania, and let there be a careful investigation in every case into the individual psychological peculiarities which lie at the bottom and give rise to this impulse. . . . To include cases of fire setting under the title of 'pyromania' is the necessary but evil result of a superficial classification" (Griesinger 1867). One hundred forty years later, however, the pyromania diagnosis is still present, now in the fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV; American Psychiatric Association, 2000).

In nineteenth-century Europe, the typical arsonist was thought to be a female domestic in her teens, uprooted from her home and family and suffering from nostalgia. In the early twentieth century, Wilhelm Stekel (1924) and Sigmund Freud (1932) postulated sexual roots of fire setting and advanced a psychoanalytic formulation centered on disordered psychosexual development, specifically fixation at or regression to the phallic-urethral stage of psychosexual development (see the section "Psychodynamic Models" below).

In the *Archives de Neurologie* for December 1904, Dr. Raoul Leroy, assistant physician at the Evreux Asylum, refers to the subject of pyromania in young persons of both sexes as a form of mental disorder that leads to acts of incendiaryism. "Whenever repeated fires occur," he says, "in a village or in the country suspicion generally falls on persons of incomplete

mental or physical development—weak-minded youths or girls among the inhabitants—and it generally proves to be well founded". The mental state of such incendiaries, says Dr. Leroy, is peculiar and characteristic. They are weak-minded and are often members of families in which epilepsy, insanity, or alcoholism occurs. Reference is made to the fact that among the peasant population of Normandy, where alcoholism prevails to a high degree, juvenile crimes of incendiaryism are common (Leroy, 1905).

Until 1950, the case history approach to the study of fire setting, with, all of its weaknesses, was dominant. No study involved a group of patients presenting with pyromania. In 1951, Lewis and Yarnell described several groups of arsonists, having reviewed the case histories of 1626 arsonists collected largely from the National Board of Fire Underwriters (Lewis & Yarnell, 1951). In 688 cases (42%), no objective reason explained the fire setting. From these cases, Lewis and Yarnell derived a still modern conception of pyromania: "Unexplained cases of fire-setting may correspond to pyromaniacs, acting under irrational impulsions, with possible internal tension, agitation, or derealisation". Although research has continued, our understanding of pyromania remains limited. The most difficult point is to identify the general psychological or biological mechanism that could explain the majority of cases of pyromania.

The DSM-I (American Psychiatric Association, 1952) considered pyromania as an obsessive-compulsive reaction. Patients were believed to be obsessed with fire, and their fire-setting behavior was seen as a form of compulsion. However, the majority of American medical literature in the 1960s rejected the concept of pyromania as a specific mental disorder. Fire setting was generally considered from a medico-legal point of view regarding the damages induced by the repeated fires. In 1968, the DSM-II (American Psychiatric Association, 1968) did not mention the term at all. In the 1970s, pyromania appeared again as a form of impulse control disorder (Mavromatis & Lion, 1977) and was included in the DSM-III (American Psychiatric Association, 1984) in the group of "disorders of impulse control not elsewhere classified." Pyromania is still included in this category of disorders.

Definitions and Differential Diagnosis

The consequences of repeated and voluntary fire setting are well known. It results in hundreds of fatalities each year and property losses estimated to range from hundreds of millions to more than \$2 billion annually (Putnam & Kirckpatrick, 2005). With the

exception of status offenses, fire setting is the only crime in the United States that is more commonly perpetrated by juveniles than adults (Vaughn et al., 2010).

Pyromania is much more difficult to define precisely and to distinguish from other types of fire setting. Many pathological behaviors, in addition to typical pyromania, implicate deliberate fire setting. Most of them are criminal rather than psychiatric.

Fire setting is the broadest term because it does not require the act to be intentional.

Arson refers to the act of deliberately setting fire to property of any kind. Authors writing in the field of criminology and law presume that most cases of arson are motivated by financial benefit to the individual. Most of these criminal acts are frauds committed in order to get money from insurance companies. Arson is a major source of property damage, injury, and death, and its incidence appears to have increased in both the United States and Europe (see the section “Prevalence” below). Some studies suggest that the frequency of criminal fire setting increases in times of economic recession, whereas in times of high inflation, the frequency is low (Lejoyeux et al., 2006). Other criminal reasons for arson include the attempt to prepare or to conceal another crime or to threaten or blackmail people.

Pathologic arson or fire setting designates the act as secondary to a medical, neurological, or psychiatric disorder.

According to the DSM-IV-TR (Text Revision) diagnostic criteria, fire setting by pyromaniacs is not done for criminal reasons, for profit or sabotage, for monetary gain, as an expression of sociopolitical ideology (an act of terrorism or protest) or anger, or for revenge. Pyromania must be distinguished from fire-setting behavior better explained by schizophrenia, bipolar disorder, substance abuse, personality disorders, dementia, and mental retardation. The DSM-IV-TR also excludes self-immolation and “communicative arson.” (Some individuals with mental disorders use fire to communicate a desire or need; see Geller 1992a). Pyromania, in the sense of arson without a separate motive, is a rare phenomenon that seldom explains repeated fire-setting behavior. The validity of pyromania as a psychiatric diagnosis continues to be questioned; some do not believe that this mental disorder really exists.

A Broad Classification of Arson

Jeffrey Geller (1992) reviewed medical and legal literature on arson over a 150-year period. He found

that during some periods there were frequent diagnoses of pyromania, while at other times there were very few. He noted that the definition of arson had changed several times since the mid-1800s—sometimes it was recognized as a mental illness, sometimes it wasn’t, and sometimes there seemed to be no conclusion. Geller suggested that the reasons for these changes did not reflect differences in the behavior itself, but rather shifts in the field of psychiatry as each generation attempted to sort out whether an individual is truly responsible for his or her own actions. Geller (1992a) used his clinical experience and his literature review to provide a clinically focused classification of arson (Table 11.1). He emphasized the methodological difficulties of work on arson: (1) samples of patients are often derived from very specialized populations (hospitals, prisons, etc.), and (2) while typologies artificially segregate fire setters into two groups, the so-called motivated group and the so-called motiveless group, pathological fire setters assigned to the latter category may have motives that have not been recognized.

Leroy (1905) noted that pyromaniacs are prone to set fire to buildings or other objects in revenge against their owners or, in some cases, merely to amuse themselves with the spectacle. A few individuals suffer from the influence of an obsession that irresistibly impels them to such acts, such cases forming a special form of insanity to which the term *pyromania* is applied.

Pyromania is especially difficult to define among schizophrenic patients. In these individuals, a significant percentage of fire setting can be accounted for by reasons other than delusional ideation or hallucinations. Youth who set a fire secondary to a delusional system often have targeted fire sites, such as churches or schools. Fires are set as a direct response to disordered thinking, a misperception of reality, and thinking errors that may revolve around religious, political, personal, or even counterintuitive agendas and result in related targets for fire setting. Some individuals can be sophisticated enough to set fires for reasons independent of hallucinations but then blame the arson on “the voices”! They may, however, set fire for other motives, such as expressing social or political ideas, revenge or anger, or to improve their living circumstances. Fire setting may be the most spectacular expression of their impaired judgment. No study has addressed this question specifically and has tried to clarify the motives for fire setting of schizophrenics.

Table 11.1 Classification of Arson

Arson Unassociated with Psychobiological Disorders	Arson Associated with Mental Disorders
Arson for profit	Disorders of thought or perception
Insurance fraud	Delusions
Welfare fraud	Hallucinations
Bankruptcy scam	Disorders of mood
Property improvements	Depression
Building stripping	Mania
Burglary	Disorders of judgment
Business modifications	Developmental disorders
Employment	Dementia
Crime concealment	Psychoactive substance-induced disorders
Revenge	Disorders of impulse control
Vanity of recognition	Intermittent explosive disorder
Hero syndrome	Pyromania
Fire buff	Communicative arson
Vagrant	
Vandalism	
Political	
Riot	
Terrorism	
Protest	
Arson Associated with Medical or Neurological Disorders	Juvenile Fire
Chromosomal disorders	Fire setting
Klinefelter's syndrome	Fire play
XYY syndrome	
Central nervous system disorders	
Epilepsy	
Head trauma	
Brain tumor	
Infectious diseases	
Acquired immune deficiency syndrome (AIDS)	
Endocrine and metabolic disorders	
Late luteal phase dysphoric disorder	
Hypoglycemia	

Diagnosis

In the DSM-IV-TR, pyromania (Code 312.33) is classified as an impulse control disorder (ICD) not elsewhere classified. The DSM-IV-TR diagnostic criteria (Table 11.2) exclude fire setting better explained by any other diagnosis. Pyromania is characterized by a failure to resist impulsive, repetitive, deliberate fire-setting urges that are unrelated to external reward (money, revenge, or a political act).

Table 11.2 Pyromania (Code 312.33): DSM-IV-TR Criteria

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 (i.e., utensils, utilization, and 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, sociopolitical ideology, criminal purpose, out of anger or revenge, or in response to delusional ideas, hallucinations, or judgment disorder (i.e., dementia, mental retardation, or substance intoxication).
F. The fire setting is not better explained by a conduct disorder, a manic episode, or an antisocial personality.

In the *International Classification of Diseases*, 10th edition (ICD-10; World Health Organization, 2002), pyromania is listed in the category “habit and impulsive disorders” (Code F63). The definition closely resembles the DSM-IV-TR definition (Table 11.2) but substitutes the phrase “persistent preoccupation with subjects related to fire and burning” for the “fascination” and “interest” mentioned in DSM-IV-TR.

Apart from the DSM-IV-TR and ICD-10 definitions, the term *pyromania* has sometimes been used to denote all acts of fire setting motivated by the pleasure of watching the fire (Vreeland & Lewin, 1980). Some authors have used the term *pyromaniac* for individuals who seem to attain sexual satisfaction from fire setting (Prins, 1980). Others have used the term as a diagnostic label for individuals who are motivated by an irresistible impulse to set fires (Mavromatis & Lion, 1977). Pyromania could also be considered a compulsive behavior, but no published study justifies this label. To our knowledge, a relationship between pyromania and the obsessive-compulsive spectrum has not been demonstrated.

The notion of behavioral addiction could also be applied to pyromania. The recent work of Grant and Kim (2007), described later in this chapter, confirms that pyromania shares many phenomenological characteristics with other impulse control disorders and addictive disorders:

Urge or craving state prior to engaging in the behavior

Pleasure derived from the behavior

Repetitive engagement in the behavior despite negative consequences

Diminished control over the behavior

Increased frequency and intensity of the behavior over time (tolerance)

Clinical Picture of Pyromania and Fire Setting

Sarah Wheaton (2001)—who was a college student when she was admitted to a psychiatric hospital for compulsive fire setting—described a lifelong fascination with fires and an irresistible impulse to set them. “Fire became a part of my vocabulary in preschool days. . . . Each summer, I would look forward to the beginning of fire season as well as the fall. . . . I may feel abandoned, lonely, or bored, which triggers feelings of anxiety or emotional arousal before the fire. . . . I want to see the chaos as well as the destruction that I or others have caused. . . . [After the fire is out] I feel sadness and anguish and the desire to set another fire”. The essential feature of pyromania is thus fascination with fire and the equivalent of a craving for fire setting. This excitation induced by fire setting incites pyromaniacs to deliberately light multiple fires for the purpose of achieving pleasure or gratification. Persons experience an urge to engage in fire setting, along with a loss of control, despite the negative consequences. Some pyromaniacs, however, make meticulous preparations before acting on the urge and experience tension or emotional arousal during this preparation phase. This excitation has been extensively described by Sarah Wheaton: “I never light a fire in the exact place other fires have occurred. I set fires at random, using material I have just bought or asked for at a gas station—matches, cigarettes, or small amounts of gasoline. I set fires only in places that are secluded, such as roadsides, back canyons, cul-de-sacs, and parking lots. I usually set fires after nightfall because my chances of being caught are much lower then. I may set several small fires or one big fire, depending on my desires and needs at the time. It is at the time of lighting the fire that I experience an intense emotional response like tension release, excitement, or even panic”.

Raoul Leroy (1905) described another typical case of pyromania in a young girl: “she felt the sudden morbid impulse to set something on fire. It grew stronger though she struggled against it, suffering great mental and physical distress in the process. The morbid obsession occupied her mind to the exclusion of all other ideas and caused such distress and agony that she could resist no longer.

Fire setting was followed by instant relief of distress and an agreeable feeling of satisfaction. These obsessions occurred from time to time and on three such occasions she set fire to outbuildings and parts of her master’s house”.

Pyromaniacs are often recognized as regular watchers at fires in their neighborhoods. Sarah Wheaton also explained to her psychiatrists: “I watch the local news broadcasts for fires that have been set each day and read the local newspapers in search of articles dealing with suspicious fires. I read literature about fires, arsonists, pyromania, pyromaniacs, arson, and arsonists. I contact government agencies about fire information and keep up-to-date on the arson detection methods investigators use. I watch movies and listen to music about fires. My dreams are about fires that I have set, want to set, or wish I had set”.

Typical pyromaniacs have indeed a passion for the institutions, equipment, and persons who struggle against fire. They may spend time in the local fire station or even work as firefighters. Some collect firemen’s tools and fire photos. They may activate false fire alarms to see firemen. “I love to drive back and forth in front of fire stations, and I have the desire to pull every fire alarm I see”, said Sarah Wheaton. Pyromaniacs are usually indifferent to the material or human consequences of a fire they have caused. Some may experience a certain pleasure while viewing the resulting destruction. Others are ashamed of the consequences of their acts. Their “addiction to fire,” however, is stronger than their remorse, and their behavior tends to repeat itself.

Grant and Kim (2007) described clinical characteristics of 21 individuals with a DSM-IV lifetime diagnosis of pyromania. The mean (\pm SD) age at onset of pyromania was 18.1 (\pm 5.8) years. Eighteen subjects (85.7%) reported the urge to set fires. Subjects reported setting a fire a mean (\pm SD) of once every 5.9 (\pm 3.8) weeks. Most fire settings did not meet the legal definition of arson: subjects reported setting “controlled” fires in their bathrooms, in dumpsters, in backyards, or in vacant lots. They found pleasure in setting fires, regardless of what they were burning, and were aware of the significant amount of time they spent on this behavior. The majority (66.7%) described planning the fires, buying utensils to set them, and planning what items would burn most intensely; 57.2% reported watching fires, even those they had not set, and 38.1% reported traveling to fires when they heard fire engines. All patients reported a “rush” when setting or watching a fire, and most reported

a sexual feeling. Of these 21 subjects, 76.2% reported that the frequency of fire setting and the intensity of the fires increased over time. This element is like the tolerance phenomenon characteristic of physiological dependence. Triggers for setting fires were most often stress or boredom. All subjects reported pleasure or relief when setting fires, but 19 subjects (90.5%) reported feeling severe distress afterward. Some had thoughts of suicide.

More data are needed to elucidate the course of pyromania. Adolescence is a period of increased risk taking, and controversy exists about how frequently adolescent fire-setting behavior continues into adulthood (Barnett & Spitzer, 1994).

The normal human interest in fire starts between 2 and 3 years of age (Nurcombe, 1964). This curiosity usually wanes with age and with a firm understanding of the dangers of fire. Kafry's (1980) study of normal schoolboys at the ages of 6, 8, and 10 found that interest in fire was almost universal. Among children (Kosky & Silburn, 1984), the distinction between normal interest in fire and excessive interest leading to pyromania is not always clear. Simply playing with matches is not, however, a symptom of pyromania. Kolko and Kazdin (1989a, 1989b) found that children who set fires had more curiosity about fire, recent involvement in fire-related activities, early experience with fire, and contact with peers or family members involved with fire than did children who had not set fires. Thus, a continuum may exist between excessive interest in fire and pyromania.

Slavkin (2002) summarized the characteristics of juveniles who set fires. The fire-setting behavior is associated with aggression, vandalism, antisocial behavior, and deficits in social skills. Predictors of fire setting also include level of deviancy (lying, truancy, running away from home) and disturbed family dynamics (family conflicts, lax discipline, violence at home, parental alcohol or drug abuse, physical or sexual abuse, or emotional neglect).

An abnormal interest in fire is especially important in populations of fire officers who become pyromaniacs. Lewis and Yarnell (1951) described a series of 90 volunteer firemen who deliberately set fires. A notable proportion of fire fetishists are found among pyromaniacs, whether or not they are working as fire officers. McGuire, Carlisle, and Young (1965) showed that a "fire experience" may become a "fire fetish." A fire fantasy—whether imagined or a recollection of a real event—occurring just before orgasm is conditioned by the positive feedback of orgasm to become more and more exciting.

Noblett and Nelson (2001) studied females presenting with pyromania or arson. Arsonists more frequently had a history of deliberate self-harm and sexual abuse, but both groups had often suffered psychosocial traumas and difficulties. Noblett and Nelson state that pyromania may be a displacement of aggressivity. Persons with pyromania may be unable to confront people directly and may channel their aggression into fire-setting behavior in an attempt to influence their environment and increase their self-esteem when other means have failed. Geller (1987) explained fire setting as an attempt at communication by individuals with few social skills.

Barnett and Spitzer (1994) reviewed the motives of individuals setting fires. Revenge, hatred, envy, jealousy, and disappointed love are most often seen in adults. In adolescents, motives such as malignity, anger, rage, the fun of extinguishing fire, arrogance, and the craving for recognition prevail. In quite a few cases, several motives are present, revenge often being one of them (Hill et al., 1982). In many cases, the act of fire setting is carried out under the influence of alcohol (Geller, 1987; Hill et al., 1982).

Many cases of nonpsychotic fire setters who had no obvious motive have also been documented. In these cases, the forensic psychiatric expert is considerably handicapped. Psychological research into the personality of such fire setters is, however, suggestive of personality disorders characterized by intolerance of frustration, overinhibition of aggression in ordinary behavior, striving for power, readily feeling insulted, and a tendency to act aggressively (Lejoyeux et al., 2002).

Group Fire Setters

Based on clinical observations in a series of studies on Finnish alcoholic, violent offenders, Virkkunen et al. (1994) asserted that the impulsive fire setters represented an extreme group of type 2 alcoholics. They also observed that these subjects were vulnerable to hypoglycemia after the administration of an oral glucose load. Furthermore, they showed that while they are hypoglycemic, the impulsive offenders are particularly irritable and aggressive. In general, riot fires represent multiple acts of fire setting in slum suburbs of large cities in times of social and racial strain. The phenomenon is most common in the United States. However, recent acts of fire setting in shelters for refugees in Germany and in suburbs of large cities in France are just as much the result of social and racial tensions. Fueled by rage, revenge fire setters may identify their targets in a

methodical and purposeful manner and determine the type of havoc they wish to create. This type of arsonist is devoid of much emotion other than resentment, suspicion, or anger. Characteristically, revenge fire setters feel little guilt or remorse because they feel justified for their actions. Interestingly, the threat of discovery and/or arrest does not seem to deter revenge fire setters from their criminal behavior.

Gangs of fire setters sometimes act for political reasons. Fire setting by adolescents can also be motivated by boredom. In most cases, the vandals are driven by the desire to destroy and the excitement involved in carrying out the act (Molnar et al., 1984). An examination of childhood histories reveals that members of this subtype appear to be raised in families that teach the concept of "getting even" for real or imagined slights. As a group, male revenge fire setters enjoy collecting weapons and have often spent time in the military or participated in military-type activities with a clear and intense, systematic structure. Male revenge fire setters appear to pose an extremely high risk for recidivism because they are convinced of the appropriateness of their acts.

Assessment

Most assessment scales of impulse control disorders do not include a section on pyromania. Fire setting is included, however, in the Minnesota Impulsive Disorders Interview (MIDI), a semistructured clinical interview with excellent classification accuracy in adults with impulse control disorders (ICDs; Grant et al., 2005). The MIDI includes questions for each ICD, with additional follow-up questions reflecting DSM-IV criteria. With regard to fire setting, patients are invited to indicate whether they have lit a fire intentionally and are asked about their urge to set a fire. The MIDI excludes fire setting for financial motives and during periods of anger and examines fires lit to escape from a depressive mood. The last question concerns fascination with fire.

Three instruments are available to detect children at risk for fire setting:

Lowenstein Fire Raising Diagnostic Test
(Lowenstein, 1981, 1989)

Children's Fire-setting Interview (Kolko & Kazdin, 1989a)

Fire-setting Risk Interview (Kolko & Kazdin, 1989b)

No such instruments are available for the adult population.

Prevalence

The only study of the prevalence of fire setting derived from the National Epidemiological Survey on Alcohol and Related Conditions (Vaughn et al., 2010). A total of 43,093 structured interviews were completed by trained interviewers between 2001 and 2002. The prevalence of lifetime fire setting in the U.S. population was 1%. Respondents who were men (odd ratio 0.64), white, 18 to 35 years old, born in the United States (odd ratio 2.59), and living in the western region of the United States had significantly higher rates of fire setting. The majority of other epidemiological studies have focused on fire setting in childhood or adolescence (Dell'Osso et al., 2006): prevalence has been estimated at between 2.4% (Jacobson, 1985) and 3.5% (Kolko & Kazdin, 1989b; Kosky & Silbrun, 1984). Sixty percent of all fires in large U.S. cities are lit by individuals under 18 years of age (Raines & Foy, 1994). Among juveniles, fire setting is more prevalent in males than in females (Soltys, 1992).

Pyromania is rare in recent studies of psychiatric populations, in contrast to results from earlier periods. This contrast may result in part from the fact that, before the publication of DSM-III, the term *pyromania* was often misapplied in the psychiatric literature and referred to all pathological fire setters. This still may be the case in the nonpsychiatric literature on arsonists, in which the prevalence of pyromania is reported as considerably higher than in the psychiatric literature.

The prevalence of pyromania among adult arsonists is not well established. In a prospective study between 1983 and 1993 of 153 adult arsonists in Yorkshire, England, the next most common motive after revenge (31%) was excitement (11%), which could correspond to pyromania (Rix, 1994). In a retrospective study of 282 arsonists, the rate of pyromania was high: pyromania was found in 23% of arsonists who had committed other nonviolent crimes and in 12% of arsonists with a history of violent crimes (Repo et al., 1997). In a recent Finnish study describing a forensic psychiatric population of 90 arson recidivists, only 3 subjects (3.3%) fulfilled the diagnostic criteria for pyromania (Lindberg et al., 2005).

The current prevalence of pyromania in 204 adult psychiatric inpatients was 3.4% ($n = 7$) and the lifetime prevalence was 5.9% ($n = 12$; Grant et al., 2005). A French study involving 107 depressed patients found that 3 (2.8%) met the DSM-IV-TR criteria for pyromania (Lejoyeux et al., 2002).

Another study of 79 alcoholic inpatients found no case of pyromania (Lejoyeux et al., 1999).

In one study in a child psychiatric outpatient population, the diagnosis of pyromania applied in only 2 of 32 fire setters (6.3%; Heath et al., 1985). A recent study looking for ICDs in 102 adolescent psychiatric inpatients found a 6.9% current prevalence rate for pyromania, with a statistically significantly higher rate in girls (Grant et al., 2007).

Grant and Kim (2007) described 21 individuals meeting DSM-IV lifetime criteria for pyromania. Fourteen were adults, of whom 3 (21.4%) were female and 7 were adolescents, all of whom were female. Their ages ranged from 15 to 49 years (mean \pm SD = 26.1 \pm 11.8 years). The majority of the subjects were white (85.7%) and single or divorced (71% of the adults and all adolescents). Adults had good educational attainment (57.1% were high school graduates), and most (71.4%) were employed. Five (23.8%) alleged histories of sexual abuse, and 7 (33.3%) reported histories of physical abuse.

Comorbidities of Arson and Pyromania

Establishing rates of psychiatric comorbidities among individuals with pyromania is difficult because most data are reported for broader groups such as fire setters or arsonists. These heterogeneous data are summarized here, and an attempt is made to distinguish clearly the groups described.

The National Epidemiological Survey on Alcohol and Related Conditions (Vaughn et al., 2010) found that fire setting is significantly associated with a wide range of antisocial behaviors. Multivariate logistic regression analyses identified strong associations between lifetime alcohol and marijuana use disorders, conduct disorder, antisocial and obsessive-compulsive personality disorders, and a family history of antisocial behavior. Intentional illicit fire-setting behavior is associated with a broad array of antisocial behaviors and psychiatric comorbidities. The most relevant behaviors for persons with a history of fire setting were staying out late without permission, cutting class and leaving without permission, and shoplifting. The least prevalent behavior was forcing someone to have sex. The strongest association between antisocial behaviors and fire setting was found for destroying others' property, robbing/mugging someone, or snatching a purse. The most prevalent psychiatric disorders among persons with a history of fire setting were any lifetime alcohol use disorder (71.7%), antisocial personality disorder (51.46%), marijuana use disorder (43.17%), and nicotine dependence (42.95%).

A family history of antisocial behavior was also frequent (59.96%).

Only one study has examined psychiatric comorbidity in a sample of individuals ($n = 21$) with a history of pyromania (Grant & Kim, 2007). Thirteen subjects (61.9%) had a current comorbid mood disorder, 10 (47.6%) met criteria for another ICD, and 7 (33.3%) had a diagnosis of a substance use disorder. Some subjects felt that their mood symptoms and substance abuse were responses to their distress over setting fires. Of the 21 subjects, 95% had at least one lifetime comorbid psychiatric disorder, particularly major depression (48%), bipolar disorder (14%), another ICD (67%), or a substance use disorder (33%). Interestingly, all subjects who no longer met criteria for pyromania reported that symptoms of another ICD or substance use disorder had developed since they stopped setting fires (Grant & Kim, 2007).

The true rate of mental illness in fire setters is hard to establish, in part because many fire setters are not apprehended. Hence, rates ranging from about 10% to 20% (U.S. Department of Justice, 1982) to over 60% (Taylor & Gunn, 1984) have been reported. The disorders most frequently present are personality disorders, psychotic disorders, and mental retardation (Barnett & Spitzer, 1994 [fire setters]; Lindberg et al., 2005 [arsonists]; Rix, 1994 [arsonists]).

In a study of 106 fire setters referred for psychiatric examination (Gunzel, 1987), 56% were found to suffer from mental retardation, a personality disorder, dementia, or alcoholism; 7.6% had a psychotic disorder; more than 30% had no apparent separate motive; and the act was rationally motivated in only 3%.

In a Finnish study (Joukamaa & Touvinen, 1983), fire setters for whom a psychiatric expert opinion had been obtained received a diagnosis of psychosis or borderline personality disorder more frequently than other delinquents. Alcoholism and mood disorders are also frequent diagnoses among arsonists (Koson & Dvoskin, 1982).

In a study of mental health records and/or prison files of 283 arsonists, Ritchie and Huff (1999) found that 36% had a diagnosis of schizophrenia or bipolar disorder and 64% were abusing alcohol or drugs at the time of fire setting; only 3 cases carried a pyromania diagnosis. Other authors have noted a close link between fire setting, aggression, and anti-social behavior in juvenile fire setters (Stickle & Blechman, 2002).

Fernandez-Aranda et al. (2008) compared symptom patterns, severity of illness, and comorbidity in

individuals with eating disorders with and without ICDs and documented the temporal pattern of illness onset. Their work was not focused on pyromania; this disorder, however, was systematically assessed among all other ICDs. Lifetime ICDs were present in 16.6% of 709 women with a history of eating disorders. The most common syndromes were compulsive buying disorder and kleptomania. The authors found two cases (0.3%) of pyromania. Impulse control disorders—including pyromania—occurred more often in individuals with binge eating subtypes, and were associated with significantly greater use of laxatives, diuretics, appetite suppressants, and fasting, and with greater body image disturbance, higher harm avoidance, neuroticism, cognitive impulsivity, and lower self-directedness. In addition, individuals with ICDs were more likely to have obsessive-compulsive disorder, any anxiety disorder, specific phobia, depression, Cluster B personality disorder, and avoidant personality disorder and were more likely to use psychoactive substances. Among those with ICDs, 62% reported that the ICD predated the eating disorder and 45% reported the onset of both disorders within the same 3-year period.

Mc Elroy et al. (1996) studied the association between ICDs, including pyromania, and bipolar disorders. The two disorders share a number of phenomenological similarities. The types of behavior are similar in both conditions, generally involving harmful, dangerous, sensation-seeking, and/or pleasurable actions. For both ICDs and mania, these behaviors include fire setting, aggressive outbursts, excessive or inappropriate spending, and gambling. The thinking of patients with ICDs and mania is automatic or rapid and without reflection. Behaviors in both conditions are often performed without forethought. Further, both ICDs and mania are associated with poor insight into the danger, harmfulness, and/or consequences of the enacted behaviors like fire setting. The symptoms of ICD are frequently accompanied by affective symptoms that resemble those of bipolar disorder. Impulsive fire settings are often associated with tension or anxiety similar to that which can occur with depression or mixed affective states, or with arousal that resembles the elevated mood, increased energy, and enhanced clarity of thought that characterize hypomania. Pyromaniac actions are often associated with pleasurable feelings, variously described by patients as feeling “high,” “euphoric,” “a thrill,” or “a rush,” which resemble the elevated mood or euphoria of hypomania or mania. Finally, after performance of

an impulsive action and resolution of the associated high, individuals with pyromania often describe the acute onset of depressive symptoms similar to those of bipolar depression, including depressed mood, feelings of guilt and self-reproach, and fatigue.

The reported prevalence of personality disorders in arsonists and fire setters varies between 25% and 66% (Bourget & Bradford, 1989; Bradford, 1982; Bradford & Dimock, 1986; Hill et al., 1982; O’Sullivan & Kelleher, 1987; Rice & Harris 1990). A study by Virkkunen et al. (1989) in which subjects were selected for the impulsive character of their fire setting reported a prevalence of personality disorders of 90%. When specific diagnoses are provided, the most common personality disorders are antisocial and borderline (Bradford & Dimock, 1986; Virkkunen et al., 1989). Forehand et al. (1991) suggest that juvenile fire setting represents an advanced stage of antisocial behavior and is not a unique syndrome. In their study, juvenile fire setters and non–fire setters who had a comparable number of conduct disorder symptoms did not differ on the child behavior checklist subscales of adolescent psychopathology.

Arson has also been reported to be associated with schizophrenia and other psychotic disorders (Geller, 1987; Koson & Dvoskin, 1982; Lewis & Yarnell, 1951; O’Sullivan & Kelleher, 1987; Virkkunen, 1974). Many studies of schizophrenic fire setters show that they may set fires under the influence of their psychotic symptoms. However, all studies of psychotic fire setters note that, in the majority of cases, nonpsychotic motives such as revenge or hatred prevail.

In some cases, arson is secondary to major depressive disorder (Lewis & Yarnell, 1951; O’Sullivan & Kelleher, 1987) or to mania in bipolar disorder (Geller, 1987; Koson & Dvoskin, 1982; Lewis & Yarnell, 1951). Arson has been frequently associated with mental retardation (Bradford, 1982; Bradford & Dimock, 1986; Geller, 1987; Harris & Rice, 1984; Hill et al., 1982; Koson & Dvoskin, 1982; Lewis & Yarnell, 1951; O’Sullivan & Kelleher, 1987) and dementia (Harris & Rice, 1984; Lewis & Yarnell, 1951; Yesavage et al., 1983).

Grant et al. (2006) assessed pyromania among patients presenting with obsessive-compulsive disorder (OCD). They studied 293 consecutive subjects with lifetime DSM-IV OCD (56.8% females; mean age = 40.6 ± 12.9 years). Forty-eight (16.4%) OCD subjects had a lifetime ICD, and 34 (11.6%) had a current ICD. Skin picking was the most common lifetime (10.4%) and current (7.8%) ICD,

followed by nail biting, with lifetime and current rates of 4.8% and 2.4%, respectively. Pyromania was present in only one patient (0.3%). Unsurprisingly, the tendency to set fire is not associated with the presence of an OCD. Lastly, arsonists may be under the influence of psychoactive substances, especially alcohol (Bourget & Bradford, 1989; Bradford, 1982; Bradford & Dimock, 1986; Hill et al., 1983; Koson & Dvoskin, 1982; Lewis & Yarnell, 1951; Yesavage et al., 1983).

A French study (Yesavage et al., 1983) compared fire setters found not guilty for psychiatric reasons with mentally healthy fire setters. Motives such as revenge and “fun with fire” were much more frequent in the psychiatric group compared with the nonpsychiatric group. In the latter group, the act was frequently denied or motives were unknown to the delinquents. In both groups, the authors found divorced parents, especially absence of the father.

Studies of fire-setting psychiatric inpatients show that most suffer from schizophrenia (Geller, 1987; Geller & Bertsch, 1985) or borderline personality disorder (Boling & Brotman, 1975; Rosenstock et al., 1980), whereas mental retardation, mood disorders and other personality disorders are less frequent (Geller, 1987, Geller & Bertsch, 1985). One must keep in mind that these diagnoses were not made using current diagnostic criteria.

Medical and neurological disorders associated with cases of arson include:

Chromosomal disorders such as Klinefelter’s syndrome and XYY syndrome (Cowen & Mullen, 1979; Eberle, 1989; Kaler, White, & Kruesi, 1989; Miller & Sulkes, 1988; Nielsen, 1970)

Epilepsy (Byrne & Walsh, 1989; Carpenter & King, 1989; Singer et al., 1978; Stone, 1986)

Head trauma (Hurley & Monaham, 1969)

Brain tumors (Tonkonogy & Geller, 1992)

Cerebellar arachnoid cyst (Heidrich et al., 1996)

Huntington’s chorea (Yoshimasu, 1965)

Moebius syndrome (congenital palsy of the sixth and seventh cranial nerves, often associated with other nerve palsies and other abnormalities; Woolf, 1977)

Infectious disorders, especially AIDS (Cohen et al., 1990)

Endocrine and metabolic disorders include:

Late luteal phase dysphoric disorder (Dalton, 1980)

Reactive hypoglycemia measured by the glucose tolerance test (Virkkunen, 1982, 1984)

Biological Approaches to Pyromania

A low 5-hydroxyindoleacetic acid (5-HIAA) concentration in the cerebrospinal fluid (CSF) is associated with disorders of impulse control (Linnoilam et al., 1983). In one study, impulsive fire setters had lower CSF concentrations of 5-HIAA and 3-methoxy-4-hydroxyphenylglycol (MHPG) than a control group matched for age, sex, and height (Virkkunen et al., 1989). In this study, the recidivists who set fires during the follow-up period had lower CSF 5-HIAA and MHPG concentrations than did the nonrecidivists. As noted above, acts of fire setting have been reported in patients with various organic brain disorders.

The observed switch from one impulsive behavior to another, as well as the high rates of co-occurring impulsive and addictive disorders in pyromania, have raised the question of whether a similar neuropathophysiology underlies various pathological behaviors that are strongly characterized by reward seeking (Grant & Kim, 2007).

Pyromania may be explained by a mixture of impulsive and planned thrill seeking to alleviate dysphoric states. Pyromania does not correspond to a unique phenotype with a unique neurobiology. As with other ICDs, neuroimaging, genetic studies, and clinical trials will be needed to identify its pathophysiology (Lejoyeux et al., 2006).

Psychodynamic Models

The psychoanalytic approach aims to explore the unconscious motivation behind fire setting and the symbolism of fire. One of the first descriptions of fire setting refers to Samson, the biblical hero (Barker, 1994). “Samson set the torches alight and set the jackal loose in the standing corn of Philistines. And he burnt up standing corn and stocks as well, and vineyards, and olive groves.” A symbol of comfort and warmth, but also of hell, destruction, valor, and power, fire still pervades cultural imagery.

Wilhelm Stekel (1924), discussing 95 cases of fire setting, asserted that “awakening and ungratified sexuality impel the individual to seek a symbolic solution to his conflict between instinct and reality”. Writing before psychoanalytic theory was created, Henri Marc (1833) also noted a sexual dimension to pyromania: “Incendiary acts are chiefly manifested in young persons, in consequence of the abnormal development of the sexual function, corresponding with the period of life between twelve and twenty”.

Freud postulated that, in fantasy, fire is extinguished with a stream of urine. Hence, psychoanalysts

suggest that fire setters are fixated at the urethral or phallic-urethral phase of psychosexual development (Freud, 1932; Grinstein, 1952; Schumacher, 1991). Freud (1932) turned to classical mythology to support his formulation, citing the story of Prometheus, the Titan who brought mankind the fire he had stolen from the gods, hidden in a fennel stalk, a hollow rod. Freud saw the fennel stalk as a penis symbol and, invoking the mechanism of reversal, he suggested that it was not fire that man harbors in his penis but the means of extinguishing fire: the water of his stream of urine.

In related works, it has been suggested that adult fire setters suffered from enuresis as children (Gold, 1962; Greenberg, 1966). However, the theoretical relationship between fire setting and urethral fixation has been discussed. Fire setting may also be explained as the result of an oral fixation (Kaufman et al., 1961).

Impulsivity and Sensation Seeking

We assessed impulsivity and sensation seeking in a population of depressed patients presenting with ($n = 31$) and without ($n = 76$) ICDs (Lejoyeux et al., 2002). The Zuckerman Sensation Seeking Scale (Zuckerman et al., 1978) did not allow us to show significant differences between the two groups in the overall score or in subscale scores (thrill and adventure seeking, disinhibition, boredom susceptibility, and experience seeking). Pyromaniacs did not present a higher level of sensation seeking. Sensation seeking did not differ between subtypes of ICDs. Mean total impulsivity scores, assessed with the Barratt scale (Barratt & Patton, 1983), were not significantly different in the ICD+ (53.5) and ICD- (47.8) groups. But the ICD+ and ICD- groups differed significantly in Barratt scale mean motor impulsivity scores (18.1 versus 14.3, $p = .01$). Barratt scale motor impulsiveness is defined as acting without thinking, while cognitive impulsiveness is characterized by quick cognitive decisions and non-planned impulsiveness by lack of anticipation. Barratt scale motor, cognitive, and nonplanned impulsivities were not increased in patients with pyromania ($n = 3$) compared with the other ICD patients.

Impulse control disorders in general and pyromania in particular were not associated with antisocial or borderline personality disorders. These two disorders correspond to a behavior style that is impulsive and unable to tolerate frustration. In depressed patients, these personality types do not seem to bring about unusual impulsive behavior.

Surprisingly, patients with ICDs were more often married. Other authors have suggested that ICDs induce marital disruption and affective loneliness (McElroy et al., 1992). Our three pyromania patients were married.

Conclusion

Among ICDs, pyromania remains one of the less studied diseases. In most cases, patients are not recognized as being incapable of resisting their impulsivity. They are arrested and judged as criminals. In jail, they are still not recognized as psychiatric cases, and when they leave the jail, they demonstrate a high frequency of recidivism. Some clinical characteristics appear, however, as specific to pyromania. Interest in and morbid fascination with fire often appear early in life and could be identified as a major risk factor. Among the psychological models explaining pyromania, the most convincing one refers to impulsivity. Intolerance of frustration, difficulty in planning behavior, and differential responses to stress may represent important endophenotypes for pyromania and other ICDs (Brewer & Potenza, 2009). Identifying these psychological mechanisms should help us understand the etiology of fire setting, develop more effective prevention strategies, and optimize behavioral and pharmacological treatments.

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The Phenomenology and Epidemiology of Intermittent Explosive Disorder

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Abstract

Intermittent explosive disorder (IED) is characterized by recurrent episodes of impulsive, uncontrollable aggression out of proportion to the severity of provoking agents. Few epidemiological studies have been carried out on the prevalence and correlates of IED. Data are reported here from the most recent and largest of these studies: the U.S. National Comorbidity Survey Replication (NCS-R) and the World Health Organization World Mental Health (WMH) surveys. These studies show that IED is a commonly occurring disorder that typically has an early age of onset, a persistent course, and strong comorbidity with a number of other usually secondary mental disorders. This disorder is almost twice as common among men as women. It is often associated with substantial distress and impairment. However, only a minority of people with IED obtain treatment for their uncontrollable anger. This combination of features makes IED an ideal target for early detection and intervention aimed at secondary prevention of anger attacks as well as primary prevention of secondary disorders.

Keywords: intermittent explosive disorder, impulse control disorders, aggression, anger, epidemiology, comorbidity

Criteria for a diagnosis of intermittent explosive disorder (IED) in the *Diagnostic and Statistical Manual of Mental Disorder* (4th ed; DSM-IV; American Psychiatric Association, 1994) include the requirement that the patient have recurrent episodes of serious aggression that involve assaultive acts or destruction or property out of proportion to the provocation or to precipitating stressors. The attacks must be characterized by a failure to control aggressive impulses and the inability to be better accounted for by the presence of another mental disorder or by the physiological effects of a substance. Intermittent explosive disorder is the only diagnosis in the DSM-IV that involves impulsive aggression as the core feature. Although a version of the same diagnosis was also included in earlier versions of the DSM, the criteria for the disorder changed so much over time that it is impossible to make reasonable

comparisons across studies using the different diagnostic systems (Coccaro, 2000). In DSM-III, for example, IED could not be diagnosed in the presence of generalized aggression or impulsivity that occurred between aggressive episodes, leading to a significant underestimation of prevalence (Coccaro et al., 1998). Although this criterion was dropped from the DSM-IV, other uncertainties about diagnostic thresholds continue to remain, such as the frequency and severity of aggressive acts needed to meet criteria for a diagnosis of IED. The DSM-IV requires "several discrete episodes of failure to resist aggressive impulses that result in serious assaultive acts or destruction of property" without any requirement for temporal clustering of the episodes, but it is unclear whether this is the best threshold. This kind of uncertainty, coupled with the changing diagnostic criteria across successive DSM revisions,

has resulted in a dearth of knowledge about the epidemiology of IED in the general population.

Despite these diagnostic uncertainties, clinical studies of patients who clearly meet diagnostic criteria for IED show them to be characterized by recurrent, impulsive acts of aggression (Coccaro et al., 1998; McCloskey et al., 2006; Posternak & Zimmerman, 2002) and to have high scores on scales of impulsivity (Coccaro et al., 1998) and trait hostility (Coccaro et al., 1998; McCloskey et al., 2006; Posternak & Zimmerman, 2002). Although not required to meet diagnostic criteria for the disorder, patients with IED also report high levels of distress and functional impairment (Coccaro et al., 2004; Fincham et al., 2009; McCloskey et al., 2006; McElroy et al., 1998).

The aggressive acts committed by people with IED often occur in response to interpersonal provocation (Coccaro, 2000; McElroy et al., 1998). Individuals with IED have been found to respond to even minor provocation with aggressive behavior (McCloskey et al., 2006). Indeed, elevated retaliatory aggression in response to provocation may represent a core feature of the disorder. In one study, patients with IED were much more likely than those with other Axis I and Axis II disorders and those without a mental disorder to respond to provocation with extreme aggression in a laboratory-based behavioral task but not to differ in aggressive behavior from others in situations that did not involve provocation (McCloskey et al., 2006). The aggressive acts committed by individuals with IED typically have a rapid onset and are short in duration, most often triggered by provocations from people with whom the individual is acquainted (Coccaro et al., 2004; McElroy et al., 1998).

Patients with IED report that feelings of pent-up tension or distress sometimes increase to a point of explosion just prior to their attacks (McElroy, 1999) and that a sense of release or relief occurs once the attacks occur (McElroy et al., 1998). Although the attacks are sometimes described as being pleasurable when they occur because of the tension release, this is not true in the long term, as they are subsequently associated with remorse. High emotional reactivity, predisposition to aggressive responses to distress, low impulse control, and involuntary control of attention all appear to be involved in these attacks (Coccaro, 2003; Felthous & Barratt, 2003; Koelsch, 2009).

Only a handful of epidemiological studies have examined the prevalence and correlates of IED. The focus of the current report is on data from a large

national epidemiological survey carried out in the United States: the National Comorbidity Survey Replication (NCS-R; Kessler & Merikangas, 2004). Some data are also presented from the World Health Organization World Mental Health (WMH) surveys, a series of epidemiological surveys carried out in nine countries that assessed DSM-IV IED. Only two previous surveys had estimated the prevalence of DSM-IV IED (Coccaro et al., 2004; Posternak & Zimmerman, 2002). One of them was a survey of 1300 patients in a university private practice clinic. The point prevalence of IED in that survey was 3.1% (Posternak & Zimmerman, 2002). The second was a study carried out in a nonprobability subsample of 253 respondents in the Baltimore Epidemiologic Catchment Area (ECA) Follow-Up study. The lifetime and 1-month prevalence estimates of IED in that sample were 4.0% and 1.6%, respectively; Coccaro et al., 2004). Retrospective lifetime reports in the second sample suggested that IED has an early age of onset (usually in childhood or adolescence) and a persistent course associated with significant psychosocial impairment and low rates of treatment.

Epidemiological Samples

The NCS-R was a nationally representative, face-to-face household survey ($n = 9282$) conducted between February 2001 and April 2003 in a multi-stage clustered area probability sample of the continental United States (Kessler et al., 2003, 2004b). The response rate was 70.9%. Details about the NCS-R design have been reported elsewhere (Kessler et al., 2004b) and will not be repeated here. The WMH surveys were replications and extensions of the NCS-R carried out in a number of other countries throughout the world (Kessler & Üstün, 2008), six of which included an assessment of IED. These six included surveys in Columbia, Iraq, Lebanon, Mexico, the People's Republic of China (PRC), Romania, and Ukraine. A total of 33,180 adults (age 18+) participated across the seven surveys (Table 12.1). All these surveys involved nationally representative household samples except Colombia, which was based on a nationally representative sample of households in urban areas. Weights were used to adjust for differential probabilities of selection within households and to match the samples to population sociodemographic distributions. The weighted average response rate across these surveys was 77.0%, with a range from 70.0% (Lebanon) to 95.2% (Iraq). Further details about the methodology of the WMH surveys are available elsewhere (Heeringa et al., 2008).

Table 12.1 WMH Sample Characteristics in Countries That Assessed IED

Country	Survey ¹	Sample Characteristics ²	Field Dates	Age Range	Sample Size				Response Rate ⁴
					Part I	Part II	Part II and Age ≤ 44 ³		
Colombia	NSMH	Stratified multistage clustered area probability sample of household residents in all urban areas of the country (approximately 73% of the total national population)	2003	18–65	4426	2381	1731	—	87.7
Iraq	IMHS	Stratified multistage clustered area probability sample of household residents. NR	2007	18+	4332	4332	—	—	95.2
Lebanon	LEBANON	Stratified multistage clustered area probability sample of household residents. NR	2002–3	18+	2857	1031	595	—	70.0
PRC	B-WMH S-WMH	Stratified multistage clustered area probability sample of household residents in the Beijing and Shanghai metropolitan areas.	2002–3	18+	5201	1628	570	—	74.7
Romania	RMHS	Stratified multistage clustered area probability sample of household residents. NR	2005–6	18+	2357	2357	—	—	70.9
Ukraine	CMDPSD	Stratified multistage clustered area probability sample of household residents. NR	2002	18+	4725	1720	541	—	78.3
United States	NCS-R	Stratified multistage clustered area probability sample of household residents. NR	2002–3	18+	9282	5692	3197	—	70.9
Total					33180	19141	6634		

¹NSMH (The Colombian National Study of Mental Health); IMHS (Iraq Mental Health Survey); LEBANON (Lebanese Evaluation of the Burden of Ailments and Needs of the Nation); B-WMH (The Beijing World Mental Health Survey); S-WMH (The Shanghai World Mental Health Survey); RMHS (Romania Mental Health Survey); CMDPSD (Comorbid Mental Disorders during Periods of Social Disruption); NCS-R (The US National Comorbidity Survey Replication).

²Most WMH surveys are based on stratified multistage clustered area probability household samples in which samples of areas equivalent to counties or municipalities in the United States were selected in the first stage followed by one or more subsequent stages of geographic sampling (e.g., towns within counties, blocks within towns, households within blocks) to arrive at a sample of households, in each of which a listing of household members was created and one or two people were selected from this listing to be interviewed. No substitution was allowed when the originally sampled household resident could not be interviewed. These household samples were selected from census area data. In all, 6 of the 10 surveys are based on nationally representative (NR) household samples.

³Iraq and Romania did not have an age-restricted Part II sample. All other countries, with the exception of the People's Republic of China and Ukraine (which were age restricted to ≤ 39), were age restricted to ≤ 44.

⁴The response rate is calculated as the ratio of the number of households in which an interview was completed to the number of households originally sampled, excluding from the denominator households known not to be eligible either because of being vacant at the time of initial contact or because the residents were unable to speak the designated languages of the survey. The weighted average response rate is 77.0%.

Diagnostic Assessments

Diagnoses in the NCS-R and WMH surveys were based on Version 3.0 of the World Health Organization Composite International Diagnostic Interview (CIDI; Kessler & Üstün, 2004), a fully structured, lay-administered diagnostic interview that generates diagnoses according to DSM-IV (American Psychiatric Association, 1994) criteria. In addition to IED, the CIDI assessed lifetime and recent prevalence of mood disorders (major depressive disorder or dysthymic disorder, bipolar I-II disorder), anxiety disorders (panic disorder, generalized anxiety disorder, phobias, posttraumatic stress disorder, obsessive-compulsive disorder, separation anxiety disorder), other impulse control and behavioral disorders (oppositional-defiant disorder, conduct disorder, eating disorders, pathological gambling disorder), and substance disorders (alcohol and drug abuse with or without dependence). As detailed elsewhere (Haro et al., 2006; Kessler et al., 2004a), blind clinical reinterviews using the Structured Clinical Interview for DSM-IV (SCID; First et al., 2002) as the gold standard carried out in a probability subsample of respondents from the NCS-R and several WMH surveys found generally good concordance between DSM-IV diagnoses based on the CIDI and independent diagnoses based on the SCID.

DSM-IV Criterion A for IED requires the occurrence of several episodes of failure to resist the impulse to engage in aggressive behavior that result in serious violent acts or destruction of property. The CIDI operationalized this criterion by requiring the respondent to report at least one of three types of anger attacks: (1) “when all of a sudden you lost control and broke or smashed something worth more than a few dollars”; (2) “when all of a sudden you lost control and threatened to hit or hurt someone”, and (3) “when all of a sudden you lost control and hit or tried to hurt someone.” Three or more lifetime attacks were required to meet the DSM-IV requirement of “several” attacks. A second, narrower definition of lifetime IED required that three or more attacks occurred in the same year. We defined 12-month prevalence using three successively more stringent requirements. The most inclusive definition required three lifetime attacks and at least one attack in the past 12 months. The intermediate definition required three lifetime attacks in the same year and at least one attack in the past 12 months. The narrowest definition required three attacks in the past 12 months.

DSM-IV Criterion B requires that the aggressive behavior is out of proportion to the provocation or

precipitating stressors. This criterion is operationalized in the CIDI by requiring the respondent to report either that he or she “got a lot more angry than most people would have been in the same situation,” that the attack occurred “without good reason,” or that the attack occurred “in situations where most people would not have had an anger attack.” Finally, the CIDI operationalizes Criterion C, requiring that the aggressive behavior is not better accounted for by another mental disorder, the physiological effects of a substance, or a general medical condition by excluding all respondents who either have a lifetime history of bipolar disorder, reported that their anger attacks occurred only when they had been drinking or using drugs or when they were involved in an episode of depression, or reported that their anger attacks were due to organic causes.

Prevalence of Anger Attacks in the United States

Nearly half of all adults in the NCS-R (46.0%) reported experiencing at least one anger attack at some time in their life that involved either destroying property, threatening interpersonal violence, or engaging in interpersonal violence (Table 12.2). Attacks that involved threatening violence were most common (38.8%), followed by attacks that involved actual violence (25.8%) and destroying property (24.5%). A majority of respondents who reported anger attacks (65.9%) had attacks that involved more than one of these three kinds of behavior.

Approximately one-quarter of NCS-R respondents (25.1%) reported having three or more anger attacks in their lifetime. Respondents who reported attacks involving all three types of assaultive behavior represented the most common profile among those with three or more lifetime attacks (35.5%). A smaller proportion of NCS-R respondents reported having three or more lifetime attacks that involved anger out of proportion to the precipitating stressors (15.8%). An even smaller proportion of respondents reported three or more lifetime attacks that both involved disproportionate anger and loss of control (8.5%).

The vast majority (87%) of the NCS-R respondents who reported three or more lifetime attacks that both involved disproportionate anger and loss of control met full lifetime criteria for a broadly defined DSM-IV diagnosis of IED, the others being excluded either because all their attacks occurred as a result of alcohol or drug use, in the context of a

Table 12.2 Lifetime Prevalence of Anger Attack Types and Profiles in the NCS-R (*n* = 9282)

	Prevalence		At Least Three Attacks		At Least Three Attacks and Out of Proportion		At Least Three Attacks and Out of Control	
	% ¹	(se)	% ¹	(se)	% ¹	(se)	% ¹	(se)
I. Types								
Broke things	24.5	(0.5)	15.2	(0.4)	11.0	(0.5)	6.5	(0.4)
Threatened people	38.8	(1.1)	21.8	(0.6)	13.6	(0.5)	7.5	(0.4)
Physically attacked people	25.8	(0.8)	14.8	(0.5)	9.6	(0.5)	5.9	(0.4)
Any	46.0	(1.0)	25.1	(0.6)	15.8	(0.5)	8.5	(0.4)
II. Profiles								
Only broke	7.2	(0.3)	3.3	(0.2)	2.2	(0.2)	1.0	(0.1)
Only threatened	8.5	(0.4)	4.1	(0.2)	1.8	(0.1)	0.6	(0.1)
Broke and threatened	4.6	(0.3)	2.9	(0.2)	2.1	(0.2)	1.0	(0.1)
Threatened and attacked	13.0	(0.6)	5.9	(0.3)	2.9	(0.2)	1.4	(0.2)
All three	12.8	(0.5)	8.9	(0.4)	6.7	(0.4)	4.4	(0.3)

¹Assessed among the NCS-R total Part I sample; that is, in the first row, 24.5% is the percent that broke something among the Part I sample; 15.2% is the percent that broke something *and* at least had three attacks among the total Part I sample, etc.

Table 12.3 Distribution of Lifetime Anger Attacks in the NCS-R (*n* = 9282)

	Mean # of Attacks		Range		Interquartile Range		Total # of Attacks (prevalence *mean)	Distribution of Attacks in the Total Population			
	Prevalence ¹	AVG	(se)	Low	High	25.0%	75.0%	%			
				Extreme	Extreme						
1–2 lifetime attacks	20.8	(0.7)	1.4	(0.0)	1	2	1.0	2.0	(30.1)	4.5	(0.4)
3+ attacks not out of proportion	9.3	(0.4)	16.6	(2.5)	3	500	3.0	10.0	(155.0)	23.4	(3.3)
3+ out-of-proportion attacks not out of control	7.3	(0.3)	20.7	(1.7)	3	500	4.0	10.0	(150.7)	22.7	(2.5)
3+ out-of-proportion/out-of-control attacks disqualified due to diagnostic hierarchy and/or organic exclusions	1.0	(0.1)	33.3	(8.8)	3	500	4.0	15.0	(34.1)	5.1	(1.5)
Broadly defined IED	7.4	(0.4)	39.4	(4.4)	3	500	5.0	20.0	(292.7)	44.2	(3.0)
Total	46.0	(1.0)	14.4	(1.0)	1	500	1.0	6.0	(662.5)	100.0	(0.0)

¹Assessed among the NCS-R total Part I sample.

manic or hypomanic or depressive episode, or as a result of an organic cause. The 7.4% of the NCS-R sample with lifetime IED represent only about 16% of all people who reported ever having a lifetime anger attack. However, they account for nearly half (44%) of all anger attacks in the population (Table 12.3). This high percentage is due to the fact that people with IED reported an average of 39.4 lifetime attacks, whereas the majority of other people with anger attacks reported only 1 or 2 lifetime attacks (20.8% of the sample).

Prevalence and Onset of IED in the United States

Lifetime prevalence estimates of broadly and narrowly defined IED (with standard errors in parentheses) in the NCS-R are 7.4% (0.4) and 5.5% (0.3), respectively. Twelve-month prevalence estimates are 4.1% (0.3) using the broad definition, 3.6% (0.3) using the intermediate definition, and 2.8% (0.3) using the narrow definition. Mean age of onset (AOO) of the first anger attack is in early adolescence for both narrowly defined lifetime cases (13.5) and for cases that meet only the broad lifetime definition (broad-only; 14.0; $\chi^2_1 = 2.5, p = .12$). The full AOO distributions are also quite similar for narrow and broad-only lifetime cases as well as for males and females of each type (Figure 12.1).

The majority of people with lifetime narrow (67.8%) and broad-only (71.2%) IED have a history of interpersonal violence during their anger attacks, while most others (20.9% narrow, 14.9% broad-only) have a history of threatening interpersonal

violence during their attacks. Only a small minority of respondents (11.4% narrow, 13.9% broad-only) reported attacks that never included either interpersonal violence or threats of interpersonal violence.

Lifetime Persistence and Severity of IED in the United States

Narrowly defined lifetime IED in the NCS-R is significantly more persistent than broad-only IED. This can be seen indirectly by calculating the ratios of any 12-month anger attack to the lifetime prevalence estimates reported in the previous section. These ratios are 49.8% (2.7) for narrow and 26.7% (3.6) for broad-only lifetime IED ($z = 5.4, p < .001$). Higher persistence of narrow than broad-only cases can be seen more directly by comparing mean number of lifetime attacks (50.6 vs. 6.2; $z = 11.4, p < .001$), mean number of years with at least one attack (11.5 vs. 6.2; $z = 6.9, p < .001$), and highest number of attacks in a single year (28.6 vs. 1.6; $z = 19.9, p < .001$; Table 12.4). Persistence is greatest among respondents whose attacks feature both interpersonal violence and property damage (e.g., an average of 59.7 lifetime attacks versus 24.4–30.2 in other subgroups; $F_{4,620} = 6.8, p < .001$).

Narrow cases are also more severe, on average, than broad-only cases, as indicated both by a higher mean monetary value of objects damaged during anger attacks (\$1574.30 vs. \$443.40; $z = 4.8, p < .001$) and by a higher mean number of times someone needed medical attention because of an anger attack (224.0 vs. 36.7 times per 100 cases; $z = 3.4, p < .001$). Severity, like persistence, is highest

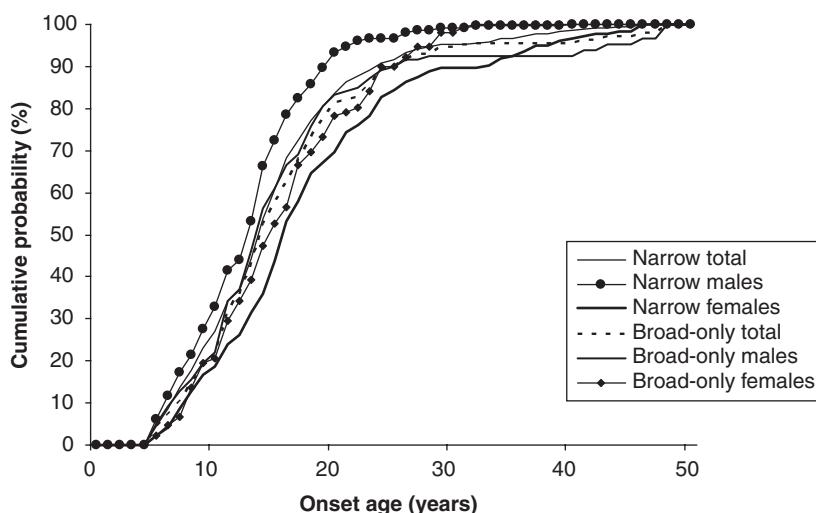


Fig. 12.1 Age of onset distributions of narrow and broad-only lifetime DSM-IV intermittent explosive disorder separately among males ($n = 4139$) and females ($n = 5143$) as well as overall ($n = 9282$).

Table 12.4 Course and Severity of Lifetime DSM-IV/CIDI IED in the NCS-R

	Narrow ¹		Broad-Only ¹		Broad ¹	
	Mean	(se)	Mean	(se)	Mean	(se)
I. Course						
Number of lifetime attacks	50.6*	(6.0)	6.2	(0.4)	39.5	(4.4)
Number of years with attacks	11.5*	(0.5)	6.2	(0.5)	10.2	(0.4)
Highest number of annual attacks	28.6*	(4.3)	1.6	(0.0)	21.8	(3.0)
II. Severity						
Property damage (\$) ²	1574.3	(135.6)	443.4	(132.6)	1340.7	(109.9)
Medical attention (per 100 cases) ³	224.0	(49.6)	36.7	(12.8)	176.7	(36.8)
(n)	(502)		(178)		(680)	

*Significant difference in means between the narrow and broad-only subsamples at the .05 level, two-sided test.

¹Narrow = three or more annual attacks in at least 1 year of life; Broad-only = three or more lifetime attacks without ever having as many as three attacks in a single year; Broad = Narrow or Broad-only.

²Estimated cost of all the things ever damaged or broken in an anger attack.

³Number of times during an anger attack that someone was hurt badly enough to need medical attention per 100 cases of IED.

Source: A version of this table appeared previously in Kessler, R. C., Coccato, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. E. (2006). The prevalence and correlates of DSM-IV Intermittent Explosive Disorder in the National Comorbidity Survey Replication. Archives of General Psychiatry, 63(6), 669–678, © 2006 American Medical Association, Used with permission.

among respondents whose attacks feature both violence and property damage (e.g., an average of \$1780 in property damage versus \$462–\$463 in other subgroups that included property damage; $F_{2,622} = 37.6$, $p < .001$; and an average of 180 instances of someone requiring medical attention per 100 cases versus 34–229 in other subgroups that included violence; $F_{2,622} = 14.2$, $p = .001$). (More detailed results are available on request.) It is important to note, though, that these differences can be explained by the frequency of attacks. Indeed, the mean value of lifetime property damage *per attack* is actually lower for narrow IED (\$31) than for broad-only IED (\$72). The same is true for injuries requiring medical attention (4.4 per 100 attacks for narrow IED and 5.9 for broad-only IED).

Twelve-Month Duration and Role Impairment of IED in the United States

The average number of anger attacks in the past year is much higher for 12-month narrow (19.5) than intermediate-only (1.5) or broad-only (1.3) cases ($F_{2,347} = 28.1$, $p < .001$; Table 12.5). Similar variation exists in number of weeks with an attack ($F_{2,347} = 33.1$, $p < .001$). Severe 12-month role impairment, as assessed by the Sheehan Disability Scales (SDS), in comparison, varies much less across the three 12-month IED subsamples. In fact, the proportion of 12-month cases reporting severe role impairment

during the worst month of the year does not differ meaningfully across these subsamples for three of the four SDS domains ($F_{2,347} = 0.5$ –1.6, $p = .21$ –.60). The exception is the domain of interpersonal relationships, where severe impairment is considerably more common for narrow (26.5%) and intermediate-only (18.1%) than broad-only (11.6%) cases ($F_{2,347} = 3.7$, $p = .033$).

Sociodemographic Correlates

Statistically significant sociodemographic correlates of broadly defined lifetime IED in the NCS-R include being male, young, “other” race-ethnicity (i.e., not Non-Hispanic Black, Non-Hispanic White, or Hispanic), having low education, never having been married, not retired, not a homemaker, and having low family income (Kessler et al., 2006). The odds ratios (ORs) for these sociodemographic correlates were mostly modest in magnitude (1.5–2.0), with the exception of age (1.6–4.3), where the contrast category of respondents ages 60+ has a very low reported prevalence (2.1%). Investigation of this association in a survival framework showed that the lifetime risk of IED based on retrospective age-of-onset reports was inversely related to the age at interview. One plausible interpretation of this finding is that the prevalence of IED might have increased over time in the United States among people in the age range of the NCS-R.

Table 12.5 Duration and Impairment of 12-Month DSM-IV/CIDI IED in the NCS-R

	Narrow ¹	Intermediate-Only ¹		Broad-Only ¹		Broad ¹		
	Mean/% ²	(se)	Mean/% ²	(se)	Mean/% ²	(se)	Mean/% ²	(se)
I. Twelve-month persistence								
Number of 12-month attacks	19.5*	(2.4)	1.5	(0.1)	1.3	(0.1)	13.8	(1.7)
Number of weeks with attacks	12.1*	(1.3)	1.3	(0.1)	1.3	(0.1)	8.7	(0.9)
II. Severe role impairment (Sheehan Disability Scales)								
Home	14.2	(2.6)	10.7	(3.9)	4.3	(3.0)	12.2	(1.9)
Work	11.1	(2.4)	11.9	(4.0)	6.8	(3.4)	10.7	(2.1)
Interpersonal	26.5*	(3.7)	18.1	(5.4)	11.6	(4.6)	23.0	(3.2)
Social	22.1	(3.3)	16.8	(4.8)	15.3	(5.0)	20.2	(2.7)
Summary	39.8*	(3.6)	25.2	(5.6)	21.3	(6.4)	34.7	(2.9)
(n)	(247)		(75)		(56)		(378)	

*Significant difference in prevalence across the narrow, intermediate-only, and broad-only subsamples at the .05 level, two-sided test.

¹Narrow = three or more 12-month attacks; Intermediate-only = three or more lifetime attacks in a single year (lifetime narrow) and one or two 12-month attacks; Broad-only = three or more lifetime attacks without ever having more than three in a single year (lifetime broad) and having one or two 12-month attacks; Broad = Narrow or Intermediate-only or Broad-only.

²Assessed among the NCS-R total Part I sample. The top two rows (number of 12-month attacks, number of weeks with attacks) are continuous variables, and the means and standard error are displayed. The final five rows are dichotomous variables, and the percent and standard error are displayed.

Source: A version of this table appeared previously in Kessler, R. C., Coccaro, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. E. (2006). The prevalence and correlates of DSM-IV Intermittent Explosive Disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 63(6), 669–678. © 2006 American Medical Association. Used with permission.

Another possibility, though, is that older NCS-R respondents underreported their IED more than younger respondents. If this is the case, though, then the prevalence of IED is substantially underestimated in the NCS-R, as the reported prevalence among respondents in the age range 18–29 is 12.2%. In either case, this result suggests that IED is a considerably larger problem than implied by the total-sample prevalence estimate.

Gender is of special interest in light of broader evidence that males have a higher risk than females of impulsive aggression (Struber et al., 2008). We found, consistent with this evidence, that males have a significantly higher odds of lifetime IED than females (OR 1.7, 95% CI 1.4–2.0). This difference is driven entirely, though, by the fact that males are more likely than females to have at least one anger attack ever in their life (OR 1.6, 95% CI 1.5–1.8). Among respondents who reported at least one lifetime anger attack, though, there were no gender differences in progression to three or more attacks or to attacks that were out of control. However, males with IED reported that their attacks were more severe than those of females with IED in terms of

extent of property damage, physical damage, and subjectively reported role impairment.

Among NCS-R respondents who met broadly defined lifetime criteria for IED, none of the sociodemographic variables considered in our analysis, including gender, distinguished narrowly defined cases from other broadly defined cases. Nor were any significant sociodemographic correlates found for 12-month persistence among lifetime cases. These results, taken together with the fact that the sociodemographic ORs predicting broadly defined lifetime IED were all quite modest in magnitude, suggests that IED is widely distributed throughout the U.S. population.

Comorbidity of IED with Other DSM Disorders in the United States

The vast majority (81.9%) of respondents with lifetime broadly defined IED in the NCS-R met criteria for at least one of the other lifetime DSM-IV disorders assessed in the NCS-R (Table 12.6). Indeed, broadly defined lifetime IED was significantly and positively related to each of these other disorders after controlling for age, sex, and

race-ethnicity, with ORs in the range 2.2–4.0. The ORs involving narrowly defined IED were consistently higher than those involving other broadly defined cases of IED, but the ratios of these two ORs are elevated only modestly with mood disorders (1.2–1.3) and most anxiety disorders (1.0–1.8). The ratios are more substantially elevated, in comparison, with specific phobia (1.6) and generalized anxiety disorder (2.0), all the impulse control disorders (2.0–2.6), and alcohol abuse (2.7).

We also examined comorbidity of 12-month IED with other 12-month DSM-IV disorders among respondents with a lifetime history of both disorders in the pair. Sparse data made it necessary to focus on broad disorder classes (i.e., any mood disorder, any anxiety disorder, any substance use disorder). As with lifetime comorbidity, ORs involving broadly defined IED were meaningfully elevated (mood 2.7, anxiety 2.2, substance 2.2), while ORs involving intermediate and narrowly defined 12-month cases were generally similar in magnitude to those of broadly defined IED.

Gender differences in comorbidity of broadly defined IED with other disorders were found to be significant but to differ by type of disorder. Associations of IED with impulse control and

substance use disorders were significantly stronger among males than females (OR 1.8, 95% CI 1.2–2.6 for impulse control disorders; OR 1.8, 95% CI 1.3–2.4 for substance disorders). Associations of IED with anxiety and mood disorders, in comparison, were significantly weaker among males than females (OR 0.5, 95% CI 0.3–0.7 for anxiety disorders; OR 0.4, 95% CI 0–0.6 for mood disorders).

Treatment of IED in the United States

Although roughly two-thirds of respondents with broad lifetime IED received lifetime treatment for emotional problems, only a minority (28.8%) were ever treated specifically for IED (Kessler et al., 2006). The probability of receiving treatment overall as well as within particular services sectors did not differ significantly, depending on the breadth of diagnostic criteria. One-third (33.6%) of respondents with broadly defined 12-month IED received treatment for emotional problems in the year before the interview, but only one-third of that number (11.7% of all 12-month cases) received treatment specifically for IED. As with lifetime treatment, probabilities of overall and sector-specific 12-month treatment did not differ significantly across cases that met broad, intermediate, or narrow diagnostic criteria.

Table 12.6 Lifetime Comorbidity of DSM-IV/CIDI IED with Other DSM-IV/CIDI Disorders in the NCS-R

	Broad ¹				Narrow ¹ : Broad-Only ^{1,2}			
	% ³	(se)	OR ⁴	(95% CI)	% ⁵	(se)	OR ⁶	(95% CI)
I. Mood disorders								
Major depressive disorder	37.6	(2.2)	3.0*	(2.4–3.6)	38.3	(2.6)	1.2	(0.7–2.0)
Dysthymia	9.8	(1.5)	3.3*	(2.4–4.5)	10.2	(1.7)	1.3	(0.7–2.6)
Any mood disorder	37.8	(2.1)	2.9*	(2.4–3.6)	38.5	(2.6)	1.2	(0.7–2.0)
II. Anxiety disorders								
Agoraphobia	6.5	(1.1)	3.5*	(2.3–5.2)	6.7	(1.3)	1.3	(0.6–3.0)
Generalized anxiety disorder	18.7	(1.8)	3.7*	(2.9–4.8)	20.6	(2.3)	2.0*	(1.3–3.1)
Obsessive compulsive disorder	6.3	(1.9)	2.7*	(1.4–5.2)	6.4	(2.4)	1.4	(0.3–7.3)
Panic disorder	11.7	(1.5)	3.3*	(2.3–4.7)	12.4	(1.7)	1.4	(0.8–2.6)
Post-traumatic stress disorder	15.2	(1.5)	3.1*	(2.3–4.2)	16.6	(2.0)	1.8	(1.0–3.2)
Social phobia	28.0	(1.5)	3.1*	(2.6–3.7)	28.9	(1.8)	1.3	(0.8–1.9)
Specific phobia	24.3	(1.7)	2.6*	(2.2–3.1)	26.2	(2.2)	1.6*	(1.0–2.5)
Separation anxiety disorder	10.5	(1.1)	3.0*	(2.2–4.0)	10.5	(1.4)	1.0	(0.5–1.8)
Any anxiety disorder	58.2	(1.8)	3.9*	(3.2–4.7)	60.3	(2.3)	1.5	(1.0–2.2)

(continued)

Table 12.6 Lifetime Comorbidity of DSM-IV/CIDI IED with Other DSM-IV/CIDI Disorders in the NCS-R (continued)

	Broad ¹				Narrow ¹ : Broad-Only ^{1,2}			
	% ³	(se)	OR ⁴	(95% CI)	% ⁵	(se)	OR ⁶	(95% CI)
III. Impulse-control disorders								
Oppositional defiant disorder	24.9	(2.2)	3.5*	(2.6–4.7)	27.8	(2.8)	2.0*	(1.1–3.6)
Conduct disorder	24.5	(2.5)	3.6*	(2.8–4.7)	27.5	(3.1)	2.1*	(1.2–3.7)
Attention-deficit/hyperactivity disorder	19.5	(2.0)	3.3*	(2.5–4.3)	22.3	(2.6)	2.6*	(1.4–4.9)
Any impulse-control disorder	45.1	(2.1)	4.1*	(3.3–5.1)	49.7	(2.7)	2.2*	(1.3–3.7)
IV. Substance use disorders								
Alcohol abuse	32.8	(3.0)	3.1*	(2.3–4.2)	37.4	(3.8)	2.7*	(1.7–4.2)
Alcohol dependence with abuse	17.0	(2.0)	3.6*	(2.6–5.1)	18.5	(2.5)	1.7	(1.0–2.8)
Drug abuse	21.7	(2.3)	2.7*	(2.0–3.7)	23.5	(3.1)	1.5	(0.9–2.7)
Drug dependence with abuse	10.6	(1.4)	3.5*	(2.4–5.2)	11.4	(1.8)	1.5	(0.7–3.2)
Any substance disorder	34.9	(3.0)	2.9*	(2.2–4.0)	39.3	(3.7)	2.4*	(1.6–3.8)
V. Any disorder								
At least one disorder	81.9	(2.0)	5.7*	(4.3–7.7)	84.4	(2.3)	1.8*	(1.1–3.0)
Exactly one disorder	16.0	(1.4)	0.9	(0.7–1.1)	14.0	(1.4)	0.6*	(0.3–1.0)
Exactly two disorders	17.0	(1.6)	1.8*	(1.4–2.4)	16.7	(2.1)	0.9	(0.5–1.6)
Three or more disorders	48.8	(2.6)	4.9*	(3.7–6.3)	53.7	(3.2)	2.3*	(1.5–3.6)
(n)	(5692)				(627)			

*Significant at the .05 level, two-sided test, controlling for age, sex, and race-ethnicity.

¹Narrow = three or more annual attacks in at least 1 year of life; Broad-only = three or more lifetime attacks without ever having as many as three attacks in a single year; Broad = Narrow or Broad-only.

²Narrow: Broad-only = comparing lifetime Narrow to lifetime Broad-only by restricting the sample to cases with lifetime Narrow or lifetime Broad-only and treating lifetime Broad-only as the reference category.

³Prevalence of the row variables among the column variables. For example, in the first row, there are 37.6% of people with lifetime broad IED with major depressive disorder, and there are 29.3% of males with lifetime broad IED with major depressive disorder.

⁴Bivariate logistic regression models controlling for age, sex, and race-ethnicity to predict the comorbidity of lifetime broad IED with other DSM-IV disorders.

⁵Prevalence of the row variables among lifetime Narrow IED. For example, in the first row, there are 38.3% of people with lifetime Narrow IED with major depressive disorder, and there are 31.4% of males with lifetime Narrow IED with major depressive disorder.

⁶Bivariate logistic regression models controlling for age, sex, and race-ethnicity to predict the comorbidity of lifetime Narrow IED with other DSM-IV disorders. The subsample is restricted to cases with either lifetime Narrow IED or lifetime Broad IED; lifetime Broad IED is the left-out category.

Source: A version of this table appeared previously in Kessler, R. C., Coccato, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. E. (2006). The prevalence and correlates of DSM-IV Intermittent Explosive Disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry* (63)6, 669–678. © 2006 American Medical Association. Used with permission.

Cross-national Prevalence of IED

Lifetime and 12-month prevalence were assessed only for broadly defined IED in the WMH surveys other than in the United States. Lifetime prevalence estimates ranged from a low of 1.3% in Romania to a high of 4.7% in Colombia (Table 12.7). These estimates indicate considerable variability in the

cross-national lifetime prevalence of IED. The lifetime prevalence estimate is higher in the United States (7.4%) than in any of the other WMH countries, although estimates in Colombia and Ukraine are closer to the U.S. estimate. The estimates in the remaining WMH countries are less than half of those in the United States. Less cross-national variability

Table 12.7 Estimated Prevalence and Median Age of Onset of Broadly Defined DSM-IV/CIDI IED in the WMH Surveys

	Lifetime Prevalence		Median Age of Onset (years)	12-Month Prevalence		Ratio 12 Month to Lifetime
	%	(se)		%	(se)	
Colombia ¹	4.7	(0.4)	19	2.9	(0.3)	0.62
Iraq ²	1.7	(0.2)	19	1.5	(0.2)	0.88
Lebanon ³	1.7	(0.5)	—	0.8	(0.2)	0.47
PRC BJ/S ⁴	1.9	(0.3)	15	1.2	(0.2)	0.63
Romania	1.3 ⁵	(0.3) ⁵	—	1.3 ⁶	(0.3) ⁶	0.99
Ukraine ⁷	4.3	(0.3)	21	2.8	(0.3)	0.65
United States ⁸	7.4	(0.4)	14	4.1	(0.3)	0.55

— Information not available.

¹Source: Posada-Villa et al. (2008).

²Source: Alhasnawi et al. (2009).

³Source: Karam et al. (2008).

⁴Source: Huang et al. (2008).

^{5,6}Sources: Florescu et al. (2009a, 2009b).

⁷Source: Bromet et al. (2008).

⁸Source: Kessler et al. (2008).

was found in 12-month prevalence estimates, which ranged from a low of 0.8% in Lebanon to a high of 2.9% in Colombia.

The ratio of 12-month to lifetime prevalence of IED, a rough indicator of disorder persistence, was higher in all but one of the WMH countries (ratios ranging from 0.62 to 0.99) than in the United States (0.55). The exception was Lebanon (0.47). These findings indicate that although the prevalence of IED is higher in the United States than in the other countries considered here, the disorder may be more persistent outside the United States. The AOO of IED was examined in four WMH countries (Colombia, Iraq, Beijing-Shanghai, and Ukraine). Median AOO was very similar to the U.S. estimates in all of these countries, with the earliest median in Beijing-Shanghai (age 15) and the latest in Ukraine (age 21).

Discussion

The findings reported here based on national epidemiological surveys from the United States and six other countries document that DSM-IV IED is a relatively common disorder, especially among young adults in the United States. No data are yet available from the other WMH surveys regarding age-related differences in IED prevalence to indicate if the evidence of a dramatically increasing prevalence among young people in the United States exists as well in

other countries. The U.S. prevalence estimates are equivalent to 11.5–16.0 million lifetime cases and 5.9–8.5 million 12-month cases, depending on whether we use broad or narrow criteria. These prevalence estimates are somewhat higher than those found in the two previously published U.S. studies of DSM-IV IED, although neither of those earlier studies was based on a national sample (Coccaro et al., 2004; Posternak & Zimmerman, 2002). The WMH estimates indicate that the prevalence of IED varies substantially across countries, although the narrower range of estimates for 12-month than lifetime prevalence raises a question about lifetime recall bias that needs to be examined in future investigations. This pattern again suggests that a focus on younger people, in whom recall bias is likely to be less pronounced, would be valuable.

As noted earlier, the finding of a higher IED prevalence among males than females is consistent with broader evidence that impulsive aggression is more common among males than females (Struber et al., 2008). This result needs to be interpreted with caution because the only previous study of IED in a community-based sample found no significant gender difference in the lifetime prevalence of the disorder (Coccaro et al., 2004). Prior work involving clinical samples also failed to find a gender difference in the prevalence of IED (Coccaro et al., 1998, 2005). The other sociodemographic correlates

of IED found here involving disadvantaged social status (i.e., low education and income, never married), though, are consistent with those found in previous studies (Coccaro, 2003). The strong inverse association with age is especially noteworthy because it indicates either that the prevalence of IED has been on the rise in recent cohorts or that sample selection bias or recall bias led to an underestimation of lifetime prevalence among older respondents. These possibilities both suggest that the lifetime prevalence of IED in current cohorts is likely to be considerably higher in recent cohorts than in the total sample.

A number of important issues remain unresolved regarding the diagnosis of IED. The first of these issues relates to the distinction between broad and narrow definitions of IED. The stipulation in DSM-IV that the presence of only three serious lifetime episodes of aggression may be sufficient to make the diagnosis of IED is one of the few instances in which DSM-IV does not have a temporal clustering requirement (e.g., three episodes in 1 year). It is noteworthy in this regard that even though the most severe form of IED in our study (narrow) is much more persistent than the less severe form (broad), the two did not differ significantly in most measures of functional impairment in the NCS-R. As such, these data raise questions as to when to treat individuals with IED. Prospective treatment data will be needed to resolve this uncertainty.

A second diagnostic issue involves the types of aggressive behaviors that should be included in Criterion A of the IED diagnosis. An alternative set of diagnostic criteria for IED has been proposed that extends the definition to include recurrent aggressive outbursts that do not include threatened or actual violence, assaultive behavior, or physical force (e.g., verbal aggression against others such as insults or arguments out of proportion to the provocation; Coccaro, 2000; Coccaro et al., 1998). Although such individuals were not included in the NCS-R or WMH analyses, other research has shown that such individuals have levels of anger, hostility, aggressive responses to provocation, and functional impairment equivalent to those of individuals who meet full DSM-IV criteria for IED (McCloskey et al., 2008). The Baltimore ECA study findings suggest that IED prevalence would have been roughly 25% higher if cases had been identified using these alternative criteria for IED (Coccaro et al., 1998). Because verbal aggression against others in the absence of threats, physical violence, or property destruction is significantly impairing and has been

shown to respond to psychopharmacological treatment (Coccaro et al., 2009), a rationale exists for including these behaviors in the definition of IED in DSM-V. In addition, at least two recent studies demonstrate that individuals who meet criteria for IED based on a modified criterion set (Integrated Research Criteria) that includes these nonassaultive/nondestructive behaviors have greater serotonergic dysfunction compared to individuals defined by DSM-IV criteria (Coccaro et al., 2010a, 2010b).

Another diagnostic issue concerns whether IED is sufficiently distinct from other mental disorders to warrant inclusion as a separate diagnostic entity in the DSM. The NCS-R results indicate substantial rates of comorbidity between IED and other Axis I and Axis II mental disorders. This is consistent with findings from a number of previous studies of IED in clinical samples (Coccaro et al., 1998; Fincham et al., 2009; McCloskey et al., 2006). Moreover, acts of impulsive aggression occur in a wide range of mental disorders other than IED, including substance use disorders and a number of personality disorders (Berman et al., 1998; Critchfield et al., 2008; Eronen et al., 1996; Fals-Stewart et al., 2005; Swanson et al., 1990). Aggressive behavior is a core feature of several mental disorders other than IED, including oppositional defiant disorder and conduct disorder in children and adolescents and antisocial personality disorder in adults (Fals-Stewart et al., 2005; Goldstein et al., 2006; Matthys et al., 1999; Schaeffer et al., 2003). Together, these findings raise questions about whether IED should be classified as a distinct mental disorder.

It is relevant in this regard that recent evidence from a taxometric analysis carried out in the Collaborative Psychiatric Epidemiological Surveys (CPES) indicated that IED is best characterized as taxonic (i.e., categorical rather than dimensional) and is distinguishable from nonpathological aggression and both antisocial and borderline personality disorders (Ahmed et al., 2010). The IED taxon was characterized by frequent anger attacks that were disproportionate to the provocation and involved both loss of control and negative social consequences. These findings suggest that IED represents a valid diagnostic category that is distinct from other disorders involving impulsivity and aggression. The base rate of IED in the population was estimated at 5.5% in the CPES study, which is identical to the lifetime prevalence of narrow IED documented in the NCS-R.

The NCS-R data are quite clear in showing that IED typically begins in adolescence, is associated

with substantial role impairment, and is highly comorbid with other DSM-IV mood, anxiety, and substance use disorders. Although these results cannot be compared directly with the results of clinical studies, it is worth noting that similar patterns have consistently been found in clinical studies of IED (Coccaro, 2000; Coccaro et al., 1998; Felthous et al., 1991; Lejoyeux et al., 1999; Monopolis & Lion, 1983; Zimmerman & Mattia, 2000). This greater severity, combined with the evidence of a relatively higher prevalence, argues that IED is an important disorder that has been comparatively neglected in epidemiological research.

The finding that IED typically begins in adolescence emerged consistently across the five countries where age of onset was retrospectively studied in the WMH series, as did the finding that IED is quite persistent over the life course. It is important to note, though, that this high persistence could have been overestimated to the extent that earlier lifetime prevalence was underreported by older respondents. If the latter was the case, the true lifetime prevalence would be higher than estimated here, while the true persistence would be lower than estimated here. There is no definitive way to adjudicate between these possibilities with the cross-sectional data available to us in the NCS-R and other WMH surveys.

The early age of onset of IED is an important finding with regard to comorbidity because it means that IED is temporally primary to many of the other DSM-IV disorders with which it is comorbid (Coccaro, 2003). This raises the possibility that IED might be either a risk factor or a risk marker for temporally secondary comorbid disorders (Kraemer et al., 1997). Consistent with this possibility, a recent family study showed that the children of depressed adults with anger attacks have higher rates of delinquency and aggressive behavior than the children of depressed adults without anger attacks (Alpert et al., 2003). This suggests that intermittent explosive behavior might emerge quite early in subjects at risk of the subsequent onset of mood disorders. However, we are aware of no systematic research on the possibility that IED is a risk marker for temporally secondary disorders. It is interesting to note in this regard that the one published study that examined the family aggregation of IED found high intergenerational continuity of the disorder independent of comorbid conditions (Coccaro, 2010), which means that common genetic factors are unlikely to account for the comorbidity of IED with other DSM disorders.

This last observation suggests that the association of IED with the later first onset of secondary comorbid disorders is unlikely to be due to common underlying genetic risk factors or to phenotypic factors that are under strong genetic control, such as an impulsive personality style. If IED is a causal risk factor, in comparison, it might promote secondary disorders by leading to divorce, financial difficulties, and stressful life experiences that promote secondary disorders. If this last scenario is correct, then the fact that so few people obtain treatment for IED becomes especially important because it means that an opportunity is being missed to intervene in the disorder at a point in time when it might still be possible to prevent the onset of secondary disorders. A related question for future research is whether successful early detection, outreach, and treatment of IED would help prevent the onset of secondary comorbid disorders. Given the age of onset distribution of IED, early detection would most reasonably take place in schools and might well be an important addition to ongoing school-based violence prevention programs (Flay et al., 2004; Meyer et al., 2004).

It is noteworthy that a detailed analysis of delays in seeking treatment for IED found that the minority of people with IED who obtain professional help for their anger attacks typically wait a decade or more after onset before first treatment contact (Wang et al., 2005). Given the differences in the typical age of onset of IED compared to temporally secondary comorbid disorders (Kessler et al., 2005), this means that initial treatment usually occurs only after the onset of most temporally secondary disorders and that the focus of treatment is probably on the comorbid disorders. This interpretation is consistent with the finding that the majority of people with IED were found to receive treatment for emotional problems at some time in their life, but not for their anger. It is not clear from this result whether the low rate of treatment of anger is due to greater reluctance to seek professional help for anger than other emotional problems or due to failure to conceptualize anger as a mental health problem. Given that so many people with IED obtain treatment for other emotional problems, the question can also be raised concerning why treating clinicians do not include anger as a focus of their treatment or whether the anger problems of their patients with IED are not recognized. We have no data in the NCS-R to adjudicate among these possibilities.

Acknowledgments

Portions of this chapter appeared previously in Kessler, R. C., Coccaro, E. F., Fava, M., Jaeger, S., Jin, R., & Walters, E. E. (2006). The prevalence and correlates of DSM-IV Intermittent Explosive Disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 63(6), 669–678, © 2006 American Medical Association, and in Kessler, R. C., Coccaro, E. F., & Fava, M. (2007). The prevalence and correlates of DSM-IV intermittent explosive disorder. *Directions in Psychiatry*, 27(4), 221–229, © 2007 Hatherleigh Company, and are reproduced here with the permission of the publishers. Preparation of this chapter was supported by NIMH Grant (U01-MH60220) with supplemental support from the National Institute on Drug Abuse (NIDA), the Substance Abuse and Mental Health Services Administration (SAMHSA), the Robert Wood Johnson Foundation (RWJF; Grant 044780), and the John W. Alden Trust as part of the activity of the National Comorbidity Survey Replication (NCS-R). A complete list of NCS publications and the full text of all NCS-R instruments can be found at <http://www.hcp.med.harvard.edu/ncs>. Send correspondence to ncs@hcp.med.harvard.edu. The NCS-R is carried out in conjunction with the World Health Organization World Mental Health (WMH) Survey Initiative. We thank the staff of the WMH Data Collection and Data Analysis Coordination Centres for assistance with instrumentation, fieldwork, and consultation on data analysis. These activities were supported by the National Institute of Mental Health (R01 MH070884), the John D. and Catherine T. MacArthur Foundation, the Pfizer Foundation, the U.S. Public Health Service (R13-MH066849, R01-MH069864, and R01 DA016558), the Fogarty International Center (FIRCA R01-TW006481), the Pan American Health Organization, Eli Lilly and Company, Ortho-McNeil Pharmaceutical, Inc., GlaxoSmithKline, and Bristol-Myers Squibb. A complete list of WMH publications can be found at <http://www.hcp.med.harvard.edu/wmh/>.

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Compulsive Sexual Behavior: Phenomenology and Epidemiology

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Abstract

Compulsive sexual behavior (CSB), a relatively common disorder, is characterized by having sexually related thoughts, urges, and behaviors that cause significant psychosocial distress and functional impairment. This chapter describes the phenomenology and etiology of CSB. Gender, age, and ethnic/cultural influences on CSB are discussed, as well as comorbidity issues and the relationship between CSB and Parkinson's disease. Addiction and obsessive-compulsive spectrum theoretical models of CSB are also summarized. Finally, the chapter provides future directions for clinicians and research to pursue.

Keywords: compulsive sexual behavior, phenomenology, epidemiology, comorbidity, theoretical models

Introduction

Compulsive sexual behavior (CSB), also referred to as *sexual addiction* and *hypersexual behavior* (for the purposes of this chapter, these terms will be used interchangeably), has a long history in the medical literature. In his book *Nymphomania or a Dissertation concerning the Furor Uterinus*, D. T. de Bienville, a French physician, asserted that overstimulation of a woman's nerves through impure thoughts, too much chocolate and rich food, or reading novels, might result in excessive sexual desire (Bienville, 1775). P. Henry Chavasse, in his book *Physical Life of Men and Women, or Advice to Both Sexes* (1871), argued that extreme sexual restraint may result in nymphomania and satyriasis (Chavasse, 1871). Jean-Etienne Dominique Esquirol (1838/1845) asserted that nymphomania was not due to a lack of sexuality, but rather was a physical disorder originating in the reproductive organs that affected the brain. In his book *Psychopathia Sexualis*, Richard von Krafft-Ebbing (1886), a German psychiatrist, described

the impact that excessive sexual behavior can have on an individual's life:

sexual instinct . . . [which] permeates all his thoughts and feelings, allowing of no other aims in life, tumultuously, and in a rut-like fashion demanding gratification without granting the possibility of moral and righteous counter-presentations, and resolving itself into an impulsive, insatiable succession of sexual enjoyment. (p. 70)

Although not currently classified as a formal psychiatric disorder according to the *Diagnostic and Statistical Manual for Mental Disorders* (DSM-IV-TR; APA, 2000), CSB is characterized by excessive, uncontrollable, culturally normative sexual behavior, urges, and/or thoughts that results in distress and functional impairment (Black et al., 1997; Coleman, 1992; Gerevich et al., 2005).

Diagnostic criteria for CSB have been proposed. Martin Kafka (2010) proposed the following diagnostic criteria, which reflect characteristics of

both substance use disorders and impulse control disorders:

A. Over a period of at least 6 months, recurrent and intense sexual fantasies, sexual urges, or sexual behaviors in association with 3 or more of the following 5 criteria:

A1. Time consumed by sexual fantasies, urges or behaviors repetitively interferes with other important (non-sexual) goals, activities and obligations.

A2. Repetitively engaging in sexual fantasies, urges or behaviors in response to dysphoric mood states (e.g., anxiety, depression, boredom, irritability).

A3. Repetitively engaging in sexual fantasies, urges or behaviors in response to stressful life events.

A4. Repetitive but unsuccessful efforts to control or significantly reduce these sexual fantasies, urges or behaviors.

A5. Repetitively engaging in sexual behaviors while disregarding the risk for physical or emotional harm to self or others.

B. There is clinically significant personal distress or impairment in social, occupational or other important areas of functioning associated with the frequency and intensity of these sexual fantasies, urges or behaviors.

C. These sexual fantasies, urges or behaviors are not due to the direct physiological effect of an exogenous substance (e.g., a drug of abuse or a medication) (p. 379).

Alternatively, Donald Black and colleagues (1997) have proposed that sexual preoccupation or excessive sexual behavior accompanied by either subjective distress or impairment are adequate criteria for diagnosing CSB.

Epidemiology

Compulsive sexual behavior appears to be relatively common, with an estimated adult lifetime prevalence of about 3% to 6% (Carnes, 1991; Coleman, 1992). No large-scale epidemiological studies, however, have been conducted to measure the prevalence of CSB. One study assessing impulse control disorders on a college campus ($n = 791$; 67.9% female), found that 3.7% of students reported symptoms consistent with current CSB (Odlaug & Grant, 2010). Evaluating the prevalence of CSB is difficult due to the embarrassment and shame frequently reported by those with CSB and society's judgmental position toward the expression of sexuality

(Black et al., 1997). Therefore, there is a need for comprehensive research to be conducted in order to determine accurate rates of CSB.

There is also a lack of research investigating the prevalence of CSB within various psychiatric populations, despite the high levels of comorbidity (see the "Clinical Characteristics" section). Research has found that lifetime rates of CSB (4.9%) in an adult psychiatric inpatient sample ($n = 204$; 54.9% female; mean age = 40.5 ± 13.2) were the same as current rates of CSB (4.9%) in an adolescent psychiatric inpatient sample ($n = 102$; 54.9% female; mean age = 15 ± 1.4 ; Grant et al., 2005, 2007). These findings highlight future research needs to evaluate the rates of CSB within various clinical populations in order to gain a better understanding of CSB.

Clinical Characteristics

The majority of treatment-seeking individuals with CSB are males (Black et al., 1997; Carnes, 1991; Raymond et al., 2003) with a primary onset of compulsively sexual behaviors during late adolescence (Black et al., 1997; Kafka, 1997). In a cross-sectional study consisting of 36 subjects (mean age = 27 ± 8 ; 72% male) with CSB recruited through newspaper advertisements, the majority reported experiencing a preoccupation with sexual fantasies or being overly sexually active (92%), uncontrollable, distress-producing sexual urges (67%), and engaging in repetitive, uncontrollable, distress-producing sexual behavior (67%; Black et al., 1997). A sample of 25 subjects with CSB (mean age = 38 ± 11 ; 92% male) recruited through newspaper advertisements reported having sexually related thoughts (64%) and urges (44%) and engaging in sexual behaviors (52%) at least once a day, while less than half (40%) reported having good control over their thoughts. However, the majority of subjects (80%, 72%, and 68%) spent 60 minutes or less having CSB-related thoughts, urges, and resisting urges, respectively (Raymond et al., 2003).

Individuals with CSB cite certain mood states triggering sexual behavior (96%), most commonly sadness or depression (67%), happiness (54%), or loneliness (46%; Black et al., 1997). Furthermore, the majority of subjects in the Black et al. (1997) sample (64%) reported a dissociative state while participating in compulsive sexual behaviors. After engaging in the behavior, most felt shame (79%) and experienced a negative mood change for periods ranging from minutes to days (75%; Black et al., 1997). Even though the individuals dislike

behaving and thinking in such a manner, it distracts them from other concerns, reduces anxiety or tension, improves mood, and makes them feel important, powerful, excited, and gratified (Black et al., 1997; Kafka & Prentky, 1997).

Additionally, individuals with CSB report that significant marital, occupational, and financial difficulties are consequences of their sexual urges and behaviors (Coleman et al., 2003) as well as significant distress caused by the amount of time consumed by their sexually related urges, thoughts, behaviors, out-of-control feelings, and postbehavior guilt (Black et al., 1997; Raymond et al., 2003).

Types of CSB

Research has shown that individuals with CSB have a mean of three different compulsive sexual behaviors (Carnes, 1991; Schneider & Schneider, 1996). The most commonly reported compulsive sexual behaviors are masturbation (17%–75%; Black et al., 1997; Briken et al., 2007; Kafka, 1997; Kafka & Hennen, 1999; Kafka & Prentky, 1992; Raymond et al., 2003; Reid et al., 2009; Wines, 1997); compulsive use of pornography (48.7%–54%; Black et al., 1997; Briken et al., 2007; Kafka, 1997; Kafka & Hennen, 1999; Kafka & Prentky, 1992; Reid et al., 2009); and protracted promiscuity/compulsive cruising and multiple relationships (22%–76%; Black et al., 1997; Briken et al., 2007; Kafka, 1997; Kafka & Prentky, 1992; Kafka & Hennen, 1999; Raymond et al., 2003; Wines, 1997). Researchers have identified seven types of nonparaphilic CSB, including compulsive cruising and multiple partners, compulsive fixation on an unattainable partner, compulsive autoeroticism, compulsive use of erotica, compulsive use of the Internet, compulsive multiple love relationships, and compulsive sexuality in a relationship (Coleman, 1992; Coleman et al., 2003).

Use of the Internet

The Internet is another avenue for CSB because it is a “Triple-A Engine,” as referred to by Cooper (1998), highlighting the Internet’s access (available 24 hours a day, 7 days a week), affordability (the plethora of free and low-cost sex websites), and anonymity (belief that no one else knows one’s true identity). About 72 million people visit pornography websites annually, and research has shown that 10%–17% of those who use the Internet for sexual purposes have online sexual problems (Carnes et al., 2007; Cooper et al., 2000). Cooper and colleagues (2000) defined cybersex compulsion as spending

11 or more hours online per week for sexual activities. Studies have shown that sexual compulsion is strongly related to the amount of time spent pursuing online sexual activities (Cooper et al., 1999), which in turn is positively associated with perceiving online sexual activity as problematic (Cooper et al., 1999; Parsons et al., 2007). Online sexual activities include pornographic audio, video, and text stories, real-time chatting with fantasy partners, using dating websites (Carnes et al., 2007), searching for a sex partner (Daneback et al., 2006; Parsons et al., 2007), replying to sex advertisements, purchasing sex products, and contacting with prostitutes (Daneback et al., 2006).

The Internet plays a unique role in the gay, lesbian, bisexual, transgender (GLBT) community. The Internet allows men who are secretive about their sexuality to explore it more comfortably (Chaney & Chang, 2005; Parsons et al., 2007). A study of 183 gay and bisexual men (mean age = 36 ± 8.33 ; 59% white) found that men who have sex with men are more likely to use the Internet for sexual pursuits than either females or heterosexual males. Yet, human immunodeficiency virus (HIV)–negative men are more likely to report Internet use as problematic compared to HIV-positive men, despite no differences between the two groups in reported hours of Internet sex, suggesting that the Internet may have differing roles in CSB, depending on the individual’s HIV status (Parsons et al., 2007).

Gender Differences

Researchers have found that more males than females report symptoms consistent with CSB (78%–92% of study samples are males with CSB; Black et al., 1997; Carnes, 1991; Raymond et al., 2003) and have estimated that there is a 4:3 male:female ratio (Carnes, 1991). These estimates, however, are based on samples recruited through advertisements and therefore may not accurately reflect the gender composition for CSB. In a study of adolescent psychiatric inpatients ($n = 102$), of the 5 subjects with CSB, all were female (Grant et al., 2007). Yet, in a sample of 791 college students, 6.7% of males and 2.2% of females screened positive for CSB ($p = .002$; Odlaug & Grant, 2010). These conflicting results demonstrate the necessity for further research measuring the prevalence of CSB in both genders because the gender ratios for CSB are relatively unknown.

Several factors may influence the gender disparity in CSB. First, the majority of individuals seeking

treatment for CSB are males (Black, 2000), and compared to men, women experience more CSB-related shame (Schneider, 2000). Second, research has found that compared to females, males have more sexual fantasies, masturbate more frequently, become aroused more easily, and have more causal attitudes toward sex (Kafka, 2010). Males also typically engage in sexual relations for pleasure and esteem reasons, whereas females participate in sexual relations to further their relationships (Impett & Paplau, 2003) and to develop long-term commitments for child-rearing purposes (Kafka, 2010). Another factor that may contribute to the higher proportion of CSB in males is the cultural double standard by which men who are highly sexually active are labeled as being "men," while females who behave in a similar fashion are viewed as promiscuous (Feree, 2002).

Given that males and females pursue sexual relations for differing reasons, it is possible that CSB presents differently in females. For many females, being in a relationship may be more enticing than the sexual activity (Feree, 2002). Women may therefore be more likely to engage in multiple love relationships rather than compulsive cruising or sex with multiple partners (Coleman et al., 2003).

Adolescent CSB

Little research has explored the area of adolescent CSB. Grant et al. (2007) observed that 4.9% of adolescents in a psychiatric inpatient unit had a comorbid diagnosis of CSB. To date, this is the only known empirical study assessing rates of CSB within an adolescent population.

Other research has examined factors associated with sexual behaviors among adolescents. Results from the 2007 National Youth Risk Behavior Survey (a three-stage cluster sample, administered to 9th-through 12th-grade students in private and public U.S. schools) indicated that 47.8% of students had had sexual intercourse, 7.1% of students had had sexual intercourse before the age of 13, 14.9% of students had had sexual intercourse with four or more individuals in their life, 35.0% of students had had sexual intercourse with at least one person during the 3 months prior to the survey, and of the 35.0% of sexually active students, 22.5% drank or used drugs before their last sexual intercourse (Eaton et al., 2008). Another survey of public high school students in grades 9 through 12 ($n = 3805$; 48% male; 60% white) found that alcohol was significantly related to the number of sexual partners among all participants and that among males, the

number of partners was also significantly associated with the carrying of a weapon (Valois et al., 1999).

Ethnic and Cultural Aspects of CSB

Given the lack of research on CSB in general, there is virtually no research devoted to ethnic and cultural aspects of CSB. Needel and Markowitz (2004) completed a chart review investigating CSB within a Hasidic Jewish inpatient population ($n = 52$; 62% male). When comparing Hasidic Jewish with non-Hasidic Jewish subjects, they found that 69% and 40% of the Hasidic males and females, respectively, reported hypersexual behavior (defined as public masturbation, exhibitionism, attempting to touch others' private regions, questioning others about their sex life, or offering unsolicited information about their own sex life) compared to 19% and 20% of the non-Hasidic men and females, respectively. The Hasidic community has very rigid views of sexuality, including no masturbation, no interaction with members of the opposite sex outside of the family, and the belief that homosexuality is sinful (Needel & Markowitz, 2004), suggesting that cultural views influence the definition of CSB. Overall, further research should evaluate ethnic and cultural influences on CSB.

How CSB Differs from Paraphilic Disorders

A primary feature of CSB is the excessive, uncontrollable, culturally normative sexual behavior, urges, and/or thoughts that result in distress and functional impairment (Black, 2000; Black et al., 1997; Coleman et al., 2003; Raymond et al., 2003). In contrast to CSB, paraphilic disorders involve an uncontrolled preoccupation with deviant forms of sexual arousal and behaviors (such as achieving sexual arousal from or using behaviors that involve nonhuman objects; or the suffering or humiliation of oneself or one's partner, or that of children or other nonconsenting persons; APA, 2000).

There are several differences between CSB and paraphilic disorders. In a sample of 120 outpatient males (mean age = 37.1 ± 9.5) with paraphilia-related disorders ($n = 32$) or paraphilic disorders ($n = 88$), Kafka and Hennen (2002) found that those with paraphilic disorders reported significantly higher rates of physical abuse (17.0% vs. 3.1%; $p \leq .05$), school problems (40.9% vs. 9.3%; $p \leq .005$), arrests (61.3% vs. 18.7%; $p \leq .005$), unemployment/disability (18% vs. 3.3%; $p \leq .05$), psychiatric hospitalizations (25.0% vs. 3.1%; $p \leq .01$), lifetime cocaine use (18.1% vs. 3.1%; $p \leq .05$), any lifetime attention deficit hyperactivity disorder (42.0% vs. 18.7%; $p \leq .01$), and

any lifetime conduct disorder (22.7% vs. 0.0%; $p \leq .005$) compared to those with paraphilia-related disorders.

CSB and the GLBT Community

Relatively little research has been done on CSB within the GLBT community, and the research that has been done mainly focuses on males who have sex with other males. Some researchers have suggested that sexual compulsivity may be more frequent in the gay and bisexual male community due to higher numbers of reports of more total sexual partners than heterosexual males (Qualand, 1985), as well as the availability of gay-oriented sexual outlets, such as sex parties, bathhouses, and sex websites that may facilitate the development of CSB (Parsons, 2005).

Research has suggested that sexual compulsivity is positively associated with sexually risky behaviors in both homosexuals and heterosexuals (Kalichman & Rompa, 1995). Kalichman and Rompa (1995), in a sample of 296 gay men (mean age = 36.4 ± 10.9) and 158 inner-city, low-income heterosexual men and women (35.1 ± 9.1), found that sexual compulsivity was significantly associated with unprotected intercourse ($p = .05$ in gay men; $p = .01$ in heterosexual individuals), total number of sexual partners ($p = .01$ in gay men and in heterosexual individuals), and sexual sensation seeking ($p = .01$ in both gay men and heterosexual individuals). However, compared to heterosexual males and females, men who have sex with other men participate in significantly more sexually risky behaviors (defined as inconsistent condom use and multiple sex partners in the past 12 months; 11.6%, 5.2%, and 18.6%, respectively) in a sample collected from the Los Angeles County Health Surveys (population-based telephone household surveys) during 1997, 1999, and 2003 (Brooks et al., 2008).

Another study of 180 inner-city men self-identified as gay or bisexual with HIV found that compared to those with low sexual compulsivity, men with high sexual compulsivity (measured by the Sexual Compulsivity Scale) were more likely to be both the insertive partner and the receptive partner in unprotected intercourse (35.4% vs. 77.8%, respectively; 11.4% vs. 28.6%, respectively) and less likely to report that they disclosed their HIV status during unprotected intercourse in which the participant was the insertive partner (55.3% vs. 28.6%, respectively; Reece, 2003).

Studies measuring sexual compulsivity have found that 20%–28% of GLBT subjects score highly on sexual compulsivity measures (Kelly et al.,

2009; Reece, 2003) and 11%–23% of individuals with CSB engage in same-sex relations (Black et al., 1997; Kafka & Prentky, 1992). Yet, there are conflicting reports on the percentage of individuals with CSB who self-identify as homosexual. Carnes (1991) found that 29% of 932 sexual addicts self-identified as either homosexual or bisexual, while Reece and Dodge (2004) found that 87.7% of 30 men who cruised for sex on a college campus self-identified as either gay or bisexual.

Kelly and colleagues (2009) assessed 1543 individuals (78.7% men; male mean age = 37.46; female mean age = 34.03) in the GLB community in New York, finding that 28% of the sample had CSB (as measured by a score of 24 of the Kalichman Sexual Compulsivity Scale). They also found that men who have sex with men (MSM) had significantly higher sexual compulsivity scores than women who have sex with women. Further analysis indicated that individuals with CSB report more use of specialized sexual behaviors (SSB, such as bondage and discipline, sadomasochism, exhibitionism, and sex with urine) and that women with CSB were especially more likely than women without CSB to engage in SSB (45.7% vs. 21.6%, respectively).

When asked about the origins of their sexual compulsivity, gay and bisexual men cite both intrinsic (i.e., poor mental health, low self-esteem, need for validation and affection, stress reduction, and biological predisposition) and extrinsic (i.e., relationships issues, availability of sex, childhood sexual abuse, and maladjusted parental relationships) reasons (Parsons et al., 2008).

Other studies of CSB within the GLBT community have linked higher levels of internalized homophobia (IH) with greater sexual compulsivity (Dew & Chaney, 2005; Smolenski et al., 2009). A survey of self-identified Latino men who have sex with men ($n = 963$; age = 28.2 ± 7.8) found a moderate correlation between IH and sexual compulsivity. Interestingly, results also indicated that there is a strong association between sexual compulsivity and high-risk sexual behavior in individuals high in IH and who were members of a gay organization, but no association between sexual compulsivity and high-risk sexual behavior in those who had high IH and were *not* members of a gay organization, suggesting that social networks may play a role in CSB in male homosexuals (Smolenski et al., 2009).

Family History

Research suggests that the majority of individuals with CSB come from dysfunctional families (86.8%

and 77% were from disengaged and rigid families, respectively; Carnes, 1991). Carnes (1989) proposes that as a child, the sexual addict's emotional needs were not met, either because of parental rigidity or lack of follow-through, resulting in the child believing that people are unreliable and that they can, therefore, only depend on themselves. These beliefs create a sense of distrust and the need to be in control, which are common among all addictions. Thus for sexual addicts, sex becomes an important source well-being because sex become a reliable and available way of feeling cared about. Carnes (1989) also notes that many sexual addicts report a family environment in which sexuality was labeled as bad and/or sexuality was out of control.

A history of substance abuse and mental illness is also common in the relatives of individuals with CSB. In a survey of 76 recovering sexual addicts (84% male) and 74 coaddicts (89% female), most participants (81%) had experienced at least one addiction in their family. Forty percent reported at least one chemically dependent parent, 36% reported at least one sexually addicted parent, 30% reported at least one parent who had an eating disorder, and 7% reported at least one parent with a compulsive gambling problem (Schneider & Schneider, 1996). Carnes (1998) reports that only 13% of sexual addicts come from a family without any addictions.

Medical Comorbidity

Individuals with CSB may face a variety of medical complications including, but not limited to, unwanted pregnancies, sexually transmitted infections, HIV/AIDS, and physical injuries due to repetitive sexual activities (e.g., anal and vaginal trauma, burns from overuse of a vibrator; Carnes, 1991; Coleman, 1992; Coleman et al., 2003).

A major health risk for those with CSB is sexually transmitted infections, such as HIV/AIDS. Studies have found that high levels of sexual compulsion are related to more unprotected sexual acts, a greater number of sexual partners (Benotsch et al., 1999; Kalichman et al., 1997; Kalichman & Rompa, 1995), and being diagnosed with multiple sexually transmitted infections (Kalichman & Cain, 2004). A study of 294 HIV-positive individuals (mean age = 40.5 ± 7.4 ; 69% male; 77% African American) found that compared to those who were not sexually compulsive, those with high levels of sexual compulsion (16% of the sample), were significantly more likely to report engaging in unprotected vaginal or anal intercourse,

having more sexual partners (both of HIV-negative and unknown HIV status), and engaging in sexual behaviors that could lead to HIV transmission (such as unprotected anal intercourse). In addition, four times as many new HIV infections could be expected in the HIV-negative partners of sexually compulsive participants, which was significantly more than the rate in the nonsexually compulsive group (Benotsch et al., 2001).

The World Bank (2000; as cited in Mmidi & Delmonico, 2001) estimated that worldwide, of all individuals with HIV infection, 63% live in sub-Saharan Africa, suggesting the importance of investigating rates of CSB within this population. Researchers found that in a sample of non-treatment-seeking males, Batswanan males ($n = 52$; mean age = 35.1) had a significantly higher mean score on the Sexual Addiction Screening Test than American males ($n = 42$; mean age = 36.4; 9.08 ± 5.75 vs. 3.93 ± 4.16 , $p < .001$, respectively; Mmidi & Delmonico, 2001). This finding suggests that more research is needed on the prevalence of CSB within countries with high rates of HIV infection.

Psychiatric Comorbidity

Psychiatric comorbidity appears common in CSB. Raymond and colleagues (2003; $n = 25$) and Black et al. (1997; $n = 36$) found that 100% and 84%, respectively, of their participants met criteria for a lifetime diagnosis of another Axis I DSM-III disorder and that 88% and 63%, respectively, met the criteria for a current Axis I DSM-III disorder. Raymond et al. (2003) found that the majority of individuals with CSB had a lifetime diagnosis of an anxiety (96%, especially social phobia [21%]), substance abuse (71%, especially alcohol [63%], and cannabis [38%]), or mood (71%, especially major depression [58%]) disorder. There was also a high prevalence of sexual dysfunction (46%) and impulse control disorders (38%; Raymond et al., 2003). Black and colleagues (1997) reported similar findings, with elevated lifetime rates of substance use (64%, especially alcohol abuse or dependence [58%]), mood (39%, specifically major depression or dysthymia [39%]) disorders, and anxiety (50%, especially phobic disorder [42%]) disorders.

Personality disorders are also common among individuals with CSB. One study found that 46% of individuals with CSB had an Axis II personality disorder (Raymond et al., 2003). The most common Axis II disorders included paranoid (20%), passive-aggressive (20%), and narcissistic (18%) personality disorders (Raymond et al., 2003).

Similarly, Black and colleagues (1997) found that 44% of their subjects reported a personality disorder; most commonly histrionic (21%), paranoid (15%), obsessive-compulsive (15%), and passive-aggressive (12%) personality disorder.

Substance Use and CSB

Many studies have documented the connection between substance use and sexual behaviors (Black et al., 1997; Kafka & Hennen, 2002; Maranda et al., 2004; Raymond et al., 2003; Valois et al., 1999; Wines, 1997), but few have specifically examined the connection between CSB and substance use. Substances can alter the experience of sexual behaviors. Methamphetamine increases sexual desire and sensations while decreasing sexual inhibition (Degenhardt & Topp, 2003; Semple et al., 2002), while cocaine leads to feelings of well-being, self-confidence, and alertness (Frishman et al., 2003). Research has shown that 34%–71% of individuals with CSB have co-occurring lifetime substance abuse disorders (Black et al., 1997; Kafka & Hennen, 2002; Raymond et al., 2003; Wines, 1997). In a national treatment study of 4939 individuals, researchers found a significant positive relationship between crack cocaine use and the number of sexual partners (Maranda et al., 2004).

The substance use–sex connection may be stronger within the homosexual population, because substance users typically feel more confident and desirable, and have an easier time cruising for sex and making contact with another person, resulting in greater success in finding a partner (Guss, 2000). Harawa and colleagues (2008) examined drug use in 46 African American males (mean age 41.5 years) who have sex with other males and found that drug use facilitated feelings of hypersexuality or sexual compulsion that were most easily satisfied by male partners, increased their comfort with approaching other men, and allowed them to cope with feelings of homo/biphobia. Research has also found that individuals who have sex with an individual of the same gender who have CSB are more likely to use substances prior to sex, and more specifically, that men who have sex with other men with CSB are more likely than men without CSB to use alcohol with sex (46% vs. 38.9%, respectively) and drugs with sex (39.4% vs. 31.5%, respectively; Kelly et al., 2009).

Etiology

There is a limited amount of information on the etiology of CSB; however, many studies have linked

the development of CSB to childhood abuse (Anderson & Coleman, 1990; Carnes, 1989, 1991; Perera et al., 2009; Tedesco & Bola, 1997). In a survey of 290 individuals in advanced sexual addiction recovery, significant rates of emotional (97%), sexual (81%), and physical (72%) abuse were found (Carnes, 1991). Other researchers have observed that 28%–80% of sexual addicts have experienced childhood sexual abuse (Black et al., 1997; Carnes & Delmonico, 1996; Kafka & Prentky, 1992) and about 22% have experienced physical abuse (Black et al., 1997; Kafka & Prentky, 1992). In a study comparing childhood abuse rates in sexual addicts ($n = 176$) versus controls ($n = 184$), Carnes (1989) found that 63% of female and 39% of male sex addicts compared to only 20% and 8% of female and male controls endorsed a history of childhood abuse.

Besides abuse, there are numerous other environmental and biological factors that may contribute to the development of a sexual addiction. Poor family environments (Perera et al., 2009); restrictive environments regarding sexuality; dysfunctional attitudes about sex and intimacy; anxiety; and depression (Anderson & Coleman, 1990) have been mentioned as contributing factors. In addition, CSB has been linked to a variety of neuropsychiatric conditions, including head traumas, brain surgeries, neuropsychiatric illnesses, both prescription and nonprescription medications, frontal lobe lesions, frontal lobe dysfunction, frontal and temporal lesions, temporal lobe epilepsy, temporal lobe abnormalities, dementia, Kluver-Bucy syndrome, multiple lesions in multiple sclerosis, and treatment of Parkinson's disease with dopaminergic agents (Coleman et al., 2003).

In children and adolescents, hypersexuality has been linked to numerous conditions, such as sexual abuse, prescription and illicit drug use (risperidone, cocaine, and methamphetamine), hypothalamic hamartoma, Kleine-Levin syndrome, Kluver-Bucy syndrome, mania (due to bipolar affect disorder and carbamazepine), shaken-baby syndrome, and virilization disorders (El-Gabalawi & Johnson, 2007).

There has been limited neurobiological research on CSB. A recent small neuroimaging study in eight subjects with CSB found that although CSB patients showed significantly lower mean diffusivity in the superior frontal region, the difference between groups on fractional anisotropy in the superior frontal lobe was not significant (Miner et al., 2009). In addition, there were no significant differences between the CSB group and the control group on any measures

in the inferior frontal region, which is inconsistent with the findings from diffusion tensor studies in other impulse control disorders (Cohen, 1988).

Parkinson's Disease and CSB

Medications used to treat Parkinson's disease (PD; e.g., levodopa, dopamine agonists) have been associated with hypersexuality. Studies have found that 1%–50% of individuals with PD report increased sexual interest while being treated with levodopa (Uitti et al., 1989) and that hypersexual behavior typically occurs in males with an earlier onset of PD (Klos et al., 2005). Uitti and colleagues (1989) found that the mean age of PD onset for those with hypersexuality was 49.5 years, while for those without hypersexual behavior, age of PD onset was somewhat greater (61.8 years).

Observational studies have supported the presence of hypersexual behavior in individuals with PD. A study of 100 individuals with PD (66% male) found that 4% (average age of PD onset = 43 years; 100% male) experienced hypersexual behavior and had taken a dopamine agonist (Pontone et al., 2006). Another study evaluated 13 PD patients (mean age at PD onset = 49.5 years; 85% male) who reported hypersexuality while taking antiparkinsonian medications (such as levadopa and other dopamine agonists) and found that 46% had a significant increases in their sexual interest/behavior compared to baseline sexual behavior; 38% were preoccupied with sexual intercourse; and 23% experienced excessive sexual desire and performance (Uitti et al., 1989). A chart review identifying 13 individuals with PD and 2 with multiple system atrophy (MSA; $n = 15$; median age of PD onset = 51 years; 100% male; 60% had another compulsive or addictive behavior) found that in 14 of the 15 individuals, hypersexual behaviors (such as increased sexual interest and urges, masturbation, and pornography use) began 8 months after starting levodopa or a dopamine agonist. Hypersexual behavior resolved in four cases when the agonist was stopped, in two cases spontaneously, and in four cases when another medication was added; there was improvement in one case when the agonist was tapered off (Klos et al., 2005). These reports suggest that although no causal relationship can be established with respect to these agents, dopamine may play a role in the pathophysiology of CSB.

Theoretical Categorization

Currently, there is an ongoing discussion about the proper categorization of CSB. Some believe that

CSB should be treated as an addiction, while others believe that it most properly fits within the obsessive-compulsive spectrum (Hollander & Wong, 1995; Wines, 1997).

Addiction Model

In this perspective, CSB has many similarities to addictions, which are characterized by (1) repetitive or compulsive engagement in the problematic behavior despite adverse consequences; (2) diminished control over the behavior; (3) an appetitive urge or craving state prior to engaging in the behavior; and (4) a hedonic quality during the performance of the behavior (Grant & Potenza, 2005). In a study assessing the applicability of substance abuse criteria to sexual addiction, Wines (1997) ($n = 53$; 88.7% male; mean age = 41.55 ± 9.06) found that a large majority of self-identified sexual addicts attending a support group reported three or more withdrawal symptoms (98%), unsuccessfully attempting to control their sexual behaviors (94%), spending significant time preparing for or recovering from addictive sexual behaviors (94%), and engaging in longer or greater amounts of sexual behavior than intended (92%). Additionally, most individuals with CSB found pleasure while engaging in sexual activities and reported urges or cravings to engage in sexual activities (Black et al., 1997). These symptoms mirror those of substance addiction.

An important reason to investigate this perspective is the high number of individuals who have CSB co-occurring with substance or behavioral addiction. Wines (1997) found that over half (56.6%) of 53 self-identified sex addict had a co-occurring addiction (most commonly, to alcohol or drugs, 34.0%). Additionally, Carnes (1998) found that 83% of sexual addicts have other addictive behaviors, most commonly chemical dependency (42%), eating disorders (38%), compulsive working (28%), compulsive spending (26%), and compulsive gambling (5%).

Obsessive-Compulsive Spectrum Model

Similar to obsessive-compulsive disorder (OCD), CSB is characterized by repetitive, ritualistic, and exaggerated ordinary behaviors (Aboujaoude & Koran, 2008), such as excessive masturbation. In addition, many individuals report that their behavior is anxiety driven and that they participate in sexual behavior to temporarily reduce anxiety, which consequently perpetuates the cycle of acting on sexual urges and thoughts to decrease anxiety

(Coleman, 1992). As previously mentioned, high rates of comorbid anxiety disorders (Black et al., 1997; Raymond et al., 2003) and reports of anxiety relief resulting from engagement in the behavior have been noted in CSB samples (Black et al., 1997).

In contrast to OCD, however, people typically experience pleasure while engaging in compulsive sexual behaviors, whereas individuals with OCD are primarily motivated by anxiety reduction rather than pleasure seeking (Aboujaoude & Koran, 2008). A study of 12 individuals ($n = 6$ OCD; $n = 6$ CSB) attending an anxiety disorders clinic who primarily complained of "sexual obsessions" suggested that sexual obsessions in OCD are experienced with an element of fear and avoidance, while those with CSB experience pleasure. In line with this finding, all CSB subjects acted on their sexual urges to obtain gratification, whereas none of the OCD participants acted on their sexual obsessions. Furthermore, the sexual obsessions for those with OCD were mainly a concern for themselves compared to the CSB group, whose sexual obsessions were mainly problematic for others (Schwartz & Abramowitz, 2003).

Conclusions and Future Directions

Compulsive sexual behavior is a relatively common disorder that generally occurs in males. A main feature of CSB is having sexually related thoughts, urges, and behaviors causing significant psychosocial distress and functional impairment. Comorbid psychiatric issues are the rule, not the exception, and many patients report a history of physical or sexual abuse. Currently, the etiology of CSB is unknown, but it appears to be a combination of psychological, environmental, and neurobiological factors. A variety of pharmacological and psychosocial treatments have shown promise in the treatment of CSB (see the chapter entitled "Impulsive/Compulsive Sexual Behavior: Assessment and Treatment"). Currently, there exists very little awareness in the community regarding the impact of CSB on the individual and on families. Disseminating information throughout the community to schools, to workplaces, and to health care providers may advance the understanding of this often disabling disorder.

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Abstract

This chapter discusses the phenomenology and epidemiology of problematic Internet use (PIU). Interest in the addictive potential of Internet activities has grown in the past two decades. Problematic Internet use can be broadly defined as uncontrolled use of the Internet that leads to significant psychosocial and functional impairments. It is currently conceptualized as an impulse control disorder that may share features with substance dependence disorders. Reliable studies have estimated the prevalence in the general population to be about 1%, but higher proportions of individuals might be at risk. Comorbidities with other psychiatric disorders are common. Concerns exist regarding the appropriateness and implications of formally recognizing PIU as a distinct disorder in current diagnostic systems. Given the ever-growing exposure to the Internet, especially in younger generations, PIU might become an emerging public health problem. Further studies are greatly needed, especially those using valid measures and longitudinal designs. Neuroimaging and genetic studies should also be explored.

Keywords: addiction, adolescence, behavior, comorbidity, computer, depression, impulsivity, Internet

Introduction

History and Background

The origins of the Internet date back to the 1960s. The original networks, such as the U.S. Department of Defense's ARPANET, were designed to provide resource-sharing links between major computational resources and computer users in academic, industrial, and government research laboratories (National Science Foundation, 2009). From the commercialization of the Internet in the early 1990s to the present, the Internet has found applications in virtually every aspect of modern human life and has become an indispensable tool to many. It is estimated that, as of June 2009, a quarter of the world's population are users of the Internet (Miniwatts Marketing Group, 2009).

There is a long history of interest in the potential for new information technologies to become addictive, dating to the invention of the radio in the 1930s (Cantril & Allport, 1935). From watching television

(Meerloo, 1954) to talking on a mobile phone (Walsh et al., 2008), problematic forms of human-technology interactions have been described. These actions have been considered behavioral addictions, which also include pathological gambling, compulsive sexual behaviors, compulsive buying, overeating, and others (Holden, 2001). The term *addiction* is not defined in the *Diagnostic and Statistical Manual of Mental Disorders* (APA, 2000), and there are ongoing debates regarding whether nonsubstance-related behaviors should be considered addictions (Martin & Petry, 2005; Potenza, 2006). In addition to their phenomenological similarities to substance dependence, emerging data from family and genetic studies also support the validity of the construct of behavioral addictions (Lobo & Kennedy, 2006).

The concept of *Internet addiction* was generated in part by New York psychiatrist Ivan Goldberg (Mitchell, 2000). In 1995, a year after the introduction of the DSM-IV (APA, 1994), Goldberg combined

the criteria for substance use and impulse control disorders and presented them as the criteria for *Internet addiction disorder* on his website. His presentation was designed as an exercise to stimulate the mental health profession to critically evaluate the usefulness of creating new disorders. However, the media subsequently began to publicize the notion of Internet addiction. Thus, the concept was socially constructed before substantial scientific or clinical research had been performed. Academic investigation expanded after the publication of clinical case studies by Young (1996) and Griffiths (1997).

Definition

Problematic Internet use can be defined as uncontrolled use of the Internet that leads to significant psychosocial and functional impairments. This pattern of use is not better accounted for by a primary psychiatric disorder such as mania or the physiological effects of a substance (Liu & Potenza, 2007). Alternative terms used include *Internet addiction* (Young, 1996), *compulsive Internet use* (Greenfield, 1999), *pathological Internet use* (Morahan-Martin & Schumacher, 2000), and *compulsive computer use* (Potenza & Hollander, 2002). Given that the term *addiction* is not well defined, we choose to use *problematic Internet use* (PIU) to remain descriptive and focused on Internet-based behaviors as opposed to other similar conditions such as (offline) video games addiction.

Within the current diagnostic system of DSM-IV-TR, patients with PIU may be given the diagnosis of impulse control disorder not otherwise specified (ICD-NOS), which is defined as disorders of impulse control that are not classified as part of the presentation of other major psychiatric disorders (APA, 2000). DSM-IV-TR contains specific diagnostic criteria for such impulse control disorders as pathological gambling, kleptomania, and pyromania, but not PIU (APA, 2000). The essential feature of impulse control disorders is the failure to resist an impulse, drive, or temptation to perform an act that is harmful to the person or to others. The individual feels an increasing sense of tension or arousal before committing the act and then experiences pleasure, gratification, or relief at the time of committing it (APA, 2000). In a case series of 20 patients with PIU diagnosed by face-to-face interviews, all fulfilled the DSM-IV criteria of ICD-NOS (Shapira et al., 2000).

Current Conceptualization

The current conceptualization of PIU as a behavioral addiction similar to pathological gambling,

presumably sharing the core feature of impulse dyscontrol, is originally based on phenomenological similarities between the two conditions (Liu & Potenza, 2007). This hypothesis has gained support from psychometric studies showing a relationship between higher impulsivity and PIU (Cao et al., 2007; Mottram & Fleming, 2009) and from epidemiological studies showing an association between attention deficit hyperactivity disorder (ADHD) and PIU in both children and adults (Yen et al., 2009b; Yoo et al., 2004). In a case control study comparing 50 Chinese adolescents with PIU and 50 matched controls, the group with PIU scored significantly higher on the Barratt Impulsiveness Scale and fared significant worse on the GoStop Impulsivity Paradigm (Cao et al., 2007). The GoStop Impulsivity Paradigm is essentially a behavioral task designed to assess an individual's ability to inhibit an already initiated response (Dougherty et al., 2005). It is noteworthy that subjects with PIU who had comorbid psychiatric disorders, the most common being ADHD (12.5%), were excluded from this analysis (Cao et al., 2007). The severity of PIU was found to correlate with the severity of ADHD symptoms in both inattention and hyperactivity-impulsivity domains in elementary school students in Korea after other important confounding factors including depression were controlled (Yoo et al., 2004). Preliminary neuroimaging data are also consistent with the hypothesized relationship between impulsivity and PIU, with a magnetic resonance imaging study showing that adolescents with PIU had gray matter density reductions in brain regions previously implicated in substance use disorders, including the left anterior cingulate cortex, left posterior cingulate cortex, and left insula (Zhou et al., 2009).

Diagnostic Criteria

Diagnostic criteria for PIU have been proposed, and one of the most commonly used sets of criteria in research studies is Young's Internet Addiction Diagnostic Criteria (Table 14.1; Young, 1996), which were modeled on the DSM-IV criteria for pathological gambling. Three of those DSM-IV criteria (chasing losses, committing illegal acts to finance gambling, and relying on others for money to relieve a financial situation caused by gambling) were deemed inapplicable in the case of PIU and were not included (Young, 1996). DSM-IV diagnostic criteria for substance dependence, impulse control disorders, and obsessive-compulsive disorder have been considered and extrapolated to proposed

Table 14.1 Young's Internet Addiction Diagnostic Criteria

Five or more of the following:

1. Is preoccupied with the Internet (thinks about previous online activity or anticipates next online session).
2. Needs to use the Internet with increased amounts of time in order to achieve satisfaction.
3. Has made unsuccessful efforts to control, cut back, or stop Internet use.
4. Is restless, moody, depressed, or irritable when attempting to cut down or stop Internet use.
5. Has stayed online longer than originally intended.
6. Has jeopardized or risked the loss of a significant relationship, job, educational or career opportunity because of the Internet.
7. Has lied to family members, therapists, or others to conceal the extent of involvement with the Internet.
8. Uses the Internet as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression).

Source: Young (1996). Reprinted with permission of Mary Ann Liebert, Inc.

diagnostic criteria for PIU (Aboujaoude et al., 2006; Shapira et al., 2003), but empirical studies to verify their validity and diagnostic accuracy have not been performed. Taiwanese researchers have empirically assessed candidate criteria taken from DSM-IV criteria for impulse control disorders and substance use disorders, as well as criteria suggested by other researchers and clinicians (Ko et al., 2005; Ko et al., 2009a). The diagnostic accuracy of each criterion was measured against the diagnosis made on the basis of a structured psychiatric interview and the subject's score on an Internet addiction scale. Criteria with high diagnostic accuracy and specificity, as tested in adolescents and college students, were kept to construct the proposed set of criteria (Table 14.2; Ko et al., 2009a). The latest version, shown in Table 14.2, includes the assertion that the diagnosis should not be given if the problematic pattern of use is accounted for by other impulse control disorders such as pathological gambling (Ko et al., 2009a).

Epidemiology

Prevalence

The prevalence of PIU in the general population has not been established primarily because of the lack of

Table 14.2 Proposed Diagnostic Criteria for Internet Addiction by Ko et al. (2009)

Distinguishing characteristics of Internet addiction:

A maladaptive pattern of Internet use, leading to clinically significant impairment or distress, occurring at any time within the same 3-month period.

A. Six (or more) of the following symptoms have been present

1. Preoccupation with Internet activities.
 2. Recurrent failure to resist the impulse to use the Internet.
 3. Tolerance: a marked increase in the duration of Internet use needed to achieve satisfaction.
 4. Withdrawal, as manifested by either of the following:
 - i. Symptoms of dysphoric mood, anxiety, irritability, and boredom after several days without Internet activity.
 - ii. Use of Internet to relieve or avoid withdrawal symptoms.
 5. Use of Internet for a period longer than intended.
 6. Persistent desire and/or unsuccessful attempts to cut down or reduce Internet use.
 7. Excessive time spent on Internet activities and leaving the Internet.
 8. Excessive effort spent on activities necessary to obtain access to the Internet.
 9. Continued heavy Internet use despite knowledge of having a persistent or recurrent physical or psychological problem likely to have been caused or exacerbated by Internet use.
- B. Functional impairment: one (or more) of the following symptoms have been present.
1. Recurrent Internet use resulting in a failure to fulfill major role obligations at work, school, and home.
 2. Important social or recreational activities are given up or reduced because of Internet use.
 3. Recurrent legal problems because of Internet behavior (e.g., arrest for disorderly conduct in a game).
- C. The Internet addictive behavior is not better accounted for by psychotic disorder, bipolar I disorder, or other disorders that are classified in impulse-control disorders and paraphilia in DSM-IV-TR.

Source: Ko et al. (2009a). Adapted with permission of Elsevier.

consensus on diagnostic criteria and validated assessment instruments. Table 14.3 summarized data from different prevalence studies of PIU. Given the relatively recent recognition of the condition, PIU has not been included in large-scale epidemiological studies such as the National Co-morbidity Survey (Liu & Potenza, 2007). Since the penetrance of the Internet is increasing and upcoming generations are exposed to it for longer periods and at younger ages, the prevalence of PIU can be expected to increase with time (Christakis & Moreno, 2009). To assess its prevalence accurately, random offline sampling of the general population is necessary. Online solicitation of self-identified excessive Internet users, as done in earlier studies (Greenfield, 1999; Whang et al., 2003), is likely to create selection biases and duplicate answering is difficult to avoid. To date there are two published studies on the prevalence of PIU as assessed by random sampling of the general population (Aboujaoude et al. 2006; Bakken et al., 2009). In the study done by Aboujaoude et al. in the United States, the point prevalence was found to be 0.7% when PIU was defined as follows: Positive cases necessitated that respondents report: (1) Internet use that interferes with relationships; (2) feeling preoccupied with Internet use when offline; (3) having either tried unsuccessfully to cut down or (4) staying online longer than intended (Aboujaoude et al., 2006). Higher percentages of respondents (3.7% to 13.7%) endorsed one or more of these features and other suggested markers of PIU. The survey used a random-digit-dial telephone method and interviewed 2513 adults (≥ 18 years of age) across all states. The fact that the study did not include people under the age of 18 and had a relatively small proportion of younger adults might have biased the prevalence estimate downward. The second study was conducted in Norway in 2007 via random sampling of the national population registry (Bakken et al., 2009). Using the Young Diagnostic Questionnaire (YDQ), researchers identified Internet addiction in 1.0% of the sample, with 5.2% scoring less than the diagnostic threshold but considered at risk (Bakken et al., 2009). These two studies are among the most methodologically rigorous, and both estimated the general prevalence of PIU to be about 1%. The diagnostic criteria used in both studies were different but had certain overlaps, and all four markers used in the study by Aboujaoude et al., as listed above, were conceptually included in the YDQ. The slightly lower prevalence observed by Aboujaoude et al. might be a result of the lower proportion of Internet users in their study (Aboujaoude et al., 2006).

Two studies to date have employed random sampling of adolescents (12 to 18 years of age) in the general population (Johansson & Gotestam, 2004; Kaltiala-Heino et al., 2004). Although they used slightly different diagnostic criteria, both studies were conducted in Europe and surveyed subjects in the same age range. Both studies found a prevalence of PIU of less than 2% (Johansson & Gotestam, 2004; Kaltiala-Heino et al., 2004). Also using the YDQ, Norwegian researchers Johansson and Gotestam surveyed a random sample of adolescents from the population registry in 1999 and found a prevalence of PIU of 1.98%, with an additional 8.68% considered at risk (Johansson & Gotestam, 2004). Problematic Internet use appears to be more prevalent in Norwegian adolescents than in adults, but a comparison of this study with that of Bakken et al. (2009) mentioned above has not been performed and it is unclear if the difference is statistically significant. Further, when Bakken et al. analyzed the adolescents in their sample (16 to 18 years of age), they found a prevalence (5.2% in boys and 3.9% in girls) higher than that in Johansson and Gotestam's study, showing that the prevalence among adolescents might have increased over the past few years, as would be expected with the increase in access to broadband and home computers.

Problematic Internet use has been reported in many countries across all continents (e.g., the United States, Korea, the People's Republic of China, Taiwan, Norway, Italy, Iran), indicating that the phenomenon is recognized worldwide. The above-mentioned studies showed that Western countries might have a similar prevalence of about 1% (Aboujaoude et al., 2006; Bakken et al., 2009). Prevalences among different regions have not been systematically compared. Preliminary comparison between Chinese and American college students showed that PIU might be more common and more severe in Chinese students than in their American counterparts (Zhang et al., 2008). Possible explanations include differences in the stage of industrialization, cultural backgrounds, and the history of Internet availability between the two countries.

Differences between Age Groups and Genders

Data from school-based studies show relatively large variations. Accurate comparison is difficult due to variations in diagnostic thresholds and survey methodologies. However, studies conducted among college and high school students consistently show much higher prevalences than those conducted

Table 14.3 Selected Prevalence Studies of Problematic Internet Use

Author and Year	Country	Sample	Assessment Instrument	Prevalence	Gender Difference
Bakken et al. (2009)	Norway	$N = 3399$ General population, age ≥ 16	Young Diagnostic Questionnaire (YDQ)	1.0%	M > F
Tsitsika et al. (2009)	Greece	$N = 897$ High school students	Young Internet Addiction Test (YIAT)	1.0%	M > F
Aboujaoude et al. (2006)	USA	$N = 2513$ General population, age ≥ 18	Four sets of diagnostic criteria with different thresholds devised by the authors	0.3% to 0.7%	Not addressed
Kim et al. (2006)	Korea	$N = 1573$ High school students	YIAT	1.6%	No gender differences observed.
Johansson & Gotestam (2004)	Norway	$N = 3237$ Adolescents in the general population	YDQ	1.98%	M > F
Kaltiala-Heino et al. (2004)	Finland	$N = 7229$ Adolescents in the general population	Internet addiction criteria analogous to DSM-IV criteria of pathological gambling developed by the authors	1.7% among boys and 1.4% among girls	No gender differences observed.
Yoo et al. (2004)	Korea	$N = 535$ Elementary school students	YIAT	0.9%	M > F
Morahan-Martin & Schumacher (2000)	USA	$N = 277$ College students	Pathological Internet Use Scale developed by the authors	8.1%	M > F

among younger adolescents and elementary school students. For example, using YDQ and another behavior inventory for diagnosis, Chou and Hsiao found a prevalence of 5.9% in a sample of 910 university students in Taiwan ages 20 to 25 years (Chou & Hsiao, 2000). Among 1708 senior high school students in Taiwan, 18.9% of boys and 7.3% of girls were identified as having Internet addiction as defined by the YDQ (Yang & Tung, 2007). In comparison, also using YDQ as the diagnostic instrument, the prevalence of PIU was found to be 2.4% in a group of younger school children in China ages 12 to 18 years (Cao & Su, 2007). In contrast, among elementary school students (mean age = 11), the prevalence was found to be 0.9% (Yoo et al., 2004). This might reflect the fact that younger children's Internet use is under adult supervision and monitoring, whereas high school and college students can often access the Internet without parental control.

Studies to date generally agree that PIU is more prevalent among males than females across age groups, and male gender is a strong predictor of PIU in regression analyses (Bakken et al., 2009; Mottram & Fleming, 2009; Yen et al., 2009a; Yoo et al., 2004), similar to the case in pathological gambling (Cunningham-Williams et al., 1998). For example, in a survey of 277 undergraduate Internet users, males were fourfold more likely (12% vs. 3%) to meet criteria for PIU than were females (Morahan-Martin & Schumacher, 2000). This may be related to the differences in impulsivity levels, coping strategies, and preferred Internet activities between males and females (Morahan-Martin & Schumacher, 2000; Mottram & Fleming, 2009; Ko et al., 2008). Males are more likely to use applications for games, cybersex, and gambling, activities that are associated with a strong emotional-motivational state that contributes to addictive behaviors (Morahan-Martin & Schumacher, 2000), whereas females tend to use the Internet for work or to seek friendships and social support online (Mottram & Fleming, 2009).

Phenomenology

Core Features

The above-mentioned proposed sets of criteria capture the core features of PIU, which include preoccupation, increased use over time, failure to cut back use, disturbed mood when use is stopped, using for longer periods than intended, failure to fulfill normal social roles, and using the Internet as a primary way of coping (Ko et al., 2009a; Young, 1996). These features describe a change in saliency hierarchy toward Internet activities. Patients often

report features similar to tolerance to and withdrawal from a chemical substance (Young, 1996). Symptoms that appear when Internet use is abruptly discontinued are primarily subjective mood disturbances including feeling depressed, irritable, and frustrated, but no studies have documented any associated physiological changes such as changes in pulse or blood pressure (Pies, 2009). Typical features of impulse control disorders have also been described, which include a rising sense of tension before logging on and relief of tension when Internet use starts (Atmaca, 2007).

In addition to impulsiveness, a level of compulsiveness is typically observed. In an Italian cohort of 15 subjects with PIU, subjects were found to have high Yale-Brown Obsessive Compulsive Scale (Y-BOCS) scores, which correlated with the severity of PIU (Bernardi & Pallanti, 2009). Interestingly, no subject fulfilled diagnostic criteria for obsessive-compulsive disorder, while ADHD was found to be a common comorbid diagnosis (Bernardi & Pallanti, 2009). In PIU, as in other impulse control disorders, impulsive features may initiate the behavior and the compulsive drive may cause it to persist (Dell'Osso et al., 2006).

Another core feature of PIU is time distortion. Patients often use the Internet for long periods of time, typically over 40 hours a week (Bernardi & Pallanti, 2009), to the exclusion of other work or social activities. Amount of time spent online was noted to be a fairly reliable reflection of the severity of PIU (Bernardi & Pallanti, 2009; Liu & Potenza, 2007). However, patients often underestimate their time used. When objectively recorded logged-on time was compared to self-reports, the discrepancy was found to be up to 40% (Shapira et al., 2003). Psychological dissociation, which has also been reported in other impulse control disorders, and detachment from reality, which is often sustained by Internet activities, may contribute to a distorted sense of time (Bernardi & Pallanti, 2009).

Physical and Psychosocial Impairments

Physical ailments as a result of exhaustion can be expected from Internet overuse and include dry eyes, blurred vision, sleep deprivation, fatigue, and musculoskeletal discomfort or pain such as carpal tunnel syndrome (Chou, 2001). In addition to excessive daytime sleepiness (EDS), which can be five times more prevalent among individuals with PIU than among controls, a wide range of sleep disturbances including insomnia, witnessed snoring, apnea, teeth grinding, and nightmares have also

been associated with PIU (Choi et al., 2009). Such sleep disruption may be related to excessive light exposure from the computer screen at night and contributes significantly to poorer perceived health among adolescents with PIU (Punamäki et al., 2007; Suganuma et al., 2007). In susceptible individuals, playing massive multiplayer online role-playing games for many hours may induce reflex seizures of various types, but a seizure warning is rarely displayed on game websites (Chuang, 2006). Multiple extreme examples of death after a marathon online gaming session have been reported in the news; the direct cause of death was likely a comorbid condition such as cardiac disease (BBC News, 2005; Spencer, 2007).

Severe psychosocial consequences may also result from PIU, including marital discord, estrangement from family, financial problems, and unemployment. The cause–effect relationship between excessive Internet use and psychological impairments including depression, loneliness, and social isolation is complex. Internet use may help extroverts to garner more social support but allow introverts to remain isolated and lonely (Kraut et al., 1998, 2002; Young & Rogers, 1998).

Comorbidities

It is uncommon for PIU to present as the chief complaint. Patients generally seek treatment for comorbid conditions such as depression and anxiety, with PIU emerging as a core issue after more detailed history taking (Bernardi & Pallanti, 2009). In fact, as many as 86% of PIU patients present with comorbid DSM-IV diagnoses (Ahn, 2007). Therefore, it has been suggested that PIU represents a symptom of underlying psychiatric disorders rather than a distinct psychopathological condition (Miller, 2007). However, treatment of other psychiatric disorders alone may not effectively address compulsive use of the Internet (Bostwick & Bucci, 2008), as is the case in substance use disorders and pathological gambling. Moreover, the Internet experience has unique properties not shared by other addictive objects such as intense and accelerated intimacy, disinhibition, loss of boundaries, and timelessness (Greenfield, 1999). Understanding the relationships between PIU and other psychiatric disorders will likely contribute to improved prevention and treatment.

Mood Disorders

In both case series and cross-sectional surveys, depression is one of the most common diagnoses

associated with PIU. In the United States, two case series systematically evaluated about 20 patients face-to-face and found frequent comorbid mood disorders (Black et al., 1999; Shapira et al., 2000). The frequencies of PIU and comorbid current major depression were 10% and 24%, respectively, and lifetime frequencies were 15% and 33%, respectively. In the series of Shapira et al., 70% of subjects had a lifetime diagnosis of bipolar affective disorder I or II. However, all had a current or recent depressive or mixed episode, and none had current or recent mania. These findings suggest that depressive symptoms, rather than manic symptoms, are more commonly associated with PIU. This relationship is supported by many cross-sectional surveys (Ha et al., 2007; Kim et al., 2006; Yen et al., 2007). The association between depression and adolescent PIU is robust and crosses genders and age groups (Yen et al., 2009a). Internet overuse can become a maladaptive way of coping for some depressed individuals. In addition, longitudinal studies showed that the reverse may also be true; that is, compulsive Internet use may increase the risk of depression in adolescents after as little as 6 months (van den Eijnden et al., 2008) and may lead to isolation and loneliness (Kraut et al., 1998), though its long-term effects are likely to be different on different personalities (Kraut et al., 2002). From a biological standpoint, a recent genetic study showed that a subgroup of problematic Internet users who have harm avoidance tendencies may share genetic traits with depressed patients, namely, higher expression of the short allelic variant of the serotonin transporter gene (*SS-5HTTLPR*) (Lee et al., 2008).

Attention Deficit Hyperactivity Disorder

In a sample of over 500 elementary school students, 22.5% of those identified as problematic Internet users had co-occurring ADHD (Yoo et al., 2004). Those with ADHD and PIU had significantly higher scores on parental, teacher, and self ratings of ADHD symptoms than those with ADHD alone (Yen et al., 2007; Yoo et al., 2004). In school-based studies, ADHD is commonly found comorbid with PIU in preadolescent boys, whose main use of the Internet is for playing games (Yoo et al., 2004), a pattern that is consistent with clinical observation. The features of deficient inhibitory control, lack of strategic flexibility, and poor boredom tolerance in ADHD may impair self-regulation of Internet use and increase the risk of PIU. In a prospective study, ADHD was shown to be an important predictive factor of PIU (Ko et al., 2009b).

Obsessive-Compulsive Disorder and Impulse Control Disorders

Obsessive-compulsive disorder (OCD) and impulse control disorders are hypothesized to lie along the same spectrum (Dell'Osso et al., 2006). Patients often report that their Internet use is impulsive and ego-syntonic rather than compulsive and ego-dystonic, as in OCD. As discussed, although a level of compusiveness is commonly seen that may perpetuate the behavior, comorbid full-blown OCD is not common (Bernardi & Pallanti, 2009). In two U.S. case series of PIU, frequencies of current OCD were 0% and 15%, and lifetime OCD frequencies were 10% and 20% (Black et al., 1999; Shapira et al., 2000). The percentages of individuals with any impulse control disorder were considerably higher (38% and 50%) in both series. A variety of impulse control disorders were identified in both series of patients, including intermittent explosive disorder, kleptomania, pathological gambling, pyromania, compulsive buying, compulsive sexual behaviour, and compulsive exercising.

Self-Injurious and Aggressive Behaviors

Hostility, aggression, and self-injurious behaviors have all been associated with PIU among adolescents (Ko et al., 2009b; Lam et al., 2009; Yen et al., 2007). These may represent behavioral manifestations of underlying impulse dyscontrol and/or mood disturbances. The relationship between aggression expressed in cyberspace and physical aggression, and the effects of violent Internet contents on behaviour, require further study.

Conclusion

Problematic Internet use is clinically recognizable and is associated with significant adverse physical and psychosocial consequences. This behavioral syndrome is inherently heterogeneous; people have different motivations to use the Internet to excess, and they have problems with different applications. For example, novelty-seeking individuals are drawn to the Internet's incessant novelty, while people with a harm-avoidance tendency find a sense of safety in the virtual world (Lee et al., 2008; Liu & Potenza, 2007; Mottram & Fleming, 2009). Nonetheless, both could develop problematic patterns of use. As is the case in other addiction syndromes, comorbidity is the rule rather than the exception, which needs to be taken into consideration in both research and clinical management of PIU (Liu & Potenza, 2007).

Recreational computer time doubled among 8- to 18-year-olds between 1999 and 2004 (Kaiser

Family Foundation, 2006). If all at-risk children achieve sufficient exposure to become problematic and compulsive users, the prevalence of PIU may quickly reach or surpass that of the most common chronic diseases of childhood (Christakis & Moreno, 2009). Given that exposure to the Internet is almost unavoidable, especially for younger generations, accurate data on its prevalence and phenomenology will have tremendous public health significance.

Clinicians should be aware of the clinical features of PIU and its potential consequences and be equipped to assess Internet use history in the clinical interview. Comprehensive clinical evaluation should reveal the patient's experience with the Internet and its relations to his or her emotional distress and psychiatric symptoms, as well as the patient's own understanding of the Internet's influences on his or her life. Co-occurring psychiatric disorders should be identified and appropriately addressed.

Future Directions

Compared to some Asian countries, little empirical research has focused on this potential problem in the United States, largely because of persistent skepticism about its existence (Christakis & Moreno, 2009). Further research is needed to establish diagnostic criteria of PIU that are validated and applicable to different age groups. Robust measures of problematic use are essential. These tools will help incorporate PIU in large-scale epidemiological studies, data from which are essential for the assessment of the public health impact of PIU.

To date, most studies on PIU have been cross-sectional in design. Some prospective studies have explored potential risk factors for PIU such as ADHD, family conflicts, and hostility (Ko et al., 2009b). Further research to identify risk factors will inform targeted preventive efforts in identifying children at greatest risk for problematic use. Longitudinal and prospective studies are essential in the evaluation of short- and long-term effects of PIU on physical and mental health. Currently, limited biological data on PIU are available. Preliminary neuron-imaging data on PIU suggest that further imaging studies may clarify whether PIU shares neurobiological bases with substance dependence and other impulse control disorders (Zhou et al., 2009). Genetic studies will also help clarify the relationship between PIU and related endophenotypes such as sensation seeking and harm avoidance (Lee et al., 2008). Although it is unlikely that PIU is a genetically determined disease per se, biological

understanding of its related traits and endophenotypes may reveal possible therapeutic avenues for both medical and psychological interventions.

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Phenomenology and Epidemiology of Pathological Skin Picking

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Abstract

Pathological skin picking (PSP), or neurotic excoriation, is characterized by the repetitive and compulsive picking of skin, causing tissue damage. It appears to have a prevalence rate of 1.4%–5.4% in the general population and is seen predominantly in females in clinical settings. Individuals with PSP may pick for hours each day, resulting in significant scarring, infections, and medical complications. Although PSP is common, most individuals with this disorder are unaware of treatment options and thus often do not seek treatment. Co-occurring psychiatric conditions are common in PSP, with depressive, anxiety, and obsessive-compulsive disorders presenting as the most prevalent conditions. Significant psychosocial impairment and activity avoidance due to shame and embarrassment are frequent. Neurocognitive research has recently shown that individuals with PSP have deficits in inhibitory control, a finding similar to that found in trichotillomania. From a public health perspective, concurrent collaboration between dermatology and the behavioral sciences is imperative for future advances in the understanding and treatment of PSP.

Keywords: clinical characteristics, comorbidity, dermatology, impulse control disorders, neurotic excoriation, obsessive-compulsive spectrum, pathological skin picking, skin

Introduction

Pathologic skin picking (PSP), also referred to as *skin picking disorder*, *neurotic excoriation*, *dermatillomania*, or *psychogenic excoriation*, is a disorder characterized by the repetitive and compulsive picking of skin, causing tissue damage (Arnold et al., 2001). Although it is not a formal disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., Text Revision; DSM-IV-TR; APA, 2000), the following diagnostic criteria have been proposed for PSP based upon other impulse control disorders: (1) recurrent and repetitive picking of the skin resulting in noticeable tissue damage; (2) a preoccupation with or experience of urges to pick skin that is reported as intrusive; (3) reported feelings of tension, anxiety, or agitation immediately preceding the picking episode; (4) feelings of pleasure, relief, or satisfaction during picking; (5) inability to

account for the picking by another medical (e.g., scabies, eczema) or mental disorder (e.g., cocaine or amphetamine use disorders, borderline personality disorder, parasitosis); and (6) significant distress or social or occupational impairment as a result of the picking behavior (Arnold et al., 2001). Much of the literature, however, uses a simpler definition that includes only criteria 1 and 6 (Keuthen et al., 2010).

History

Acne excoriation in young adolescent women who worsened their lesions by scratching and picking at them was first described in 1898 by the French dermatologist Brocq (1898). The early literature on patients with PSP describe them as “highly sensitive” (Seitz, 1951), “anger-repressing” (Michelson, 1945), “hostile” and “vindictive” (Zaidens, 1951)

individuals with an inherent inability to relate to others (Zaidens, 1951). Pickers were described as cunning individuals whom the treating physician, through experience, could learn to identify over time by observing their nervous mannerisms and facial tremors (Stokes & Garner, 1929).

The term *neurotic excoriation* was first introduced in the early 1900s by Wilson (Adamson, 1915) and was used to describe picking behaviors that were extremely difficult, if not impossible, to control (Adamson, 1915; MacKee, 1920; Pusey & Senear, 1920; Stokes & Garner, 1929). Similar to the characteristics of other grooming disorders such as trichotillomania and onychophagia (nail biting), the difficulty controlling the picking behavior helped to define the core characteristic of PSP (Bohne et al., 2005; Doran et al., 1985; Halprin, 1966; Odlaug & Grant, 2008a; Pusey & Senear, 1920). This core characteristic of an inability to control the picking aids in the proper identification of patients with PSP and differentiates the behavior from personality disorders or other psychiatric or dermatologic conditions (i.e., dermatitis artefacta or dermatitis facta) that often involve mutilation of the skin (Arnold et al., 2001; MacKee, 1920; Stokes & Garner, 1929).

Epidemiology

A recent telephone survey of 2511 adults ages 18 and older using random digit-dialing found that 16.6% picked their skin, resulting in noticeable tissue damage. This same survey found that when the additional requirements of distress or impairment were considered, 1.4% met criteria for PSP (Keuthen et al., 2010). Other studies have found that PSP may have rates as high at 5.4% in a community sample ($n = 354$; Hayes et al., 2009). In addition, PSP has been found in 4% of college students ($n = 105$; Keuthen et al., 2000), 2% of dermatology clinic patients ($n = 80$; Griesemer, 1978), and 11.8% of adolescent psychiatric inpatients ($n = 102$; Grant, Williams, & Potenza, 2007). Although PSP is a common disorder with a wide age range of affected individuals, its prevalence in the population is likely underreported, undiagnosed, and often untreated due to the fact that patients often report being unaware that efficacious psychological and pharmacological treatments are available (Grant et al., 2007a; Ko, 1999; Neziroglu et al., 2008).

Co-occurring PSP is also common in other psychiatric disorders and appears to be most prevalent in body dysmorphic disorder (BDD). In a sample of 176 patients with BDD, lifetime rates of PSP

were found in 44.9% of patients (Grant, Menard, & Phillips, 2006). Other studies have found PSP rates of 26.3% secondary to BDD (Phillips & Taub, 1995). This is much higher than the rates of co-occurring PSP found in obsessive-compulsive disorder (OCD) subjects (8.9%; Grant, Menard, & Phillips, 2006) and trichotillomania subjects (8.3%; Odlaug & Grant, 2007), although even these rates are significantly higher than those seen in the general population and may suggest neurobiological similarities between these conditions.

Clinical Characteristics

Research has suggested that the age of onset for PSP appears to be bimodal, occurring in the early 20s and then again between the ages of 30 and 45 (Arnold et al., 1998, 1999; Bohne et al., 2002; Calikuşu et al., 2003; Keuthen et al., 2000; Simeon et al., 1997). A recent study of 40 individuals with PSP, however, found that 19 (47.5%) had onset of symptoms before the age of 10 (Odlaug & Grant, 2007), a sample of 29 treatment-seeking individuals found a mean age of onset of 13.2 years (Keuthen et al., 2007), and another sample of 372 PSP subjects recruited via an online treatment program endorsed an age of onset of 14.4 years (Flessner et al., 2007).

Many individuals with PSP report that the behavior began with the onset of a dermatological condition such as acne (Wilhelm et al., 1999; Wrong, 1954), but the picking continued even after the dermatological condition cleared. For many with PSP, the preoccupation with blemishes is disproportionate to the dermatological issues faced by the patient (Zaidens, 1951); consequently, picking behaviors have been reported to worsen preexisting conditions (such as scarring from acne) for some patients due to the fact that they will not let wounds heal (Fruensgaard et al., 1978).

Individuals with PSP spend a significant amount of time each day picking their skin, with many reporting that the picking behavior lasts for several hours each day (Grant et al., 2007a) or, in some cases, is described as being nearly constant (Lochner et al., 2002). Due to the amount of time spent picking, individuals report missing or being late for work, school, or social activities (Flessner & Woods, 2006).

Although the face is the most commonly reported site of picking behavior, other areas such as the hands, fingers, torso, arms, legs, back, and stomach are also common targets (Arnold et al., 1998; Bohne et al., 2002; Calikuşu et al., 2003; Grant et al., 2007a;

Keuthen et al., 2000; Neziroglu et al., 2008; Odlaug & Grant, 2008a). A study of 60 patients with PSP reported that subjects picked from an average of 2.3 sites (Odlaug & Grant, 2008b). Many individuals report having a primary body area for picking but may pick at other areas of the body in order to allow the most significantly excoriated areas to heal (Bohne et al., 2002; Odlaug & Grant 2008b).

The affected areas are typically small in size (i.e., a few millimeters), are linear, and are either crusted over, scarred, or bleeding, presenting with postinflammatory hypo- or hyperpigmentation (Gupta et al., 1987; MacKee, 1920). For most patients with PSP, a variety of picking lesions are exhibited, ranging from a few to a few hundred (Gupta et al., 1987; Pusey & Senear, 1920). Although most individuals pick at areas they can reach with their fingernails, they also report using knives, scissors, tweezers, pins, needles, letter openers, and other objects to pick (Grant et al., 2007a; Neziroglu et al., 2008). Additionally, a small percentage (11.7%) of patients with PSP report picking the skin of their children or significant other (Odlaug & Grant, 2008b).

Triggers to pick vary greatly among patients and multiple triggers are the norm, not the exception. Stress, anxiety, downtime (e.g., sitting on the couch, watching television, reading), driving a vehicle, boredom, and feeling tired or angry have all been reported as triggering a picking episode (Arnold et al., 2001; Grant et al., 2007a; Neziroglu & Mancebo, 2001; Neziroglu et al., 2008; Simeon et al., 1997). Picking can also be triggered by the feel (e.g., a bump or unevenness) or look (e.g., a blemish or discoloration) of the skin.

Picking behavior often begins unconsciously and becomes conscious after a period of time (Grant et al., 2007a; Odlaug & Grant, 2008b). This is similar to the characteristics of other grooming behaviors such as trichotillomania (Lochner et al., 2002), in which the action of pulling has been described as automatic (i.e., unconscious) or focused (i.e., conscious; Christenson & Mansuetu, 1999). In samples of PSP patients, the percentage of time they reported being aware of their picking behavior has been found to range from 68.9% to 78.1% (Odlaug & Grant, 2007, 2008b). In many cases, however, the picking behavior becomes conscious only when someone else brings it to their attention or the site begins to bleed.

Due to the extent of damage caused by the picking, individuals often report using different forms of concealment to cover their picking exploits and paying hundreds or even thousands of dollars on

concealment products or medications to quell the negative feelings associated with skin picking (Flessner & Woods, 2006). One study of 92 subjects with PSP found that 78 (85%) reported using some form of concealment to hide their picking (Flessner & Woods, 2006). The most commonly reported concealment methods were makeup/hairstyles (44.6%) and clothing (e.g., wearing pants instead of shorts or long sleeves instead of short sleeves [40.2%]). Due to the shame and embarrassment over having to cover the excoriated areas, subjects often avoid certain activities such as social outings (40.2%), entertainment activities (20.7%), and vacations (10.9%; Flessner & Woods, 2006).

Although some patients may experience remission of symptoms without adequate treatment, most endorse the chronic nature of picking with only minimal periods of abstinence (Grant et al., 2007a; Keuthen et al., 2007; Krupp, 1977). For example, a study of 29 subjects being treated for PSP found a mean illness duration of 20.1 years (Keuthen et al., 2007), which is similar to the 18.9-year duration found in a sample of 24 subjects (Grant et al., 2007a). One recent study found that 50% of individuals with PSP reported an unsupportive family reaction to their picking behavior, which may have resulted in delay for evaluation and treatment (Neziroglu et al., 2008).

Gender Differences

It has been well established that PSP tends to occur much more frequently in females (52%–94.1%; (Arnold et al., 2001; Calikuşu et al., 2003; Fisher & Pearce, 1974; Flessner & Woods, 2006; Fruensgaard, 1984; Krupp, 1977; Mutasim & Adams, 2009; Wilhelm et al., 1999). These rates are similar to the 92.3% female preponderance seen in other grooming disorders such as trichotillomania (Woods et al., 2006). The question remains whether the higher rates in females reflect a genuine female preponderance or a hesitance in males to admit to their behavior or seek treatment (Christenson et al., 1994). Studies have shown that males presenting for treatment tend to be older than female treatment seekers (Fisher & Pearce, 1974; Krupp, 1977), with some males not presenting for treatment until their 80s (Krupp, 1977). Women often report that picking behavior either increases or decreases based upon their menstrual cycle (Wilhelm et al., 1999). Although little has been written about gender differences in PSP specifically, a study of 77 subjects with a grooming disorder (i.e., PSP or trichotillomania)

compared gender differences (Grant & Christenson, 2007). More similarities than differences were noted between males and females in this sample; however, males were more likely to have a lifetime anxiety disorder (other than OCD; [25.0%] than females [4.6%], to have greater overall functional impairment, and to report a later age of onset (median, 15 years) compared to females (median, 10 years; Grant & Christenson, 2007).

Medical Complications

Picking results in significant tissue damage and often leads to medical complications such as localized infections and septicemia (Lyell, 1972; Neziroglu et al., 2008; Odlaug & Grant, 2008b). Because PSP is often embarrassing, seeking medical help is uncommon (Flessner & Woods, 2006; Grant et al., 2007a). In a study of 31 patients with PSP, only 14 (45%) had ever sought treatment and only 6 of the 31 had ever received dermatological treatment despite resultant infections and “deep craters” (Wilhelm et al., 1999). Another study reported that only 30% of patients with severe skin picking had ever sought treatment even though 6% ultimately needed corrective surgery due to the degree of tissue damage caused by their picking behavior (Neziroglu et al., 2008). The repetitive, excoriative nature of skin picking may even warrant skin grafting (Arnold et al., 1998; Neziroglu et al., 2008; Odlaug & Grant, 2008b) and, in severe cases, resulted in the development of an epidural abscess and paralysis (Weintraub et al., 2000). In rare cases, the behavior can be life-threatening, as demonstrated by the case report of a 48-year-old female who picked through the dermis, subcutaneous tissue, and musculature on her neck, eventually exposing the carotid artery (O’Sullivan et al., 1999). Bilateral capsulotomies using gamma knife radiosurgery have also been reported for a patient with severe, life-threatening, obsessive-compulsive skin picking in which multiple medication trials and skin grafting had been unsuccessful (Kondziolka & Hudak, 2008).

Psychiatric Comorbidity

Psychiatric comorbidity is the rule, not the exception, for patients with PSP. Individuals with PSP have been described as being of an “obsessive-compulsive character” (Zaidens, 1951) and introverted (Fisher & Pearce, 1974). Comorbid conditions, such as anxiety and depressive disorders, are common, and lifetime rates of DSM-IV Axis I disorders have been found to occur in 10.4%–76%

of PSP patients (Calikuşu et al., 2003; Ehsani et al., 2009; Grant et al., 2006a, 2006b; Lochner et al., 2002; Mutasim & Adams, 2009; Neziroglu et al., 2008; Odlaug & Grant, 2007, 2008a, 2008b; Phillips & Taub, 1995; Wilhelm et al., 1999). Specifically, lifetime rates of major depressive (36.4%–58.1%), anxiety (other than OCD; 23%–33%), obsessive-compulsive (10.4%–68%), and body dysmorphic (26.8%–44.9%) disorders have been found in PSP patients. Rates of suicidal ideation (12%), suicide attempts (11.5%), and past psychiatric hospitalizations (15%) due to picking have also been reported (Arnold et al., 1998; Krupp, 1977; Odlaug & Grant, 2008b). Some researchers have speculated that the self-mutilative nature of skin picking is the symbolic equivalent of suicide (Fisher & Pearce, 1974); however, this assertion is only hypothetical. Due to the shame and embarrassment frequently associated with PSP, these co-occurring disorders can further complicate treatment and may increase the severity of the overall picking; however, the extent to which these co-occurring conditions affect the overall picking behavior is unknown.

In addition, other pathological grooming disorders such as trichotillomania and compulsive nail biting are common in patients with PSP. Stokes and Garner (1929) mention that evidence of nail and knuckle biting were common conditions in a patient with neurotic excoriation. More recently, a study of 60 patients with PSP found high lifetime rates of co-occurring trichotillomania (38.3%) and compulsive nail biting (31.7%; Odlaug & Grant, 2007). These rates are substantially higher than the rates of trichotillomania and compulsive nail biting seen in the general population, which have been noted to range from 0.6% to 3.9% and 6.4%, respectively (Christenson et al., 1991; Odlaug & Grant, 2010).

Family History

Significantly higher rates of first-degree relatives with an alcohol use disorder were recently found in a study of 50 subjects with PSP versus 50 controls (Mutasim & Adams, 2009) although the specific percentage of affected individuals was not disclosed. In a sample of 60 patients with PSP, 28.3% of first-degree family members had PSP and 10.0% had trichotillomania (Odlaug & Grant, 2008b), while another sample of the first-degree relatives of 33 PSP patients found that 15.2% had an alcohol use disorder (Odlaug & Grant, 2008a). A recent study of 40 PSP subjects found that 28 (87.5%) had a

first-degree relative with a psychiatric diagnosis, and 43% had a first-degree relative with skin picking (Neziroglu et al., 2008).

Etiology and Pathophysiology

The etiology and pathophysiology of PSP are unknown. Animal research, human genetic research, and neurocognitive examinations, however, all provide useful clues to the possible pathophysiology of PSP.

Animal Models

Existing ethological models focus on spontaneously arising repetitive or stereotypic behaviors, such as tail chasing and fur chewing (Brown et al., 1987; Stein et al., 1994) and behaviors driven by conflict, frustration, or stress, such as grooming, cleaning, and pecking (Stein et al., 1994). Bordnick and colleagues (1994) presented an avian model of feather-picking disorder that is relevant to PSP. Psychogenic feather picking in birds is associated with feather loss or damage to body areas arising from chewing, plucking, and destruction of feathers (Moon-Fanelli et al., 1999).

Genetic Research

Another candidate model of PSP is the *Hoxb8* gene knockout mouse. Greer and Capecchi (2002) reported that mice with mutations of the *Hoxb8* gene, compared to controls, groomed excessively to the point of developing skin lesions and hair removal. Mutant mice demonstrated normal cutaneous sensation and there was no evidence of an inflammatory response, suggesting that this behavior was not due to skin or other abnormalities of the peripheral nervous system. This model is promising because the excessive grooming of *Hoxb8* mutants is similar to that seen in PSP. Moreover, the *Hoxb8* gene is expressed in the orbital cortex, the anterior cingulate, the striatum and the limbic system.

Another possible genetic model for PSP can be found in the animal research on the gene encoding SAPAP3, a scaffolding protein found in excitatory glutamate-responsive synapses largely in the striatum region of the brain. Mice that were deficient in SAPAP3 demonstrated excessive grooming that continued past the point of self-harm (Welch et al., 2007). This research was later reinforced in a human genetic association study that found that a variation in the SAPAP3 gene is associated with human grooming disorders such as PSP (Bienvenu et al., 2009).

Cognitive Functioning

In humans, problems with inhibitory control may be expected in PSP, given that individuals usually report an inability to stop their behavior, and understanding these possible cognitive deficits in inhibitory control may provide clues to the possible neurobiology of PSP. Motor impulsivity is classically assessed using tasks that require volunteers to make simple motor responses (e.g., pressing a button) on some computer trials but not on others. Response inhibition as a cognitive function is dependent on neural circuitry including that of the right inferior frontal gyrus (Aron et al., 2007; Robinson et al., 2008). In the only study to assess the neurocognitive function of patients with PSP, 20 individuals with PSP and 20 controls were tested on their cognitive flexibility and set-shifting abilities (Odlaug et al., 2009). Motor inhibition (impulsivity) was examined using the stop-signal task (SST), which is sensitive to damage in the right frontal gyrus and measures the time taken to internally suppress a motor response (Aron et al., 2003). Cognitive flexibility, or set-shifting, was measured using the intradimensional/extradimensional shift task (IDED), a test assessing frontal lobe integrity (Lezak et al., 2004). The study found that subjects with PSP exhibited impaired inhibitory control on the SST but intact cognitive flexibility on the IDED task. The results proved to be consistent with those of studies examining the same tests in trichotillomania, suggesting possible cognitive and physiological similarities between the two disorders (Chamberlain et al., 2006).

Neurobiology

An electroencephalographic (EEG) study of 54 patients with PSP demonstrated significant abnormalities (51.9%) versus controls (25%) in bilateral (specifically nonfocal) theta activity (Fruensgaard et al., 1980). The authors suggest that repressed aggressive tendencies contributing to the onset of PSP may explain these abnormalities compared to controls. However, further EEG examinations with larger groups of PSP patients are necessary to validate this theory.

Psychological Theories

Psychodynamic theorists report that picking behavior and self-inflicted wounds caused by the picking are the manifestations of repressed rage toward an authoritarian parent exhibited by self-destructive acts that allow the individuals to assert themselves toward that authority (Zaidens, 1951).

Overbearing and fussy parents have also been postulated as being markers of picking behavior onset in children (Stokes & Garner, 1929), construing that the inability of the child to handle the stress associated with these types of parents may contribute to the picking behavior.

In early reports of picking behavior, researchers surmised that “hysteria” or having a family history of insanity was evidence of an unstable nervous system contributing to picking onset (Pusey & Senear, 1920).

Marital conflicts, early-life or recent deaths of loved ones, and unwanted pregnancies have also been reported as symptom onset triggers (Krupp, 1977).

More recently, it has been proposed that patients with PSP have an impaired ability to tolerate stress and subsequently respond to stress by picking (Doran et al., 1985).

Classification of PSP

It has been hypothesized that PSP is a member of a putatively related collection of obsessive-compulsive conditions (Bienvenu et al., 2000; Stein & Hollander, 1995). The repetitive motor behavior of skin picking with perceived diminished control bears a striking resemblance to OCD (Stein et al., 2006). Although the picking in PSP decreases anxiety, as do compulsions in OCD, it may also produce a feeling of pleasure (Keuthen et al., 2010), whereas the compulsions of OCD typically do not. In contrast to OCD, in which compulsions occur in a variety of situations, individuals with PSP tend to pick most often when engaged in sedentary activities (Flessner & Woods, 2006). In addition, PSP has traditionally been considered a disorder predominantly affecting females (Arnold et al., 2001; Calikuşu et al., 2003; Fisher & Pearce, 1974; Flessner et al., 2006; Fruensgaard, 1984; Krupp, 1977; Mutasim & Adams, 2009; Wilhelm et al., 1999), whereas OCD has a 1:1 gender ratio.

Rates of co-occurring OCD are significantly higher in individuals with PSP (6%–52%; Arnold et al., 1998; Lochner et al., 2002; Odlaug & Grant, 2008a) than those found in the community (1%–3%), and rates of PSP among individuals with OCD have ranged from 8.9% to 24.0% (Cullen et al., 2001; Grant et al., 2006a), which is greater than the prevalence of 1.4% found in the community (Keuthen et al., 2010). These findings raise the possibility of an underlying common neurobiological pathway for the compulsion seen in these two disorders. Cognitive testing has also shown a lack of

a relationship with PSP subjects demonstrating poor motor inhibition, whereas OCD subjects exhibit motor and cognitive inflexibility (Odlaug et al., 2009; Chamberlain et al., 2006).

Treatments evaluated for PSP and OCD include pharmacological and behavioral interventions and further suggest a possible, albeit complicated, relationship between these disorders (see the chapter “Assessment and Treatment of Pathological Skin Picking”). It is well established that the pharmacological first-line treatment for OCD is a serotonin reuptake inhibitor (SRI; e.g., clomipramine, fluvoxamine, fluoxetine, paroxetine). The data on the efficacy of SRIs for PSP, however, are less convincing. Although fluoxetine has shown some benefit for PSP (Simeon et al., 1997), other pharmacological agents (e.g., naltrexone, lamotrigine) that have shown benefit for PSP have not been effective for OCD. Obsessive-compulsive disorder and PSP respond to behavioral interventions; however, the mode of behavioral treatment differs substantially for PSP.

Despite a proposed relationship between OCD and PSP, there are important differences between these disorders. The notion of an OCD spectrum remains contentious (Chamberlain et al., 2007; Elliott & Fuqua, 2000). Thus, the obsessive-compulsive spectrum of disorders may represent a mixed group of illnesses whose central feature involves the performance of repetitive, unwanted acts. Key cross-cutting issues include demographics, comorbidity rates, inheritance patterns, neurocognitive and brain imaging profiles, and treatment response. Comparison of candidate spectrum disorders across these factors may allow hypotheses to be generated regarding the closeness or lack of closeness of their interrelationship.

Alternative Conceptualizations of PSP

Currently, PSP is classified as an impulse control disorder not otherwise specified. As such, some research suggests that the impulse control disorders, including PSP, should be thought of as behavioral addictions with common features shared with substance addictions: (1) repetitive or compulsive engagement in the behavior despite adverse consequences; (2) diminished control over the problematic behavior; (3) an appetitive urge or craving state prior to engaging in the problematic behavior; and (4) a hedonic quality during the performance of the problematic behavior (Grant et al., 2007b).

Another perspective is that PSP constitutes a member of several related body-focused repetitive

(BFR) behaviors, along with trichotillomania and nail biting (Stein et al., 2007). The BFR behaviors can be seen as constituting a spectrum from routine, commonplace, and harmless to an extreme pathological form. The latter form is not undertaken for purposes of cosmetic improvement but rather is characterized by the uncontrolled nature of the picking. Few studies have compared these behaviors within and against each other. In one such comparative study of 33 individuals with PSP, 24 individuals with trichotillomania, and 20 with co-occurring disorders, researchers found significant similarities across all three groups. Age of onset, gender ratio, previous medication treatment-seeking behavior, a high prevalence of both current and lifetime comorbid psychiatric disorders, a focus on symmetry, and overall severity as measured by the Clinical Global Impressions scale (CGI; Guy, 1976) were similar across all three groups (Odlaug & Grant, 2008a). The PSP group, however, was differentiated from the other two groups in that they were less likely to have received psychotherapy and reported spending more time overall engaging in the behavior (mean of 105.8 minutes per day).

There are many similarities between patients with PSP and those who perform self-injurious behaviors such as cutting. Favarra (1998) divided moderate self-mutilation into two categories, one based on episodic behavior (as seen in self-cutting) and the other based on compulsive behavior (as seen in PSP). One primary similarity is the report of increasing tension immediately preceding the act (Pao, 1969). Furthermore, in many cases, the acts begin unconsciously and patients become aware of the behavior after damage to the skin has been done (evidenced by bleeding; Odlaug & Grant, 2008b; Pao, 1969). This dissociative picking behavior and reported absence of pain while picking are common in PSP but are also seen frequently in other self-mutilative behaviors (Neziroglu & Mancebo, 2001). Also, most patients with PSP or cutting (and other self-injurious behaviors) will not act on this tension in the presence of other people but wait for solitude in order to engage in the behavior.

Conclusions

Pathological skin picking is a common disorder with high rates of psychiatric as well as medical morbidity. The etiology of PSP appears to be neurobiological rather than dermatological in nature. Continued exploration of the pathophysiology of PSP will clarify whether the disorder is distinct from OCD and should allow the development of more

effective treatment options. The current trend of using cognitive measures to better understand the pathophysiology of these disorders should allow pharmacotherapy and psychosocial treatment options to target more specific areas of brain dysfunction. Pathological skin picking appears more closely related pathophysiological to other grooming disorders such as nail biting and hair pulling. The neurobiological and psychological nature of this disorder appears to necessitate collaborative efforts among dermatologists, psychologists, and psychiatrists to improve treatment outcomes (Mercan & Altunay, 2006).

Future Directions

To date, very few studies have examined individuals with PSP. Future research identifying the core characteristics of this behavior may advance treatment options for patients with PSP. Furthermore, there currently exists very little awareness in the community regarding both the impact of PSP and the fact that efficacious treatments are available. Disseminating information throughout the community to schools, workplaces, and health care providers may advance the understanding of this often disabling disorder.

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Epidemiology and Phenomenology of Compulsive Buying Disorder

Donald W. Black

Abstract

Compulsive buying disorder (CBD) is defined as excessive shopping cognitions and buying behavior that leads to distress or impairment. This chapter presents an overview of its definition and recognition, clinical symptoms, epidemiology, natural history, and both cultural and family factors. Compulsive buying disorder is found worldwide and has a lifetime prevalence of 5.8% in the U.S. general population. The disorder has a female preponderance, has an onset in the late teen years or early 20s, appears to be chronic or recurrent, and occurs mainly in women. Subjects with CBD report a preoccupation with shopping, prepurchase tension or anxiety, and a sense of relief following a purchase. Compulsive buying disorder is associated with significant psychiatric comorbidity, particularly mood and anxiety disorders, substance use disorders, eating disorders, other disorders of impulse control, and Axis II disorders, although there is no special “shopping” personality. The disorder tends to run in families, and these families are filled with mood and substance use disorders.

Keywords: compulsive buying disorder, CBD, epidemiology, gender factors

Introduction

Mary, a 47-year-old divorced woman, had shopped compulsively since she first obtained credit cards at age 19. She knew her shopping behavior was excessive and reported that it had been continuous for nearly its entire duration. She had controlled her shopping briefly for only two short periods, both coinciding with bankruptcy proceedings. Mary was currently in debt on four credit cards. Her life revolved around shopping and spending, even though she worked full time. Spending gave her a “rush,” despite the guilt she experienced afterward. She would either return items or give them away. She enjoyed shopping—mostly alone—at expensive department stores and would spend her money mainly on clothing and shoes. She was unhappy with her inability to control her spending, and was ashamed that her behavior had contributed to her divorce as well as to her serious financial problems. She described the shopping behavior as relatively

spontaneous and impulsive and generally not planned. Despite her problems, she was not depressed and had never sought psychiatric care.

Compulsive buying behavior has been described for centuries. Reports mainly involve the wealthy and powerful. For example, Marie Antoinette was known for her extravagance during the turbulent time before the French Revolution (Castelot, 1957; Erickson, 1991). Mary Todd Lincoln, wife of President Abraham Lincoln, had spending binges that distressed her husband (Baker, 1987). William Randolph Hearst, publisher and inveterate collector, had an insatiable appetite for art and antiques that nearly drove him to bankruptcy during the Great Depression (Swanberg, 1961). Jacqueline Kennedy Onassis was widely known as a clothes horse and obsessive shopper whose uncontrolled behavior dismayed both of her husbands (Heymann, 1989). Whether these individuals had a compulsive buying disorder (CBD) is a matter of debate, yet

each was observed to have episodes of excessive and sometimes senseless spending that contributed to their financial or personal difficulties. Most compulsive shoppers are not famous or wealthy, and—like Mary in the case study above—are ordinary people whose excessive shopping has become an irresistible and costly way of life. These are the compulsive buyers whose lives are organized around a variety of shopping and spending experiences, and whose behavior has prompted concerns that it can lead to a clinical disorder.

The first clinical description of CBD was given by the German psychiatrist Emil Kraepelin (1915), who wrote about the uncontrolled shopping and spending behavior of “buying maniacs.” He was later quoted by the Swiss psychiatrist Eugen Bleuler (1930) in his *Lehrbuch der Psychiatrie*:

As a last category, Kraepelin mentions the buying maniacs (oniomaniacs) in whom even buying is compulsive and leads to senseless contraction of debts with continuous delay of payment until a catastrophe clears the situation a little—a little bit never altogether because they never admit all their debts. According to Kraepelin, here, too, it always involves women. The usually frivolous debt makers and who in this way wish to get the means for pleasure naturally do not belong here. The particular element is impulsiveness; they cannot help it, which sometimes even expresses itself in the fact that notwithstanding a good school intelligence, the patients are absolutely incapable to think differently and to conceive the senseless consequences of their act, and the possibilities of not doing it. They do not even feel the impulse, but they act out their nature like the caterpillar which devours the leaf. (p. 540)

Kraepelin and Bleuler each considered “buying mania” an example of a *reactive impulse* or *impulsive insanity* and placed it alongside kleptomania and pyromania. They may have been influenced by the French psychiatrist Jean Esquirol’s (1838) earlier concept of *monomania*, a term he used to describe otherwise normal persons who had some form of pathological preoccupation.

Despite this early work, CBD attracted little attention except for occasional reports in the psychoanalytic literature (Krueger, 1988; Lawrence, 1990; Stekel, 1924; Winestine, 1985). Interest was rekindled in the late 1980s and early 1990s through a convergence of events. Consumer behavior researchers showed the disorder to be widespread (Elliott, 1994; Magee, 1994; O’Guinn & Faber,

1989), and descriptive studies began to appear in the psychiatric literature (Christensen et al., 1994; McElroy et al., 1991, 1994; Schlosser et al., 1994). These reports were consistent in presenting a picture of an identifiable clinical disorder that mainly affected women, caused considerable distress, impaired psychosocial functioning, and was associated with substantial psychiatric comorbidity.

Diagnosis and Classification

Compulsive buying disorder is not included in contemporary diagnostic systems, such as the *Diagnostic and Statistical Manual of Mental Disorders—Text Revision* (DSM-IV-TR; American Psychiatric Association, 2000), or the World Health Organization *International Classification of Diseases* (10th ed.; World Health Organization, 1992).

Following in the tradition of criteria-based diagnoses, McElroy et al. (1994) developed an operational definition for CBD that emphasizes cognitive and behavioral aspects of the disorder. Their definition requires evidence of impairment from marked subjective distress, interference in social or occupational functioning, or financial/legal problems. Further, the disorder cannot be attributed to mania or hypomania. These criteria have been embraced by researchers, yet neither their reliability nor their validity has been established. Some writers have criticized attempts to categorize CBD as an illness, which they see as part of a trend to “medicalize” behavioral problems (Lee & Mysyk, 2004). Whether to include CBD in the next edition of the DSM is being considered (Hollander & Allen, 2006).

Other definitions for CBD have come from consumer behavior researchers or social psychologists. Faber and O’Guinn (1992) defined the disorder as “chronic buying episodes of a somewhat stereotyped fashion in which the consumer feels unable to stop or significantly moderate his behavior” (p. 738). Edwards (1993), another consumer behaviorist, suggests that compulsive buying is an “abnormal form of shopping and spending in which the afflicted consumer has an overpowering uncontrollable, chronic and repetitive urge to shop and spend [that functions] . . . as a means of alleviating negative feelings of stress and anxiety” (p. 67). Dittmar (2004) describes three cardinal features: irresistible impulse, loss of control, and carrying on despite adverse consequences. Some consumer behavior researchers consider CBD part of a spectrum of aberrant consumer behavior, which includes pathological gambling, shoplifting, and credit abuse (Budden & Griffin, 1996).

The appropriate classification of CBD remains elusive, a fact reflected by the many terms used to describe the condition: *compulsive shopping*, *addictive shopping*, *shopaholism*, *compulsive buying*, and even *mall mania*. McElroy et al. (1991) had suggested that compulsive shopping behavior might be related to “mood, obsessive-compulsive or impulse control disorders.” Hollander (1993) later described a spectrum of disorders that he has connected to obsessive-compulsive disorders including CBD, while Lejoyeux et al. (1996) have linked it to the mood disorders. Others consider CBD to be related to the addictive disorders such as alcohol and drug dependence (Glatt & Cook, 1987; Goldman, 2000; Krych, 1989). Finally, other investigators have followed in the tradition pioneered by Kraepelin and Bleuler and suggest classifying CBD as a disorder of impulse control. Compulsive buying disorder’s relationship to other impulse control disorders was also recognized by Wilhelm Stekel (1924), a follower of Freud, who proposed that uncontrolled buying was a *forme fruste* of kleptomania (Maier, 1997).

Hollander and Allen (2006) have suggested that CBD be included in a new diagnostic category that combines behavioral and substance addictions. In this model, *behavioral addictions* include pathological gambling, kleptomania, pyromania, CBD, Internet addiction, and compulsive sexual behavior. The National Institute on Drug Abuse considers behavioral addictions to be relatively pure models of addiction because they are not contaminated by the presence of an exogenous substance (Holden, 2001).

Differential Diagnosis

When considering a diagnosis of CBD, the patient’s pattern of shopping and spending must be distinguished from normal buying behavior. Because shopping is a major pastime for people in the United States and other developed countries (Farrell, 2003), frequent shopping does not by itself constitute evidence in support of a CBD. For the person with CBD, the frequent shopping and spending have a compulsive and irresistible quality and lead to deleterious consequences. Normal buying can take on a compulsive quality, particularly around the time of special holidays or birthdays, but the excessive buying is not persistent, nor does it lead to distress or impairment. People who receive an inheritance or win a lottery may experience shopping sprees as well. The clinician should exercise judgment in applying the diagnostic criteria of McElroy et al. (1994) and be mindful of the need for evidence of distress or impairment.

Next, bipolar disorder must be ruled out as the cause of excessive shopping and spending. When due to mania, a patient’s unrestrained spending typically corresponds with elevated mood and is accompanied by grandiosity, unrealistic plans, and an expansive or irritable affect. Shopping and spending in the person with CBD are ongoing and lack the periodicity seen in bipolar patients (Kuzma & Black, 2006). Clinicians should also rule out medical explanations as a cause of the CBD. There is a growing literature, for example, on the relationship between compulsive behaviors, including CBD, and the use of anti-parkinsonian medications such as pramipexole (Lader, 2008). Thus, clinicians need to rule out any possible effect of pramipexole or similar drugs on the patient’s behavior before diagnosing CBD.

Clinical Presentation

The hallmark of CBD is the person’s preoccupation with shopping and spending. Many hours may be devoted each week to these behaviors (Christenson et al., 1994; Schlosser et al., 1994). Persons with CBD often describe increasing anxiety and tension that are relieved only when a purchase is made. Compulsive buying disorder behaviors occur year round but can be more problematic during Christmas, other holidays, and special days such as birthdays of family members and friends.

Schlosser et al. (1994) reported that subjects show a range of behavior regarding the outcome of a purchase: returning the item, failing to remove the item from the package, selling the item, or even giving it away. Compulsive shopping tends to be a private experience, so individuals with CBD prefer to shop alone (Schlosser et al., 1994). Compulsive shopping can occur in any venue: high-fashion department stores and boutiques, consignment shops, garage sales, or catalogs (Christenson et al., 1994). Dittmar (2007) has documented how CBD has gained a strong foothold with online buying. Compulsive buyers are mainly interested in consumer goods such as clothing, shoes, crafts, jewelry, gifts, makeup, and CDs/DVDs (Christenson et al., 1994; Mitchell et al., 2006; Schlosser et al., 1994).

Compulsive shoppers often display a great fashion sense and have an intense interest in new clothing styles and products. They may report buying a product based on its attractiveness or because it was a “bargain” (Frost et al., 1998). Individually, items purchased tend not to be large or expensive, but many compulsive shoppers will buy in quantity, so that spending rapidly escalates. During a typical

episode, compulsive shoppers have reported spending an average of \$110 (Christenson et al., 1994), \$92 (Schlosser et al., 1994), or \$89 (Miltenberger et al., 2003). Compulsive buying disorder has little to do with intellect or educational level and has been observed to occur in mentally retarded persons (Otter & Black, 2007).

Several writers have emphasized the emotional significance of the types of objects purchased, which may address personal and social identity needs (Dittmar, 2007; Richards, 1996). Richards (1996) stressed the role of clothing in developing a feminine identity and noted that voids in one's identity have their roots in failed parent-child interactions. Krueger (1988) observed that emotionally deprived persons unconsciously replace what is missing with objects in an attempt to "fill the emptiness of depression and the absence of self-regulation" (p. 582). These explanations for compulsive buying behaviors may apply to some but certainly not all persons with CBD. One relevant study found that self-image concerns were more closely linked to the motivations underlying CBD in women than in men (Dittmar & Drury, 2000).

Miltenberger et al. (2003) reported that negative emotions, such as anger, anxiety, boredom, and self-critical thoughts, were the most common antecedents to shopping binges in a group of persons with CBD; euphoria or relief of negative emotions has been the most common immediate emotional reaction (Elliott et al., 1996). Lejoyeux et al. (1996) concluded that for some persons, "uncontrolled buying, like bulimia, can be used as a compensatory mechanism for depressive feelings" (p. 1528). Faber and Christenson (1996) commented on the close relationships among shopping, self-esteem, and negative emotions. Faber (1992) concluded that shopping behavior is likely to become problematic when it provides a sense of recognition and acceptance for people with low self-esteem, allowing them to act out anger or aggression while providing an escape from their day-to-day drudgery.

Natarajan and Goff (1991) have identified two independent factors in CBD: (1) buying urge or desire and (2) degree of control over buying. In their model, compulsive shoppers combine high urge with low control. This view is consistent with clinical reports that compulsive buyers are preoccupied with shopping and spending and will try to resist their urges but often have little success (Black, 2001; Christenson et al., 1994). For example, in the study of Christenson et al. (1994), 92% of persons with the disorder described attempts to resist urges to

buy but reported that their attempts were often unsuccessful. Subjects indicated that the urge to buy resulted in a purchase 74% of the time. Typically, 1 to 5 hours passed between initially experiencing the urge to buy and the eventual purchase.

Income has relatively little to do with CBD. Level of income may lead one person to shop at a consignment shop, while the other shops at a high-end boutique. Persons with a low income can be as preoccupied with shopping and spending as wealthier individuals (Black, 2001; Dittmar, 2007). Wealth does not protect against CBD either, because the presence of CBD may cause or contribute to interpersonal, occupational, or marital problems even when it does not create financial or legal problems. Moreover, Koran et al. (2006) found that compared to other respondents, individuals with CBD were more likely to report an income under \$50,000, were less likely to pay off credit card balances in full, and gave maladaptive responses regarding their consumer behavior. In this study, compulsive buyers engaged in "problem shopping" more frequently and for longer periods, and were more likely than other respondents to feel depressed after shopping, to make senseless and impulsive purchases, and to experience uncontrollable buying binges.

Prevalence

Prevalence surveys provide rates for CBD that range from 1.4% to 44%. The wide range is likely due to differences in the populations examined and the research methods used (Table 16.1). In general, adolescents and college students had higher rates of CBD than general adult populations. In one of the first studies, Faber and O'Guinn (1992) estimated the prevalence of CBD at between 1.8% and 8.1% of the general population based on results from a mail survey. The Compulsive Buying Scale (CBS; Faber & O'Guinn, 1989) was given to 292 individuals who were selected to approximate the demographic makeup of the general population of Illinois. The high and low prevalence estimates reflect different thresholds (or cut points) set for CBD. The higher figure is based on a probability level of .70 (i.e., two standard deviations above the mean). The lower figure is based on a more conservative probability level of .95 (i.e., three standard deviations above the mean). These authors recommend using the probability level of .70 with the CBS.

Dittmar (2005) conducted two studies that address prevalence. In the first, she queried 194 persons who responded to an unsolicited mail survey and were residentially matched to a group of persons

Table 16.1 Prevalence Surveys of CBD

Study	Location	Diagnostic Method	Sample Size	Setting	Findings
Faber and O'Guinn (1992) ¹	Illinois	CBS	292	General population	1.8%/8.1%
Magee (1994)	Arizona	CBS	94	College students	16%
Hassay & Smith (1996)	Manitoba, Canada	CBS	92	College students	12%
Roberts (1998)	Texas	CBS	300	College students	6%
Dittmar (2005)	England	CBS	194	General population	13.5%
Dittmar (2005)	England	CBS	195	Adolescents	44.1%
Neuner et al. (2005) ²	Germany	ABS	1527/1017	General population	6.5%/8%
Grant et al. (2005)	Minnesota	MIDI	204	Psychiatric inpatient unit	9.5%
Koran et al. (2006) ¹	United States	CBS	2513	General population	1.4%/5.8%

CBS = Compulsive Buying Scale; ABS = Addictive Buying Scale; MIDI = Minnesota Impulsive Disorders Interview.

¹The study used conservative and liberal cut points with the CBS.

²The study involved interviews with East and West Germans in 1991/2001.

with shopping problems. Using the CBS, she found that 13.4% of persons in the comparison group met the threshold for CBD. She also sampled 195 adolescents aged 16 to 18 years; 44.1% scored above the CBS scale threshold, indicating the presence of a CBD.

More recently, Koran et al. (2006) used the CBS to identify compulsive buyers in a random telephone survey of 2513 U.S. adults and estimated the point prevalence at 5.8% of respondents. The estimate was calculated by using CBS scores two standard deviations above the mean. A prevalence of 1.4% was calculated using the more strict criterion of three standard deviations above the mean.

Three small surveys of college students—all utilizing the CBS—are relevant. Magee (1994) reported that 16% of 94 undergraduates were compulsive buyers. Hassay and Smith (1996) found that 12.2% of 92 undergraduates were compulsive buyers. Roberts (1998) reported that 6% of 300 college students met the cutoff for CBD. These authors each used the recommended cutoff of two standard deviations above the mean.

Neuner et al. (2005) designed an interesting study to address whether CBD is increasing in prevalence. They reported that the frequency of compulsive buying in Germany increased from 1991 to 2001. Using the Addictive Buying Scale, these investigators found that the frequency of CBD increased from 1% to 6.5% in East Germany and

from 5% to 8% in West Germany. They attributed the rapid rise of CBD in the former East Germany in part to the acculturation process brought about by reunification.

Lastly, in the clinical setting Grant et al. (2005) utilized the Minnesota Impulsive Disorders Interview (Christenson et al., 1994) to assess CBD and reported a lifetime prevalence of 9.3% among 204 consecutively admitted psychiatric inpatients.

Cultural Factors

Because CBD occurs mainly in developed countries, cultural and social factors have been proposed as either causing or promoting the disorder (Black, 2001; Dittmar, 2007). The disorder has been described worldwide, with reports coming from the United States (Christenson et al., 1994; McElroy et al., 1994; Schlosser et al., 1994), Australia (Kyrios et al., 2002), Canada (Valence et al., 1988), England (Dittmar, 2004; Elliott, 1994), Germany (Scherhorn et al., 1990), France (Lejoyeux et al., 1997), the Netherlands (Otter & Black, 2007), Mexico (Roberts & Sepulveda, 1999), South Korea (Kwak et al., 2003), Spain (Villarino et al., 2001), and Brazil (Bernik et al., 1996).

The presence of a market-based economy, the availability of a wide variety of goods, easily obtained credit, disposable income, and significant leisure time are elements that appear necessary for the development of CBD (Lee & Mysyk, 2004). For these

reasons, CBD is unlikely to occur in poorly developed (or developing) countries except among the wealthy elite or the growing middle classes. A website specifically designed to offer resources to compulsive buyers and their families (<http://www.stoppingovershopping>) has attracted visitors from over 50 countries, suggesting that the disorder is nearly universal (A. Benson, personal communication).

Age at Onset and Gender

Compulsive buying disorder has an onset in the late teens or early 20s, which may correlate with emancipation from the nuclear family (Table 16.2), as well as with the age at which people first establish credit (Black, 2001). Roberts and Tanner (2000, 2002) showed that uncontrolled buying in adolescents is associated with a more generalized pattern of behavioral disinhibition that includes smoking, alcohol and drug use, and early sex.

Community-based clinical studies, and the survey results of Faber and O'Guinn (1992), suggested that 80% to 94% of persons with CBD are women (Table 16.2). In contrast, Koran et al. (2006) reported that the prevalence of CBD in their random telephone survey was nearly equal for men and women (5.5% and 6.0%, respectively). Their finding suggests that the reported gender difference may be spurious, and are due to the fact that women more readily acknowledge abnormal shopping behavior than do men; women also are more willing to participate in research protocols. Based on the results of a general population survey in the United Kingdom, Dittmar (2004) concluded that the gender difference is real, and not an artifact of men being underrepresented in clinical samples. In her study, 92% of respondents stated that compulsive shoppers were women. That said, the methods used in this survey have not been published, so it is

Table 16.2 Studies Involving Persons with CBD

Investigator(s)	Location	Subjects, n	Age, Years, Mean	% Female	Age at Onset, Years, Mean	Duration of Illness, Years, Mean
O'Guinn and Faber (1989)	Los Angeles, CA	386	37	92	N/A	N/A
Scherhorn et al. (1990)	Germany	26	40	85	N/A	N/A
McElroy et al. (1994)	Cincinnati, OH	20	39	80	30	9
Christensen et al. (1994)	Minneapolis, MN	24	36	92	18	18
Schlosser et al. (1994)	Iowa City, IA	46	31	80	19	12
Black et al. (1998)	Iowa City, IA	33	40	94	N/A	N/A
Ninan et al. (2000)	Cincinnati, OH; Boston, MA	42	41	81	N/A	N/A
Koran et al. (2002)	Stanford, CA	24	44	92	22	22
Miltenberger et al. (2003)	Fargo, ND	19	N/A	100*	18	N/A
Mitchell et al. (2006)	Fargo, ND	39	45	100*	N/A	N/A
Muller et al. (2008)	Bavaria, Germany	60	41	85	27	14

*Indicates that the sample recruited was female.

difficult to assess the representativeness of her sample or the completeness of the response to the survey.

Natural History and Course

There are no careful follow-up studies of CBD. Clinical reports suggest that the disorder is chronic and fluctuating in severity and intensity. Schlosser et al. (1994) reported that 59% of their subjects described their course as continuous and 41% as episodic. Of the 20 subjects described in the study of McElroy et al. (1994), 60% had a chronic course while 8% described an episodic course. For the subjects in the studies listed in Table 16.2, CDB had been present for 9 to 22 years, although these clinical samples may be biased in favor of greater severity. In a medication study, Aboujaoude et al. (2003) suggested that persons who responded to treatment with citalopram were likely to remain in remission during a 1-year follow-up period. Mueller et al. (2008) similarly reported that treatment with group cognitive-behavior therapy led to improvement that was maintained during a 6-month follow-up period. These studies suggest that treatment can alter the natural history of the disorder.

Quality of Life

Quality-of-life data are limited. Nonetheless, research shows that CBD adversely impacts the lives of those with the disorder and those of their family members. First, most persons with CBD admit that the disorder is subjectively distressing and that they feel unable to control their behavior (Christenson et al., 1994; Schlosser et al., 1994). They report that the disorder has led to marital and family problems, including separation and divorce. Financial problems that include substantial debt can lead to bankruptcy, and in some cases persons will turn to crime (e.g., embezzlement, shoplifting) to fuel their shopping or to repay their debts. Lejoyeux et al. (1997) reported that CBD is associated with suicide attempts. There are no reports of CBD leading to completed suicide.

Family History

There are few studies presenting family history data on CBD. McElroy et al. (1994) reported that of 18 individuals with CBD, 17 had one or more first-degree relatives with major depression; 11 had an alcohol or other substance use disorder; 3 had an anxiety disorder; and 3 had relatives with CBD. Black et al. (1998) used the family history method to assess 137 first-degree relatives of 31 persons

with CBD. Relatives were significantly more likely than those in a comparison group to have a depressive disorder, an alcohol disorder, or a drug use disorder. They were also more likely to have "any psychiatric disorder" and "more than one psychiatric disorder." Compulsive buying disorder was identified in 9.5% of the first-degree relatives but was not assessed in the comparison group.

Psychiatric Comorbidity

Psychiatric comorbidity is common in persons with CBD. Data from clinical studies confirm high rates of major mental (Axis I) disorders (Table 16.3), particularly for mood (21%–100%), anxiety (41%–87%), substance use (21%–46%), disorders of impulse control (21%–40%), and eating disorders (8%–35%). Three clinical studies included comparison groups: 1) Black et al. (1998) reported that major depression and "any" mood disorder were excessive; 2) Christenson et al. (1994) reported that the categories of anxiety, substance use, eating, and impulse control disorders were each excessive; and 3) Mueller et al. (2009) found that persons with CBD had higher rates of mood, anxiety, and eating disorders than community controls, and higher rates of mood and anxiety disorders compared to bariatric controls.

Compulsive buying disorder may have a special relationship with obsessive-compulsive disorder (OCD). In clinical samples, from 3% (Black et al., 1998) to 40% (Mueller et al., 2009) of individuals with CDB are reported to have comorbid OCD. Likewise, the presence of CBD may characterize a specific subset of OCD patients (du Toit et al., 2001; Hantouche et al., 1996, 1997), particularly those who hoard, a special symptom that involves the acquisition of, and failure to discard, possessions of limited use or value (Frost et al., 2001). Unlike the items kept by the typical hoarder, items purchased by the individual with CBD are not inherently useless or lacking in value.

Axis II comorbidity is also common. Schlosser et al. (1994) used both a self-report instrument and a structured interview to assess personality disorders in 46 persons recruited from the community. Nearly 60% met criteria for at least one personality disorder through a consensus of both instruments. The most commonly identified personality disorders were the obsessive-compulsive (22%), avoidant (15%), and borderline (15%) types. Mueller et al. (2008) examined 48 persons participating in a clinical trial and reported that depressive, avoidant, and obsessive-compulsive personality disorders were frequent.

Table 16.3 Lifetime Psychiatric Comorbidity in Persons with CBD

Comorbid disorder	Schlosser et al. (1994)	Christenson et al. (1994)	McElroy et al. (1994)	Lejoyeux et al (1997)	Black et al. (1998)	Ninan et al. (2000)	Koran et al. (2002)	Mitchell et al. (2006)	Muller et al. (2009)
Instrument used	DIS	SCID	SCID	MINI	SCID	SCID	MINI	SCID	SCID
Mood disorder,%	28	54	95	100	61	45	8	62	80
Anxiety disorder,%	41	50	80		42			26	87
Substance use disorder,%	30	46	40		21			33	23
Somatoform disorder, %	11		10						
Eating disorder, %	17	21	35	21	15		8	18	33
Impulse control disorder, %		21	40						20

DIS = Diagnostic Interview Schedule; SCID = Structured Clinical Interview for DSM-IV; MINI = Mini International Neuropsychiatric Interview.

In a controlled study, Mueller et al. (2009) reported that 30 persons with CBD had higher rates of personality disorders than community controls or bariatric clinic patients, particularly depressive, avoidant, obsessive-compulsive, and borderline types. Anecdotally, Krueger (1988) observed that the four patients he treated using psychoanalysis each exhibited aspects of narcissistic character pathology.

In perhaps the largest study to assess comorbidity, Mueller et al. (in press) pooled the data from 175 compulsive buyers in the United States and Germany. (Because the pooled data were previously published, these results are not included in Table 16.2.) Nearly 90% of the subjects had at least one lifetime Axis I diagnosis, including mood (74%) and anxiety (57%) disorders. Half of the subjects had at least one current Axis I disorder, most commonly an anxiety disorder (40%). Twenty-one percent had a comorbid lifetime impulse control disorder, most commonly intermittent explosive disorder (11%).

In this study, Mueller et al. (2010) used latent profile analysis and were able to identify two clusters. The second cluster included people with more severe compulsive buying who had higher rates of current and lifetime Axis I and impulse control disorders than those in the first cluster. These data are partially compatible the findings of Black et al. (2001), who divided a sample of 44 individuals with CBD into four groups ranked from most to least severe based on their CBS score. Greater severity was associated with higher rates of psychiatric comorbidity. Those with a more severe CBD also had lower gross income, were less likely to have an income above the median, and spent a lower percentage of their income on sale items. These results suggest that more severe buying disorders occur in individuals with low incomes who have an impaired ability to control or delay their urges to make inappropriate purchases and who have high rates of psychiatric comorbidity.

Dimensional Traits

In terms of dimensional personality traits, Lejoyeux et al. (1997) reported that depressed compulsive shoppers had higher scores than depressed normal buyers on the experience-seeking subscale of the Zuckerman Sensation Seeking Scale (Zuckerman et al., 1994), as well as the cognitive impulsivity, motor impulsivity, nonplanning activity, and total scores for the Barratt Impulsiveness Scale (Barratt, 1959). O'Guinn and Faber (1989) reported high

levels of compusivity, materialism, and fantasy but lower levels of self-esteem in compulsive buyers compared to normal buyers. Partially consistent with these results, Yurchistan and Johnson (2007) reported that compulsive buying behavior was negatively related to self-esteem and positively related to perceived social status associated with buying, materialism, and apparel-product acquisition. These findings suggest that many compulsive shoppers use possessions to boost their perceived social position, possibly in an attempt to boost low self-esteem.

Summary

Research over the past two decades has contributed to a greater understanding of the epidemiology, phenomenology, and family history of CBD. The disorder is common, associated with important comorbid psychiatric disorders, and can lead to serious functional impairment, financial problems, and legal entanglements. More work is needed to better understand the disorder. First, although several definitions have been proposed, most prominently the criteria of McElroy et al. (1994), their reliability and validity have not been established. Finally, CBD may apparently be chronic or intermittent, but two studies suggest that its course may be modified with treatment. Follow-up studies would be helpful in charting the course of the disorder, tracking its emergence or subsidence, and establishing its relationship to other psychiatric disorders. The issue of gender differences has been little studied. The survey by Koran et al. (2006) suggests that CBD affects men and women equally, yet this conclusion is at odds with the results of other surveys as well as nearly all clinical data. Whether CBD represents a single construct or has multiple subtypes, each with its own etiologies or pathophysiology, is just beginning to be explored. The classification of CDB is unclear, and research has linked it to mood, substance use, obsessive-compulsive, and impulse control disorders. Neurobiological and genetic studies would help clarify these links.

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PART 3

Etiology of Impulse Control Disorders

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Impulsivity and Drug Addiction: A Neurobiological Perspective

T. W. Robbins

Abstract

A conceptual analysis of the impulsivity construct in behavioral and neurobiological terms is followed by an analysis of its causal role in certain forms of drug addiction in both human and animal studies. The main focus of this chapter is on a rat model of impulsivity based on premature responding in the five-choice serial reaction time task and a more detailed characterization of this phenotype in neurobehavioral, neurochemical, and genetic terms. Evidence is surveyed that high impulsivity on this task is associated with the escalation subsequently of cocaine self-administration behavior and also with a tendency toward compulsive cocaine seeking. Novelty reactivity, by contrast, is associated with the enhanced acquisition of self-administration, but not with the escalation of intravenous self-administration of cocaine or the development of compulsive behavior associated with cocaine seeking. These results indicate that the vulnerability to stimulant addiction may depend on different factors, as expressed through distinct presumed endophenotypes. These observations help us further to dissociate various aspects of the impulsivity construct in neural as well as behavioral terms.

Keywords: impulsivity, stimulants, substance use disorders, genetic factors

Introduction

Impulsivity has been defined as the predisposition toward “actions which are poorly conceived, prematurely expressed, unduly risky or inappropriate to the situation and that often result in undesirable consequences” (Durama & Barnes 1993). Close examination of that definition reveals that at least three distinct psychological processes are implicated: motor disinhibition, overzealous motivation to attain a goal, and inadequate reflection or cognitive analysis. Instruments such as the Barratt Impulsivity Scale, designed to capture impulsivity, have recognized this complexity, with a factor structure leading to three subscales of impulsiveness: motor, inattention, and nonplanning.

These considerations are important when analyzing the role of impulsivity as a candidate endophenotype for a variety of neuropsychiatric

disorders, including drug addiction, attention deficit/hyperactivity disorder, antisocial behavior, and mania, as the precise manifestations of impulsivity may be associated with distinct clinical symptoms. Equally, the different facets of impulsivity may reflect altered functioning of different neural systems, including chemical modulatory pathways.

To tap into the different forms of impulsivity in experimental animals, as well as in humans, it is necessary to devise paradigms that isolate its different aspects according to theoretically rationalized principles, a requirement expressly realized originally by Evenden (1999). A number of test procedures are available, whether measuring impulsive choice through the delayed discounting of reward, the capacity to inhibit an initiated response (the stop reaction time test, SSRT; Eagle & Robbins, 2003a; Feola et al., 2000; Logan et al 1984), or the tendency

to respond prematurely on the basis of inadequate information (e.g. Kagan's Matching Familiar Figures task and its analogues; Kagan, 1966).

Dissociable Neural Substrates of Impulsivity

The role of dopamine-dependent functions of the nucleus accumbens in conditioned reinforcement (Taylor & Robbins, 1986) also implicates this structure in processes by which an animal normally mediates delays of reinforcement between response and reward (e.g., food delivery). Thus, the striking effects of lesions to the core subregion of the nucleus accumbens to induce enhanced choice of small, immediate rewards over large, delayed ones (Cardinal et al., 2001) were not unexpected. This profound effect might theoretically have also resulted from deficient "top-down" control by descending influences of the prefrontal cortex and cingulate descending influences. However, lesions of the anterior cingulate and medial prefrontal cortex (mPFC) had no obvious effects on choice impulsivity. Moreover, lesions of the orbitofrontal cortex had the opposite effect, enhancing the choice for the larger, delayed reward in the same paradigm (Winstanley et al., 2004b) although the effects of similar orbitofrontal lesions may lead to impulsive choice under certain conditions (Mobini et al., 2002). However, excitotoxic damage to the basolateral amygdala did produce effects similar to that of the accumbens core (Winstanley et al., 2004b). In terms of chemical neuromodulation, there is evidence of both dopaminergic and serotonergic involvement in a complex interaction between these neurotransmitters. For example, the ability of *d*-amphetamine to reduce delay aversion and produce more shallow discounting functions appears to depend on both dopamine and 5-hydroxytryptamine (5-HT; Winstanley et al., 2003b, 2005). Moreover, both dopamine and 5-HT are released under certain conditions during delayed discounting in different regions of the rat prefrontal cortex, as shown using *in vivo* microdialysis (Winstanley et al., 2006).

By contrast with delayed discounting, lesions of the nucleus accumbens core have no effect on the inhibition of speeded motor actions, as occurs in the SSRT task (Eagle & Robbins, 2003b). Lesions of the dorsomedial striatum as well as those of the orbitofrontal cortex, from which it receives afferent projections, do, however, lengthen the SSRT (Eagle & Robbins, 2003a; Eagle et al., 2008b). Similar lesions of the dorsomedial striatum do not produce choice impulsivity (Eagle, Milton, & Robbins,

unpublished), thus demonstrating a neural dissociation between choice and motor impulsivity.

These are indications that similar dissociations are present in the human literature, especially in terms of structural and functional neuroimaging studies (Aron et al., 2003; Aron & Poldrack, 2006; McClure et al. 2004). The neural control over impulsivity on the SSRT task implicates a fronto-basal ganglia loop that includes the ventrolateral PFC, as well as the subthalamic nucleus. Apart from functional neuroimaging studies (Aron & Poldrack 2006), evidence for this depends on the observation that damage to the right inferior frontal gyrus is especially related to a slowing of the SSRT measure with no consistent effects on Go reaction time (RT) (Aron et al., 2003; see also Aron et al., 2004). In neurochemical terms, Eagle et al. (2008a) have recently reviewed evidence that 5-HT is minimally involved in modulating SSRT in either humans or animals, whereas, as we have seen, it is implicated in the temporal discounting of reward (e.g., Winstanley et al., 2003b). By contrast, the selective noradrenaline reuptake inhibitor atomoxetine does speed the SSRT in both humans and rats (Chamberlain et al., 2006; Robinson et al., 2008b).

The relationship of SSRT to delay aversion was further explored in the clinical context of attention deficit/hyperactivity disorder (ADHD) by Solanto et al. (2001), who found little or no correlation between the two measures, although they exhibited excellent discriminant validity for ADHD when used together. This suggests that different forms of impulsivity, related to impulsive choice and impulsive action, may contribute to distinct forms of the diagnostic spectrum of ADHD.

Impulsivity and Addiction

Much evidence links substance dependence to impulsivity; of course, such impulsivity may further enhance the drive to addiction (Jentsch & Taylor, 1999). There is considerable evidence that addiction or dependence is associated with impulsivity for several drug classes, notably the stimulants, including nicotine, and also alcohol and the opiates (though with varying degrees of support; Caspi et al., 1996; Verdejo-Garcia et al., 2008). But the question remains of whether this impulsivity is a cause of chronic drug abuse or a preexisting condition. It is difficult to establish this from human studies alone, requiring, as it does, an enormous prospective epidemiological study. Verdejo-Garcia et al. review some relevant studies, including, for example, those of young or adolescent children of

substance abusers who themselves are at risk of substance abuse. However, in view of all of the associated factors, it is still difficult to attribute any substance abuse in such offspring simply to a pre-morbid disposition toward impulsive behavior.

Studies using experimental animals clearly could contribute much to the elucidation of a possible role of impulsivity in certain forms of addiction, as it is more feasible to control such critical variables as drug exposure and early environment (Perry et al., 2008). One of the first studies of this relationship examined how well the propensity toward impulsivity, as measured by the steepness of a temporal discounting curve for food reward, predicted subsequent oral alcohol intake (Poulos et al., 1995). Similarly, Perry et al., (2005) showed that steeply discounting female rats acquired intravenous self-administration of cocaine more readily; both of these findings thus supported the hypothesis that impulsivity may precede drug-taking behavior.

Impulsivity in the Five-Choice Serial Reaction Time Task

Neural and Neurochemical Basis

Our own animal studies of the relationship between impulsivity and addiction have utilized a different measure of impulsivity that depends on premature responses on a rodent test of sustained visual attention, termed the *five-choice serial reaction time task* (5-CSRTT). In human terms, this behavior is perhaps analogous to enhanced false alarm responses. In the 5-CSRTT, rodents are trained to detect brief flashes of light presented at random locations in order to earn a food reward. Premature responses in the waiting period prior to visual target presentation are punished by time-out from positive reinforcement (abortion of the trial and extinction of the house lights). Nevertheless, it has been found that there is a considerable propensity for a subgroup of rats, outside the bounds of the normal distribution, to display persistently high levels of impulsive responding (Dalley et al., 2002).

Much is known about the neural and neurochemical basis of this impulsive responding. Psychomotor stimulant drugs such as *d*-amphetamine are known to increase impulsive responding, an effect that depends on dopamine release in the nucleus accumbens (Cole & Robbins, 1987), probably in the shell subregion (Murphy et al., 2008). Lesions of the core or shell subregions do not produce obvious impulsive responding in this test, contrasting (for the core subregion) with the delayed discounting findings of Cardinal et al. (2001).

However, these lesions have *opposite* effects on the impulsive responding induced by systemic *d*-amphetamine, with core lesions actually potentiating the impulsivity and shell lesions reducing it. These data suggest that this form of impulsivity overlaps some of the neural mechanisms associated with steep discounting behavior, notably in the nucleus accumbens, but also shows that there appears to be an opponency between the core and shell subregions in controlling impulsivity induced by dopaminergic agents, a hypothesis further supported by recent findings of effects of a D3 dopamine receptor antagonist, nafadotride, infused directly into the shell or core regions (Besson et al., 2009).

Increases in impulsivity are also associated with global forebrain depletion of 5-HT (Harrison et al., 1997), although recent studies have shown that a 5-HT2A receptor antagonist ameliorates impulsivity, whereas a 5-HT2C antagonist exacerbates it (Fletcher et al., 2007; Robinson et al., 2008a; Winstanley et al., 2004c). Manipulation of certain regions of the medial PFC, such as the infralimbic cortex, also produces impulsivity (Chudasama et al., 2003; Murphy et al., 2005), suggesting that there is also a crucial “top-down” element in the control of this form of impulsive behavior.

Prediction of Binge Intake of Intravenous Cocaine

Dalley et al. (2007) found that high-impulsive rats had a much greater tendency to escalate their cocaine intake when exposed to a long-access, binge schedule of intravenous self-administration. These high-impulsive rats not only exhibited excessive levels of premature responding on the 5-CSRTT, but also elevated these levels considerably when exposed to probe long intertrial intervals. Thus, a relationship between preexisting impulsivity and stimulant self-administration is again confirmed. Additionally, Dalley et al. (2007) found that the high-impulsive rats exhibited diminished levels of binding of the D2/3 ligand¹⁸ F-fallypride in the ventral but not the dorsal striatum. This is a particularly salient finding, which is reminiscent of the seminal findings of Volkow et al. (1993) of similar reductions of striatal D2/3 binding in chronic substance abusers (cocaine, methamphetamine, and alcohol). However, the interpretation of the Volkow et al. data with respect to the etiology of impulsivity and addiction is unclear, because it is plausible to suppose that the change in striatal D2/3 receptors is actually caused by chronic drug exposure.

The findings of Dalley et al. actually suggest that low levels of striatal D2/3 binding may predispose toward enhanced cocaine intake. This is consistent with the impressive data of Morgan et al. (2002), which showed that striatal D2/3 binding predicted subsequent cocaine intake in rhesus monkeys. No clear behavioral phenotype of impulsivity was associated with the reduced striatal binding but these monkeys came from a socially subordinate subgroup, suggesting that social stress may have contributed to their propensity for stimulant intake.

The data of Dalley et al. (2007) raise several pertinent questions: What is the relationship of impulsivity with compulsive drug seeking and addiction, as distinct from substance abuse? What is the psychological basis of this form of impulsivity and its relationship to other forms, such as steep discounting, sensation seeking, and other phenotypes that have been associated with stimulant abuse, as well as with that of other drug classes? What is its precise relationship to the activity of the dopamine system? What is its etiological basis, that is, to what extent is it a genetically inherited trait? How can it be treated, and is such treatment likely to be relevant to effective therapy for drug addiction? The remainder of this chapter considers some of the progress that has been made in addressing these key issues.

Relationship with Compulsive Drug Seeking (Addiction)

A follow-up study (Belin et al., 2008) reveals that the cocaine-seeking and -taking behavior of high-impulsive rats shows many of the characteristics of drug addiction, as defined by DSM-IV criteria. For example, these rats continue to seek cocaine despite the possible adverse consequences of punishing foot shock. In fact, the proportion of high-impulsive rats in the outbred population of male hooded Lister rats corresponds quite well to the proportion that are especially susceptible to compulsive forms of behavior in relation to cocaine. The Belin et al. study also addressed possible overlap between the high-impulsive rats and the high-novelty reactive rats shown to be susceptible to cocaine by Piazza et al. (1989). As in the study of Dalley et al., the most impulsive rats were not also the most active in a novel environment, suggesting a dissociation between these two phenotypes in terms of how they affect cocaine self-administration behavior. Belin et al. found that the high-novelty reactive rats did show some altered characteristics with respect to cocaine self-administration; for example, they showed a vertically shifted dose-response curve for

cocaine self-administration, similar to that described previously by Piazza et al. (1989). They also showed an enhanced tendency to initiate cocaine-self-administration, which was also found by Piazza et al. but was not evident in the high-impulsive rats. Of greatest significance, however, was that the high-novelty reactive rats were not more compulsive than the low-reactive rats; they did not show the increased compulsivity evident in the cocaine self-administration behavior of the high-impulsive rats. These results indicate that the different phenotypes of high impulsivity and novelty reactivity contribute differentially to cocaine self-administration and to compulsive cocaine-seeking behavior.

Psychological Nature of Impulsivity in the 5-CSRTT

This question can be addressed in two ways: analysis of the psychological mechanisms underlying premature responding in the 5CSRTT and comparison with other measures of impulsivity, such as those derived from delayed discounting and the SSRT task. Trivial accounts of impulsivity in terms of such factors as impaired attention and altered motivation appear to be ruled out by consideration of other variables in the 5-CSRTT. Thus, typically, the high-impulsive rats are not typically grossly impaired in terms of attentional accuracy; nor do they show changed latencies to collect earned food pellets. More sophisticated analyses utilize microanalysis of the behavior shown during the 5-CSRTT and manipulations of the parameters governing the task (Mar, Robbins, & Dalley, unpublished observations). These analyses suggest that the premature responding appears to arise in part from excessive motor output. It is not apparently due to deficient timing mechanisms, nor is it due to a failure to learn to inhibit premature responding on the basis of negative feedback. Manipulation of the target duration suggests that the impulsivity is modulated by attentional load, being stronger at shorter target durations.

The other approach to considering the nature of this apparent impulsivity comes from comparisons with other traits such as sensation seeking and anxiety. Animal studies are particularly well suited to this approach. There are grounds for associating the propensity for cocaine abuse to novelty reactivity. A classic study by Piazza et al. (1989) reported that rats that were hyperactive in a novel test situation were quicker to acquire intravenous cocaine self-administration and exhibited vertical shifts in their dose-response curve. The question arises whether

this novelty reactivity tendency is related to the impulsivity dimension. However, two separate lines of evidence argue against this possibility. First, the impulsive rats are not necessarily the most active ones (Dalley et al., 2007). Second, if one segregates the same population of rats separately on the basis of impulsivity and novelty reactivity (as measured again by locomotor activity in a novel environment), the two dimensions show different characteristics with respect to compulsive cocaine-seeking and cocaine-taking behavior. The novelty-reactive rats do not exhibit the same drug-taking phenotype as the impulsive animals. First, they do not show escalation of cocaine self-administration, nor do they exhibit the same compulsive response to drug-seeking when punished with foot shock. Second, unlike the impulsive subgroup but like the novelty-reactive rats of Piazza et al., they do show enhanced acquisition of cocaine self-administration, as well as the same vertical shift in the dose-response curve (Belin et al., 2008).

Recent unpublished data (Dalley et al.) suggest that the high-impulsive rats show a weak tendency toward a reduced anxiety profile, in terms of entries into open arms, on the elevated plus maze; however, this might perhaps be expected given that open arm entries might reflect a disinhibitory tendency. Moreover, although the novelty reactive data suggest that the impulsivity is dissociated from a sensation-seeking construct, this is currently being tested more rigorously using a novelty preference test. A key question about the impulsivity in the 5-CSRTT is its relationship to the temporal discounting of reward, given the implication of the nucleus accumbens in both forms of behavior (Cardinal et al., 2001). In fact, it appears that the high-impulsive rats exhibit significantly steeper discounting functions than the low-impulsive animals (Robinson et al., 2009). These results clearly suggest that the high-impulsive rats share some of the characteristics of steeply discounting animals—a result that is consistent with the findings of Diergaarde et al., (2008) for aspects of nicotine self-administration. These authors directly compared performance on the 5-CSRTT with measures of delayed discounting and found that there were subtle differences in different aspects of the drug-seeking behavior in rats selected for high impulsivity in the two paradigms.

By contrast, when the motor disinhibitory characteristics of the high-impulsive rats were measured using the SSRTT, there were no apparent differences in the SSRT measure between high-impulsive

and-low impulsive rats (Robinson et al., 2009). Given the commonality of the steep discounting with premature responding on the 5-CSRTT, these rats could be characterized as exhibiting an inability to await rewards or other salient signals.

Neural, Neurochemical, and Genetic Bases of Impulsivity on the 5-CSRTT

Preliminary data (see above) indicated that impulsivity on the 5-CSRTT is regulated by a balanced interaction of striatal and prefrontal cortical functions, modulated by dopaminergic and serotonergic influences (Dalley et al., 2002), including the finding of reduced D2/3 receptor binding in the ventral striatum. These analyses have recently been augmented by pharmacological findings. For example, Besson et al. (2009) have shown that the D2/3 receptor antagonist nafadotride, when infused into the nucleus accumbens shell region, induces increased impulsivity on the 5-CSRTT, consistent with the association of reduced D2/3 receptors with impulsivity. However, intriguingly, the same drug infused into the core subregion actually significantly *inhibited* impulsive responding, consistent with an opponency between the core and shell regions in the control of dopamine-dependent impulsivity, as suggested by the findings of Murphy et al. (2008). How this might be related to possible top-down influences of the medial PFC remains to be determined. However, it is significant that the 5-CSRTT impulsivity produced by mPFC lesions is antagonized by infusions of the D2/3 receptor antagonist sulpiride into the nucleus accumbens (Pezze et al., 2009). This suggests that there is indeed a top-down relationship between the mPFC and the nucleus accumbens in the control of this behavior.

It is important to realize that the control of 5-CSRTT impulsivity is not simply subject to dopaminergic modulation. There is evidence for a similar modulation by 5-HT mechanisms, for example, via the 5-HT2 receptor (Dalley et al., 2002; Passetti et al., 2003; Robinson et al., 2008a; Winstanley et al., 2003a). Moreover, there may also be an important noradrenergic influence; the relatively selective noradrenaline reuptake blocker atomoxetine has the remarkable capacity to block impulsive responding in three distinct paradigms: the SSRTT, the delayed discounting paradigm, and the 5-CSRTT (Robinson et al., 2008b). This observation suggests that although these different measures of impulsivity can often be fractionated (see above; see also Winstanley et al., 2004a), the common action of atomoxetine to ameliorate all three forms

suggests some commonality in the way that they are modulated by the ascending catecholamine systems—even though they may be mediated by parallel, relatively segregated frontostriatal “loops.”

The neurochemical and neural specificity of these findings has to be followed up; assuming the modulation is noradrenergic in nature, it is possible that this is exerted at certain restricted striatal sites, specifically the shell subregion, and also at the level of the PFC (Chamberlain et al., 2009).

In general, these findings may be relevant to approaches to the control of impulsivity and investigation of etiological factors—for example, genetic mechanisms. Drugs such as atomoxetine and methylphenidate are used in the control of impulsivity in attention deficit/hyperactivity disorder (juvenile but also adult forms; see, e.g., Chamberlain et al., 2007). However, the relative contributions of dopaminergic and noradrenergic mechanisms in the therapeutic actions of these compounds are still unclear.

There is, of course, considerable evidence of D2 receptor involvement in addiction, particularly in relation to stimulant drugs (Foll et al., 2009). However, the possible role of the D3 receptor is less clear. Increasingly, however, it appears likely that traits as complex as impulsivity will be found to be related to changes in many genes, and that is the current focus of the 5-CSRTT high-impulsivity phenotype. Selective breeding of the high-impulsivity phenotype is showing that the baseline level of about 8% in the normal outbred Lister strain does amplify significantly in subsequent generation in both male and female subjects (Dalley et al., unpublished). Currently planned studies are focusing on gene expression and a genomewide association analysis for this trait. However, it also appears likely that environmental factors play a role, and the effects of early experience are also being investigated.

Conclusions

Impulsivity has long been associated with human drug addiction, but its etiological significance has remained unclear. Animal studies may have an important role in resolving issues of cause and effect. Moreover, they are better able to investigate neurobehavioral concomitants of impulsivity. This chapter has illustrated these principles from the recent literature, necessarily focusing on our own recent work. The impulsivity construct itself is complex in humans, but in rodents, it also appears likely that it can be fractionated, although there are apparently some elements in common. This may relate to psychologically distinct forms of impulsivity—for

example, impulsive choice versus impulsive action. We have found an association between one aspect of performance on the 5-CSRTT in rats, namely, premature responding, the propensity to escalate cocaine intake, and for this to become compulsive in nature. Consequently, much of our subsequent neurobehavioral analysis has focused on better understanding the psychological and neural basis of this phenotype. More detailed analysis of this behavior relates it to an inability to wait for anticipated reinforcement, associated with a dysregulation of the ventral striatum. One of many important residual questions is how this form of impulsivity relates to better-characterized theoretical models and also to human aspects of impulsivity. We anticipate that this work with animals will influence our search for the nature of impulsivity as it relates to addiction in human subjects.

Acknowledgments

I thank those colleagues who contributed to this work. The research was supported by grants from the Medical Research Council and the Wellcome Trust. This chapter is based on a plenary lecture given at the International Society for Research on Impulsivity (ISRI) meeting, San Diego, California, June 20, 2009.

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The Genetics of Impulsivity

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Abstract

Impulsivity is a complex trait that varies across healthy individuals, although when excessive, it is generally regarded as dysfunctional. Impulsive behavior may lead to initiation of drug addiction that interferes with inhibitory controls, which may in turn result in facilitation of the individual's impulsive acts. Although environmental factors play a considerable role in impulsive behavior, a body of evidence collected in twin studies suggests that about 45% of the variance in impulsivity is accounted for by genetic factors. Genetic variants studied in association with impulsivity include those for *tryptophan hydroxylase 1* and *2* (*TPH1* and *TPH2*), the *serotonin transporter* (*SERT*), serotonin receptors, and genes of the monoamine metabolism pathway (e.g., *monoamine oxidase A*, *MAOA*). Other systems may also play a role in these behaviors, such as the dopaminergic system (the dopamine receptors *DRD2*, *DRD3*, and *DRD4*, and the *dopamine transporter*, *DAT*), the catecholaminergic system (*catechol-O-methyltransferase*, *COMT*), and the GABAergic system (*GABA receptor subunit alpha-1*, *GABRA1*; *GABA receptor subunit alpha-6*, *GABRA6*; and *GABA receptor subunit beta-1*, *GABRB1*). Taking into account involvement of the hypothalamic-pituitary-adrenal (HPA) axis, the number of candidate genes implicated in impulsivity may be increased significantly and, therefore, may go far beyond those of serotonergic and dopaminergic systems. For a number of years, our group has conducted studies of the association of genes involved in the modulation of the stress-responsive HPA axis and several neurotransmitter systems, all involved in the pathophysiology of anxiety and depressive disorders, impulse control and compulsive disorders, with drug addiction. These genes include those of the opioid system: the *mu-* and *kappa-opioid receptors* (*OPRM1* and *OPRK1*) and the *nociceptin/orphaninFQ receptor* (*OPRL1*); the serotonergic system: *TPH1* and *TPH2* and the serotonin receptor *1B* (*5THR1B*); the catecholamine system: *COMT*; the HPA axis: *themelanocortin receptor type 2* (*MC2R* or *adrenocorticotropic hormone*, *ACTHR*); and the cannabinoid system: the *cannabinoid receptor type 1* (*CNR1*). In this chapter we will focus on these findings.

Keywords: impulsivity, genetic variants, polymorphisms, SNPs

Introduction

Impulsivity is a complex trait that varies across healthy populations (e.g., Patton et al., 1995), although it is generally regarded as a dysfunctional trait that might result in criminal, violent, or dangerous behaviors. Studies have shown an association of impulsivity with behavioral compulsive disorders including problem and pathological gambling (e.g., Chiu & Storm, 2009; Ledgerwood et al., 2009), a variety of eating disorders (e.g., Waxman, 2009),

and pathological high-risk sexual behavior (Seal & Agostinelli, 1994) that is commonly a predictor of sexually-transmitted diseases including acquired immune deficiency syndrome (AIDS). Impulsive behavior may also lead to initiation of drug addiction (for review, see Kreek et al., 2005b). Impulsivity often refers to *disinhibition*, meaning that the cognitive control that suppresses automatic or reward-driven responses is lost (Aron, 2007). Substance abuse and addiction may interfere with these

inhibitory controls, resulting, in turn, in facilitation of the individual's impulsive acts (for review, see Verdejo-Garcia et al., 2008).

Impulsivity has been defined as a "predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions" (Moeller et al., 2001, p. 1784). This impaired information processing occurs when there is no conscious deliberation (Barratt et al., 1997). Impulsive behavior is a major component of mental disorders including drug addiction (Bickel et al., 1999; Moeller et al., 2004; Nagoshi et al., 1991) and suicidality (e.g., Giegling et al., 2009; Neufeld & O'Rourke, 2009).

Several inventories have been developed to measure impulsivity. The most common are the Barratt Impulsiveness Scale, version BIS-11 (Patton et al., 1995; Stanford et al., 2009) and the Sensation Seeking Scale (SSS; Zuckerman et al., 1964, 1978). The BIS-11 is a 30-question self-report instrument that uses three subscales based on reviews of factor analytic studies to measure impulsivity. These are the Nonplanning (intolerance of cognitive complexity and lack of self-control), Cognitive (cognitive instability and inattention), and Motor (lack of perseverance and motor impulsiveness) subscales, which, when combined, form the total composite score. The SSS is a 40-question self-report instrument that was designed to measure a subject's level of stimulation or arousal. It has four subscales based on factor analysis: Thrill and Adventure Seeking (TAS), Disinhibition (DIS), Experience Seeking (ES), and Boredom Susceptibility (BS).

Other, more general scales have been used to classify and describe personality dimensions and to characterize the role of genetics in personality. Frequently used personality scales are the NEO Personality Inventory-Revised (NEO-PI-R), which measures openness, agreeableness, neuroticism, conscientiousness, and extroversion (Costa & McCrae, 1992), and the Tridimensional Personality Questionnaire (TPQ; Cloninger, 1987) or the more complete version thereof, the Temperament and Character Inventory (TCI), which measures novelty seeking, reward dependence, harm avoidance, and persistence (Cloninger et al., 1993).

Pioneering studies demonstrated that low levels of serotonin and metabolites of serotonin are associated with impulsive behaviors. 5-Hydroxyindolacetic acid (5-HIAA), the major metabolite of serotonin, when assayed in the cerebrospinal fluid (CSF), is a measure of serotonin metabolism. Low levels of CSF 5-HIAA were first found to be associated with

alcoholism in depressed patients with a family history of alcoholism (Rosenthal et al., 1980). Studies demonstrated that subjects with low CSF 5-HIAA had deficits in impulse control (Soubrie, 1986), a higher incidence of impulsive crimes (Linnoila et al., 1983), and aggression (Brown et al., 1979; Roy et al., 1988). Following fenfluramine challenge, low serotonin metabolism was found to be related to impulsive behavior and an increase in the risk for impulsivity in family members (Coccaro et al., 1994). Serotonergic neurons from the raphe innervate the frontal cortex, amygdala, and hippocampus. Reduced impulse control may result from impaired inhibitory control of the frontal cortex related to decreases in serotonin.

Although environmental factors play a considerable role in impulsive behavior, a large body of evidence collected in twin studies suggests that about 45% of the variance in impulsivity is accounted for by genetic factors (Hur & Bouchard, 1997; Pedersen et al., 1988; Seroczynski et al., 1999). Historically, genes of serotonergic and dopaminergic systems that are involved in mediation of neural transmission have been a target for studies of the molecular genetics of impulsivity (reviewed in Carver & Miller, 2006; Congdon & Canli, 2008). It has been shown that genes coding for products of the serotonergic system have been found to be associated with impulse control disorders. Psychostimulant drugs targeting dopaminergic systems have been shown to be effective in the treatment of the symptoms of attention deficit hyperactivity disorder. Many of the genes associated with impaired impulse control also have been identified as being involved in the vulnerability to develop drug addiction. Early in the development of drug and alcohol addiction, genes that play a role in impulsive behaviors also may have a significant role in the initiation of drug use, the initial step to addiction.

Genetic variants studied in association with impulsivity (reviewed in Carver & Miller, 2006; Congdon & Canli, 2008; Hur & Bouchard, 1997; Pedersen et al., 1988; Seroczynski et al., 1999; Verdejo-Garcia et al., 2008) include those for *tryptophan hydroxylase 1* and *2* (*TPH1* and *TPH2*), the *serotonin transporter* (*SERT*), serotonin receptors, and genes of the monoamine metabolism pathway (e.g., *monoamine oxidase A*, *MAOA*). Other systems may also play a role in these behaviors, such as the dopaminergic system (the dopamine receptors *DRD2*, *DRD3*, and *DRD4*, and the dopamine transporter, *DAT*), the catecholaminergic system (*catechol-O-methyltransferase*, *COMT*), and

the GABAergic system (*GABA receptor subunit alpha-1*, *GABRA1*; *GABA receptorsubunit alpha-6*, *GABRA6*; and *GABA receptor subunit beta-1*, *GABRB1*).

Both serotonergic and dopaminergic systems play an important role in the regulation of the hypothalamic-pituitary-adrenal (HPA) axis. Dysregulation of the HPA axis, in turn, was found to be associated with a number of physical and psychological conditions including impulsivity (e.g., Hong et al., 2003), posttraumatic stress disorder (e.g., Yehuda, 2001), major depression, and specific stressors (e.g., Holsboer & Barden, 1996). Taking into account involvement of the HPA axis, the number of candidate genes implicated in impulsivity may be increased significantly and, therefore, may go far beyond serotonergic and dopaminergic systems. For example, our studies have shown that the mu-opioid receptor is involved in modulation of the HPA axis in humans through tonic inhibition (Bart et al., 2005b; Schluger et al., 1998).

For a number of years, our group has conducted studies of the association of genes that are involved in the modulation of the stress-responsive HPA axis and several neurotransmitter systems, all involved in the pathophysiology of anxiety and depressive disorders, impulse control and compulsive disorders, with drug addiction. These genes include those of the opioid system: the *mu-* and *kappa-opioid receptors* (*OPRM1* and *OPRK1*) and the *nociceptin/orphanin FQ receptor* (*OPRL1*); the serotonin system: *TPH1* and *TPH2* and the *serotonin receptor 1B* (*5THR1B*); the catecholamine system: *COMT*; the HPA axis: *melanocortin receptor type 2* (*MC2R* or *adrenocorticotropic hormone*, *ACTHR*); and the cannabinoid system: the *cannabinoid receptor type 1* (*CNR1*). Using 10K and 100K GeneChips from Affymetrix, we performed whole genome association scans to identify other genes that might be found to be associated with specific addictive disorders. In this review, we will discuss studies of these genes.

The Opioid System

The HPA axis is a key system involved in stress regulation. Studies of our group had shown that the mu-opioid receptor is involved in modulation of the HPA axis in humans by tonic inhibition (Bart et al., 2005b; Schluger et al., 1998). Many studies have shown the involvement of this G-protein-coupled receptor in stress responsivity (e.g., Koob & Kreek, 2007; Kreek & Koob, 1998). In animal models, the mu-opioid receptor gene *Oprm1* gene knockout

mice showed a reduced emotional response to stress compared to wild-type mice (e.g., Ide et al., 2010). There were interesting results in recent animal studies in *Oprm1* knockout and *delta-opioid receptor* (*Oprd1*) knockout mice on motor impulsivity and associative learning in the signaled nose poke task (Olmstead et al., 2009). *Oprm1* knockout mice displayed a significant decrease in motor impulsivity and were insensitive to ethanol, while *Oprd1* knockout mice displayed more impulsive behavior than controls. Both *Oprm1* and *Oprd1* knockout mice showed no deficit in associative learning.

Mu-opioid Receptor Gene (OPRM1)

Polymorphisms of *OPRM1* have been studied in association with various neuropsychiatric conditions including major depression, schizophrenia, substance abuse and dependence, and pain sensitivity (reviewed in Kreek et al., 2005b). One of the common functional polymorphisms of the *OPRM1* gene is A118G (rs1799971) that encodes for a substitution of aspartic acid for an asparagine Asn40Asp (Bergen et al., 1997; Bond et al., 1998). This substitution removes an N-glycosylation site from the N-terminal extracellular domain of the receptor. In vitro studies have shown that the Asp40 (118G) isoform selectively binds beta-endorphin, an endogenous ligand of the mu-opioid receptor, with approximately three times greater affinity compared to the prototype receptor and threefold increased activation of the G-protein-activated inwardly rectifying K⁺ channels (GIRKs; Bond et al., 1998). These data suggest that individuals having at least one copy of the 118G allele of *OPRM1* may have a physiological difference in response to medications directed toward *OPRM1*.

In cell studies, variants of the receptor had lower forskolin-induced cyclic adenosine monophosphate (cAMP) accumulation and lower receptor binding site availability (Kroslak et al., 2007). In human postmortem brain, the 118G variant of the receptor was shown to have reduced agonist-induced receptor signaling efficacy but not binding (Oertel et al., 2009). In postmortem brain, the 118A allele of the *OPRM1* was reported to be expressed at higher levels than the 118G allele, resulting in an allelic imbalance of the receptor that may cause increased receptor density and function (Zhang et al., 2005).

Several studies have reported an association between the 118G variant and opioid dependence, as well as other substance dependencies (Bart et al., 2004; Deb et al., 2009; Kapur et al., 2007; Kreek et al., 2005a). In a Swedish cohort, the 118G variant

was found to be associated with alcoholism (Bart et al., 2005a) and, in two studies, with a positive pharmacotherapeutic response to naltrexone treatment for alcoholism (Anton et al., 2008; Oslin et al., 2003). The variants located in intron 1 of *OPRM1*, including IVS1+7578A>G (rs511435), IVS1+14123A>C (rs524731), IVS1+18183A>G (rs3823010), IVS1+21573C>T (rs495491), and IVS1+43344A>G (rs3778156), were found to be associated with opioid dependence in European Americans (Zhang et al., 2006); other SNPs of intron 1 of *OPRM1*, including IVS1+32289C>T (rs1381376) and IVS1+43344A>G, were found to be associated with a positive response to heroin after their first use in Chinese (Zhang et al., 2007).

Kappa-Opioid Receptor Gene (*OPRK1*)

Localized in several areas of the dopaminergic nigrostriatal and mesolimbic-mesocortical systems, the kappa-opioid receptor and its endogenous ligand dynorphin play an important role in modulation of rewarding stimuli through modulation of basal and drug-induced dopaminergic tone (Kreek et al., 2002). Dynorphin peptides decrease basal and drug-induced dopamine levels in these systems. Therefore, the kappa-opioid receptor/dynorphin system may be considered to be part of the counter-modulatory mechanisms of the brain after direct or indirect drug-induced dopaminergic stimulation (Kreek et al., 2005a).

In studies in our laboratory, we have redefined the exon-intron structure of the human *OPRK1* gene and have shown that the *OPRK1* gene has four major exons, three introns, and a 3' untranslated region (UTR) of 3096 nucleotides (Yuferov et al., 2004). This is similar to the structure of rodent *Oprk1* genes. We performed a test for association of a number of variants of this gene with vulnerability to develop heroin addiction. For this purpose, 12 single nucleotide polymorphisms (SNPs) located in the coding region, as well as in intron 1 of this gene, were chosen. A pointwise significant association of the synonymous substitution 36G>T (rs1051660) in exon 2 with vulnerability to develop heroin addiction was found using logistic regression analysis with all three ethnic groups combined (African Americans, Caucasians, and Hispanics). In Hispanics, a haplotype of eight SNPs of *OPRK1* was found to be associated with heroin addiction. The results of our reported association of the *OPRK1* variant rs1051660 with heroin addiction were replicated in a study of a European American cohort (Gerra et al., 2007). This variant was not found to

be associated with alcohol and cocaine dependence (Zhang et al., 2008). A haplotype consisting of seven SNPs including the variant 36G>T was found to be associated with alcohol dependence (Zhang et al., 2008). A study of European Americans found four polymorphisms in intron 2 of the *OPRK1*, but not 36G>T, to be associated with alcohol dependence (Xuei et al., 2006). Another study has shown an association of the 830 bp insertion located 1389 nucleotides upstream from the transcription start site with alcohol dependence also in European Americans (Edenberg et al., 2008).

Nociceptin/Orphanin FQ Receptor Gene (*OPRL1*)

A constituent of the endogenous opioid system is the nociceptin/orphanin FQ receptor (NOP-r) that is encoded by the *OPRL1* gene. NOP-r plays a role in regulating behavioral responses and tolerance to morphine through its interaction with its ligand nociceptin/orphanin FQ (N/OFQ). Both N/OFQ and NOP-r are expressed at various levels throughout the brain. N/OFQ inhibits the release of dopamine from neurons in the ventral tegmental area, possibly by increasing GABA levels, which then decreases dopamine signaling to the nucleus accumbens (Murphy & Maidment, 1999). When N/OFQ is injected into mice, morphine place preference is attenuated (Murphy et al., 1999) and there is an inhibition of mesolimbic dopamine release (Murphy et al., 1999). This may partially explain why N/OFQ has been found to inhibit the rewarding properties of addictive drugs (reviewed in Briant et al., 2010). In mice with the *Oprl1* gene deleted, the development of morphine tolerance was reduced by half (Ueda et al., 1997). Hence, it appears that N/OFQ and NOP-r block part of the rewarding properties of opiates and contribute to the development of tolerance to opiates.

We examined five common genetic variants in the *OPRL1* gene for their association with vulnerability to develop opiate addiction in three ethnicities: Caucasian, African American, and Hispanic (Briant et al., 2010). In the Caucasian group, two of the variants in *OPRL1*, IVS2+971G>A (rs6090041) and IVS2+6225C>T (rs6090043), were found to be associated with vulnerability to develop heroin addiction with pointwise significance. Furthermore, the AT haplotype formed by these variants was found to be associated with protection from developing heroin addiction in both African Americans and Caucasians, and the CG haplotype was found to be associated with vulnerability only in the Caucasians. It was

hypothesized that the alteration could be due to a disruption of an AP-2 binding site by the IVS2+971G>A variant, and the CREB and AP-1 binding sites by the IVS2+6225C>T variant, thereby altering the transcriptional regulation of *OPRL1*. In a similar study, the variant IVS2+6225C>T, and another variant, IVS2+4565G>A (rs6512305), were found to be associated with opiate addiction (Xuei et al., 2008).

The Serotonin System

The biosynthesis of serotonin is controlled by the rate-limiting enzyme tryptophan hydroxylase (Cooper & Melcer, 1961). Since this enzyme controls the biosynthesis of serotonin, we hypothesized that variants in the gene coding for this enzyme may be found to be associated with impulse control disorders such as drug addiction and alcoholism. Since a reduced level of serotonin is known to be central in impulse control disorders, genes that code for components in serotonin biosynthesis may also be involved.

Tryptophan Hydroxylase 1 and 2 Genes (TPH1 and TPH2)

In early studies on the role of the *TPH* gene, which is now known as *TPH1*, we demonstrated a relationship of *TPH1* gene variants with suicidal behavior and CSF 5-HIAA concentrations in Finnish violent offenders (Nielsen et al., 1994). Later we found, in a large Finnish pedigree study, that this same *TPH1* variant was linked with vulnerability to develop alcoholism (Nielsen et al., 1998). Therefore, we investigated these two genes, *TPH1* and *TPH2*, that code for isoforms of tryptophan hydroxylase, the enzymes catalyzing the rate-limiting steps in the production of serotonin. We resequenced the *TPH2* gene in the 5' upstream region, all 11 exons, and in the 3' downstream region in 185 subjects (Nielsen et al., 2008a). Fourteen previously identified and 23 novel variants were found. Three variants of *TPH2* and one of *TPH1* varied in allele frequency among the Caucasian, African American, and Hispanic populations. When loci of two different genes, *TPH1* and *TPH2* were examined using one *TPH1* variant and six common *TPH2* variants, both the *TPH2* variants IVS5+22751C>T (rs7963720) and the synonymous variant 1125A>T (rs4290270) from intron 10 were found to interact in Hispanics with the *TPH1* IVS7+779C>A (rs1799913) variant and were found to be associated with the development of heroin addiction. Additionally, a *TPH2* haplotype in

African Americans was found to be associated with heroin addiction.

Serotonin Receptor 1B Gene (HTR1B)

The 5-hydroxytryptamine (serotonin)-1B receptor (*HTR1B*) is a G-protein-coupled receptor that is involved in many neuropsychiatric and physiological functions including thermoregulation, locomotion, and feeding (e.g., Barnes & Sharp, 1999). Serotonin receptor 1B knockout mice showed decreased anxiety (Zhuang et al., 1999) and increased spatial memory performance (Malleret et al., 1999), impulsive aggression (Brunner & Hen, 1997; Saudou et al., 1994; Zhuang et al., 1999), and exploratory activity (Malleret et al., 1999).

Gene expression studies have found that the -161T (rs130058) allele is expressed at consistently higher levels compared to -161A (Sun et al., 2002). The haplotype-261G (rs11568817)/-161A enhances transcriptional activity 2.3-fold compared to the haplotype-261T/-161A (Duan et al., 2003). A study of micro-RNA-directed silencing has shown an allele-specific interaction of the brain-expressed microRNA miR-96 with a region containing the SNP 1997G>A (rs13212041), located in the 3' UTR of the *HTR1B* gene (Jensen et al., 2009). Allele A of rs13212041 strongly repressed the expression of the *HTR1B* gene. An increase in conductdisorder behaviors was found in individuals homozygous for the A allele of rs13212041 compared to those having GA or GG genotypes. The synonymous 861G>C (rs6296) polymorphism has been found to be associated with a history of substance abuse and a major depressive episode, but not with bipolar disorder, schizophrenia, or alcoholism in mixed ethnicities (Huang et al., 2003). The 861G>C polymorphism was found to be associated with suicide attempts in a group of patients with personality disorders (New et al., 2001). The -161A>T polymorphism was found to be associated with alcohol dependence in Han Taiwanese (Huang et al., 2003a). In our study, no association of the *HTR1B* polymorphisms -261T>G, 129C>T (rs6298), or 861G>C was found with cocaine abuse and dependence or with alcohol abuse and dependence (Cigler et al., 2001). In another study, we tested for association of a number of SNPs of the *HTR1B*, including T-261G, A-161T, C129T, G861C, and A1180G (rs6297), with heroin addiction. We also tested molecular haplotypes and statistically inferred haplotypes for association with heroin addiction (Proudnikov et al., 2006). Tests were performed separately for African Americans, Caucasians, and Hispanics. Minor allele

1180G and the statistically inferred haplotype TACGG comprising 1180G were found to be associated with protection from heroin addiction in Caucasians.

Catecholamine-O-Methyltransferase Gene (COMT)

COMT is an enzyme that degrades dopamine and other catecholamines and, therefore, is important for monoamine signaling. *COMT* has been found in both peripheral and central tissues (Mannisto & Kaakkola, 1999) and plays the major role in biotransformation of estrogens (e.g., Freedman et al., 2009; Zahid et al., 2007). *COMT* SNPs have been tested for association with various neuropsychiatric conditions including suicide, major depression, bipolar disorder, schizophrenia, panic disorder, obsessive-compulsive disorder, and ADHD, as well as for the efficacy of response to treatment of Parkinson's disease (reviewed in Oosterhuis et al., 2008).

The most frequently studied functional variant of the *COMT* 472G>A (rs4680), which is located in exon 4 and codes for a valine to methionine substitution at codon 158 (Val158Met), results in a fourfold decrease of enzymatic activity (Lachman et al., 1996; Lotta et al., 1995; Chen et al., 2004). In a study of heterozygous human lymphoblast cell lines and brains, the 472G allele of *COMT* was found to be overexpressed compared to the 472A allele (Zhu et al., 2004). Common haplotypes of the human *COMT* gene, consisting of a polymorphism from the noncoding region IVS2+1140A>G (rs6269), synonymous polymorphisms 186C>T (rs4633) and 408C>G (rs4818) from exons 3 and 4, respectively, and the 472G>A variant influence the stem-loop structure and folding potential of *COMT* mRNA (Nackley et al., 2006). Among the haplotypes studied, the GCGG haplotype showed the highest enzymatic activity and protein levels, while the ACCG haplotype showed the lowest. Another study showed an association of these *COMT* haplotypes with hyperactivity/impulsivity in adults using the ADHD Self-Report Scale (ASRS; Halleland et al., 2009). In individual tests, the strongest association with hyperactivity/impulsivity was found with the IVS2+1140A>G variant. In analysis of candidate genes for ADHD, the *COMT* 472G>A variant was found to be strongly associated with inattention and hyperactivity/impulsivity, and the 102T>C variant of *5-hydroxytryptamine receptor 2* (*HTR2A*) gene was found to be associated with hyperactivity/impulsivity, but not with inattention

(Reuter et al., 2006). These two genes together accounted for 5.8% of the variance in scores of the ASRS. The number of 158Met alleles (one vs. two) was found to be associated with the ability to experience reward in daily life (Wichers et al., 2008).

Amphetamine administration to individuals homozygous for the 472G allele enhances prefrontal functioning during a working memory task, as shown using functional magnetic resonance imaging (fMRI); this was not found in individuals homozygous for the 472A allele (Mattay et al., 2003). In Caucasians, the high-activity 472G allele was found to be associated with polysubstance abuse (Vandenbergh et al., 1997) and heroin addiction (Horowitz et al., 2000). The 472G allele was found to be associated with heroin addiction in Chinese (Cao et al., 2003) and abuse of methamphetamine in Han Chinese (Li et al., 2004). In Caucasian amphetamine abusers, the 472A allele was found to be associated with novelty seeking (Hosak et al., 2006). Different specific haplotypes of *COMT* were found to be associated with nicotine dependence in African Americans and Caucasians (Beuten et al., 2006) and with cocaine dependence in African Americans (Lohoff et al., 2008). In studies of human postmortem brain, levels of proenkephalin in the nucleus accumbens were found to be correlated with the *COMT* 472G>A genotype in heroin abusers (Nikoshkov et al., 2008). A study of dopamine transporter-*COMT* gene-gene interactions (Yacubian et al., 2007) showed that subjects homozygous for the 472A allele had greater responses in prefrontal and ventral striatum activities in anticipation of reward than did subjects homozygous for the 472G allele. Recent analyses of the 172 kilobase region surrounding the *COMT* gene in DNA samples collected from 45 populations revealed haplotypes that may harbor functional consequences (Mukherjee et al., 2010).

Some studies revealed gender-specific effects. For example, in one study, the 472A allele was found to be associated with obsessive-compulsive disorder in males but not in females (Karayiorgou et al., 1999). In another study, the 472G allele was found to be associated with alcoholism in American Indian females but not males (Enoch et al., 2006). *COMT* homozygous knockout female, but not male, mice developed increased anxiety in a light/dark model; in male mice only, increased aggressive behavior was found in *COMT* heterozygous knockouts compared to other genotypes (Gogos et al., 1998). In vitro cellular studies, physiological concentrations of 17-beta-estradiol were shown to down-regulate

COMT gene transcription and protein expression (Jiang et al., 2003; Xie et al., 1999).

In one study, our group tested a number of polymorphisms in exon 4 of *COMT* for association with heroin addiction in three ethnicities, Caucasians, Hispanics, and African Americans (Oosterhuis et al., 2008). In gender-stratified data, we found an association of G/A and A/A genotypes of 472G>A with heroin addiction in females but not males.

Melanocortin Receptor Type 2 Gene (MC2R or Adrenocorticotropic Hormone Receptor, ACTHR)

Adrenocorticotropic hormone (ACTH) is derived from the anterior pituitary peptide proopiomelanocortin (POMC); this peptide regulates adrenal glucocorticoid and androgen synthesis in the zona fasciculata and reticularis of the adrenal cortex. Adrenocorticotropic hormone binds to its specific receptor, the melanocortin receptor type 2 (MC2R), also called the ACTH receptor (Mountjoy et al., 1992). The MC2R belongs to the family of G-protein-coupled membrane receptors and is involved in the regulation of adrenal cortisol secretion, which is important in stress responsivity.

Genetics studies revealed a number of SNPs in the *MC2R* gene found to be associated with familial glucocorticoid deficiency (e.g., Clark et al., 1993; Elias et al., 1999; Tsigos, et al., 1993). Substitution of A to G in SNP -179A>G (also called -2T>C) was found to be associated with an impaired cortisol response to ACTH stimulation in vivo and results in lower promoter activity in vitro (Slawik et al., 2004). In healthy volunteers, homozygous AA individuals had a significantly higher dehydroepiandrosterone (DHEA) response than homozygous GG individuals in an ACTH stimulation test, while baseline DHEA concentrations did not differ between the groups (Slawik et al., 2004).

In one study, we searched for novel polymorphisms in the coding region of the *MC2R* gene by resequencing a group of 272 subjects of three different ethnicities (Caucasians, African Americans, and Hispanics) with approximately equal numbers of former heroin addicts and normal volunteers (Proudnikov et al., 2008). We tested a series of individual SNPs and statistically inferred haplotypes of the *MC2R* gene were found to be associated with vulnerability to develop heroin addiction. An experimentwise significant association of the A allele of -184G>A (rs2186944) and the haplotype AACT, consisting of -184G>A, -179A>G (both in the promoter region), 833A>C (rs28926182) coding

for Phe278Cys substitution, and 1005C>T (rs4797824), with a protective effect from the development of heroin addiction was found in Hispanics. The -179A>G variant that had been found to be associated with an impaired cortisol response to ACTH stimulation in German subjects (Slawik et al., 2004) was not found to be associated with heroin addiction in any ethnic group in our study. In that study of German subjects, the allelic frequency of the G allele was 0.10 (Slawik et al., 2004). We found a frequency of 0.09 in Caucasian controls and 0.12 in Hispanic controls (Proudnikov et al., 2008). The frequency of this polymorphism in the African American control group was considerably lower. In our studies, the allelic frequency of A in -184G>A of cases in Caucasians was 0.01, in African Americans it was 0.13, and in Hispanic control groups it was 0.17.

The Endocannabinoid System: The Cannabinoid Receptor 1 (CNR1) and Fatty Acid Amide Hydrolase (FAAH) Genes

The endocannabinoid system is important in mediating and modulating neurophysiological responses to drugs of abuse. The cannabinoid receptor type 1 (CNR1) is expressed in brain regions important for drug reward (Herkenham et al., 1990, 1991; Tsou et al., 1998). The other type of cannabinoid receptor, CNR2, has been found in immune cells, the spleen (Munro et al., 1993), and brainstem neurons (Van Sickle et al., 2005). The principal psychoactive component of marijuana, Δ^9 -tetrahydrocannabinol, is an agonist of both CNR1 and CNR2 (Howlett et al., 2002). N-arachidonoyl ethanolamine (anandamide; Devane et al., 1992) and 2-arachidonoylglycerol (Sugiura et al., 1995) are endogenous ligands of CNR1. These two ligands are subject to hydrolysis by the fatty acid amide hydrolase (FAAH; Deutsch et al., 2002; McKinney & Cravatt, 2005). In *FAAH* knockout mice, endogenous brain levels of anandamide and other amides of fatty acids were found to be increased up to 15-fold (Cravatt et al., 2001). In humans, the 385C>A (rs324420) coding for Pro129Thr missense substitution in *FAAH* results in increased sensitivity of this enzyme to proteolytic degradation (Sipe et al., 2002) and reduced *FAAH* expression and activity measured in lymphocytes in human studies (Chiang et al., 2004). The variant 385C>A of *FAAH* has been reported as a risk factor for "problem drug use" and "street drug use," but not alcohol or tobacco use or dependence, in Caucasians (Flanagan et al., 2006; Sipe et al., 2002). Also, in Caucasians, individuals with the

385AA genotype were found to be at reduced risk for cannabis dependence (Tyndale et al., 2007).

Several polymorphic sites, including the synonymous 1359G>A (rs1049353) in the coding region and the triplet polymorphism 18087–18131(TAA)_{8–17} (rs59269177) in the 3' UTR, have been identified in the *CNR1* gene (e.g., Zhang et al., 2004). In Caucasians, the variant 1359G>A was found to be associated with severe alcohol withdrawal (Schmidt et al., 2002). In French Caucasians, a lower representation of the 1359GG genotype in non-substance-abusing schizophrenia patients, compared to substance-abusing patients, was found (Leroy et al., 2001). In a French cohort, no association of 1359G>A with schizophrenia was found (Hamdani et al., 2008), although G allele frequency was found to be significantly higher in patients who were non-responsive to antipsychotics. In our studies, we found an association of 1359G>A with a protective effect against developing heroin addiction in Caucasians (Proudnikov et al., 2009).

The triplet polymorphism 18087–18131(TAA)_{8–17} was shown to confer susceptibility to schizophrenia, especially of the hebephrenic type, in a Japanese cohort (Ujike et al., 2002). This repeat polymorphism has been reported to be associated with intravenous injection of diverse drugs in Caucasians (Comings et al., 1997) and with cocaine dependence in an Afro-Caribbean population of Martinique island (Ballon et al., 2006) but not with heroin abuse in Han Chinese (Li et al., 2000). Variants –22959C>T (rs2180619), –6274T>A (rs806379), and –5489A>G (rs2023239) were found to be associated with polysubstance abuse in European Americans; SNPs –6274T>A, –6215A>C (rs1535255), and –5489A>G were found to be associated with polysubstance abuse in African Americans (Zhang et al., 2004). Three of these variants, –6274T>A, –6215A>C, and –5489A>G, were found to be associated with substance dependence in both European American and African American subjects (Herman et al., 2006). Variants –39884C>G (rs1884830), –17937T>G (rs6454674), and 4894T>C (rs806368) were found to be associated with drug dependence at the allele level and –39884C>G at the genotype level in European Americans (Zuo et al., 2007). The same study found associations of –39884C>G, –17937T>G, –6274T>A, and 4894T>C SNPs with comorbid drug and alcohol dependence at the allele level in European Americans.

In our study, we found that the results of allele tests of 18087–18131(TAA)_{8–17} in African American

and Caucasian groups were consistent with each other: in separate analyses for each type of the repeat number, the long allele was found to be associated with vulnerability to develop heroin addiction in African Americans, while the short allele was found to be associated with a protective effect against developing heroin addiction in Caucasians (Proudnikov et al., 2009). Association of the long repeats with vulnerability to develop heroin addiction was consistent in Caucasians when all short repeats grouped were compared to all long repeats (Proudnikov et al., 2009). These findings are consistent with the results of an association of 18087–18131(TAA)_{8–17} with polysubstance abuse in European Americans and African Americans (Zhang et al., 2004).

A study of *CNR1* mRNA expression revealed novel exonic sequences, novel splice variants, and novel polymorphisms of *CNR1*. Specific splice variants were found to be expressed differentially in different brain regions: caudate, substantianigra, and amygdala express a large amount of the splice variant E of the *CNR1* gene that contains exon 3, but not exons 1 and 2 (Zhang et al., 2004). To study the influence of different splice variants on initiation of heroin addiction, we analyzed patterns formed by the five following SNPs in splice variants A-D of *CNR1*: –22959C>T, –6274T>A, –6215A>C, –5489A>G, and 1359G>A (rs806381) and patterns formed by four SNPs in splice variant E: –6274T>A, –6215A>C, –5489A>G, and 1359G>A (Proudnikov et al., 2009). In Caucasians, in the four-SNP locus we found the pattern TT-AA-AA-GG was found to be associated with heroin addiction. In contrast, we found another pattern, TT-AA-AA-GA, which was found to be associated with protection from heroin addiction. We did not find an association of 385C>A of *FAAH* with heroin addiction (Proudnikov et al., 2009).

Genomewide Association Studies of Opiate Addiction

We have conducted two genomewide association studies to identify genetic variants involved in the vulnerability to develop heroin addiction (Nielsen et al., 2008b, 2010). In the first study, we examined DNA from former severe heroin addicts in methadone maintenance treatment and control subjects free of drug abuse using the 10KAffymetrixGeneChip that interrogates 10,000 variants in a high-throughput mode (Nielsen et al., 2008b). Analyses were performed separately with the autosomal and X chromosomal variants. The variant with the most

significant association with heroin addiction by allele frequency was the autosomal rs965972 located in the Unigene Cluster Hs.147755, a cluster of three expressed sequence tags (ESTs) cloned from kidney and testis. rs965972 is a T to C transition that creates a potential CREB transcription factor binding site and is in a region predicted to have high regulatory potential (Taylor et al., 2006). When genotypes were evaluated, the three variants with the most significant association of genotype frequency with heroin addiction were IVS2+9191A>G (rs1714984), located in an intron of the transcription factor *myocardin* gene (*MYOCD*); rs965972, located in the Unigene Cluster, as described above; and rs1867898, located in a region of high regulatory potential. Myocardin has been shown to control contractility of cerebral arterial smooth muscle cells impacting cerebral blood flow and may be involved in the development of Alzheimer's disease (Deane & Zlokovic, 2007). One genotype pattern of these three variants was found to be significantly associated with vulnerability to develop heroin addiction (odds ratio = 6) and, in this cohort, explained 27% of the population-attributable risk. Lack of another genotype pattern explained 83% of the population-attributable risk and was found to be associated with protection from the development of heroin addiction (odds ratio = 0.13). Evidence was also found for the involvement of the gene coding for the mu-opioid receptor (*OPRM1*), cryptochrome 1 (photolyase-like) (*CRY1*), the metabotropic glutamaterceptors mGluR6 (*GRM6*) and mGluR8 (*GRM8*), and the nuclear receptor, subfamily 4, group A, member 2 (*NR4A2*) in vulnerability to develop heroin addiction.

A second genomewide association study was conducted using a larger cohort and the Affymetrix 100K GeneChip(Nielsen et al., 2010). We employed a method of using pools of multiple DNA samples that was developed to reduce costs (Sham et al., 2002). This technique has been successfully employed by the Uhl group to find variants associated with several forms of drug addiction (Johnson et al., 2009; Liu et al., 2005, 2006; Uhl et al., 2001, 2008). In our study, we combined samples into 23 duplicate pools containing 25 subjects per pool (575 subjects total). Allele frequency determination was done on duplicate pools for 46 total pools. To avoid problems of population stratification in the analysis, pools were constructed by ethnicity: Caucasians, African Americans, or Hispanics. The variant that was found to be most significantly

associated with heroin addiction after correcting for multiple testing was rs10494334 in an unannotated region of the genome. One interesting variant of high significance found in this study was in the *cadherin 4, type 1, R-cadherin (retinal)* gene (*CDH4*), the gene that had been found to contain the most significant variant in a genomewide association study of cerebral brain volume (Seshadri et al., 2007). Multiple variants were found to be clustered in the *cardiomyopathy-associated 3* gene (*CYMA3*) in African Americans. This gene has been reported to be associated with autism (Faham et al., 2005). Evidence was also found for a role in heroin addiction of the *glutamate receptor, metabotropic 8* gene (*GRM8*), the *neural cell adhesion molecule 1* gene (*NCAM1*), and the *gamma-aminobutyric acid (GABA) B receptor* gene (*GABBR2*) in Caucasians, and the *phosphodiesterase 4B, cAMP-specific* gene (*PDE4B*), the *glutamate receptor, ionotropic, N-methyl D-aspartate 2A* gene (*GRIN2A*), and the *glutamate receptor, metabotropic 5* gene (*GRM5*) in African Americans.

In both the 10K and 100K GeneChip studies, the *glutamate receptor, metabotropic 8* gene, (*GRM8*) was found to be associated with vulnerability to develop heroin addiction. This gene codes for a glutamate receptor found in the presynaptic cleft in multiple brain regions (Scherer et al., 1997) and has been found to be associated with schizophrenia in a Japanese cohort (Takaki et al., 2004). The 10K GeneChip study found evidence for involvement of *glutamate receptor, metabotropic 6* gene (*GRM6*) and the 100K GeneChipstudy for *glutamate receptor, metabotropic 5* gene (*GRM5*), and *glutamate receptor, ionotropic, N-methyl D-aspartate 2A* gene (*GIN2A*). These genes code for components of the major excitatory glutamatergic neurotransmitter system. Glutamatergic neurons from the prefrontal cortex innervate the nucleus accumbens, the brain region involved in the rewarding effects of drugs of abuse (reviewed in Schmidt & Pierce, 2010). Release of glutamate in the nucleus accumbens is required for heroin seeking in rodents (LaLumiere & Kalivas, 2008). It is hypothesized that alteration in the homeostasis of glutamate neurotransmission in cocaine addiction drives drug-seeking behaviors (reviewed in Kalivas, 2009).

Conclusion

The body of evidence collected by our laboratory and others suggests a strong influence of genetic components on impulsive behavior. The variants of the genes from several neurotransmitter systems,

which are involved in the modulation of the stress-responsive HPA axis, may influence the pathophysiology of a number of physical and physiological conditions including impulse control and compulsive disorders, anxiety, depressive disorders, and drug addiction. Association studies of selected genetic markers in hypothesis-driven genes and in those identified in gene expression studies using animal models provide compelling evidence that support this hypothesis.

Future Directions

Recent whole-genome association studies have revealed a number of variants of candidate genes that might be involved in the regulation of the opioid, dopaminergic, serotonergic, and other systems that may influence stress responsivity. Close scrutiny of these variants using alternative methodology is necessary to confirm the findings.

Acknowledgments

We thank Dr. Ann Ho and Dr. Lisa Borg for critical reading and Susan Russo for assistance in preparation of the manuscript. This work was supported by Grants NIH-NIDA P60-05130, NIH-NIMH R01-MH-79880 (M.J.K.), NIH/NCRR-CTSA UL1-RR024143 (The Rockefeller University Center for Clinical and Translational Science), NIH-NIMH R01-MH-076537 (H.C., subcontract PI M.J.K.), Department of Veterans Affairs Rehabilitation Research and Development Grant (B6812C: "Neurorehabilitation: Neurons to Networks" (H.L., K.A.), and NIH-NIDA P50 DA018197-06 (T.K.).

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Development of Impulse Control, Inhibition, and Self-Regulatory Behaviors in Normative Populations across the Lifespan

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Abstract

Impulsivity represents a complex multidimensional construct that may change across the lifespan and is associated with numerous neuropsychiatric disorders including substance use disorders, conduct disorder/antisocial personality disorder, and traumatic brain injury. Multiple psychological theories have considered impulsivity and the development of impulse control, inhibition, and self-regulatory behaviors during childhood. Some psychoanalytic theorists have viewed impulse control and self-regulatory behaviors as developing ego functions emerging in the context of id-based impulses and inhibitory pressures from the superego. Object relationists added to this framework but placed more emphasis on mother–child dyadic relationships and the process of separation and individuation within the infant. Cognitive and developmental theorists have viewed impulse control and self-regulation as a series of additive cognitive functions emerging at different temporal points during childhood and with an emphasis on attentional systems and the ability to inhibit a prepotent response. Commonalities exist across all of these developmental theories, and they all are consistent with the idea that the development of impulse control appears cumulative and emergent in early life, with the age range of 24–36 months being a formative period. Impulsivity is part of normal development in the healthy child, and emerging empirical data on normative populations (as measured by neuropsychological testing batteries, self-report measures, and behavioral observation) suggest that impulse control, self-regulation, and other impulsivity-related phenomena may follow different temporal trajectories, with impulsivity decreasing linearly over time and sensation seeking and reward responsiveness following an inverted U-shaped trajectory across the lifespan. These different trajectories coincide with developmental brain changes, including early maturation of subcortical regions in relation to the later maturation of the frontal lobes, and may underlie the frequent risk-taking behavior often observed during adolescence.

Keywords: impulsivity, developmental theories, self-regulation, inhibition, normative, sensation seeking, risk taking, trajectories

Introduction

Impulsivity is a complex multidimensional construct whose core features have been proposed to include (1) the tendency to execute actions too hastily or in a thoughtless manner, (2) the tendency to seek out immediate gratification at the cost of longer-term gains, and (3) difficulty withholding or inhibiting actions (Schachar et al., 1993). In late adolescence and adulthood, impulsivity is associated

with multiple neuropsychiatric disorders including substance use disorders, conduct disorder/antisocial personality disorder, and traumatic brain injury (Marcell, 2007). The question of how best to address impulsive behaviors during early childhood and into adolescence, as well as later in life, remains challenging. That stated, when discussing impulsivity, especially within the context of child and adolescent populations, it is important to consider

when impulsive behavior is normative and at which points during childhood development impulsive behaviors diminish through processes of impulse/cognitive control, inhibition, and self-regulation. Antecedents of impulse control emerge early in life and impulsivity largely decreases across the lifespan, particularly from adolescence/young adulthood into middle and older adulthood. As well, there is evidence for different temporal trajectories of impulsivity-related phenomena (e.g., motor control, cognitive/planning control, sensation seeking, and risk taking). Other chapters in this volume address impulsivity with respect to its definition, components, and associations with neuropsychiatric disorders. In this chapter, we will review the development of a nosology of impulsivity and self-regulation across developmental theories. We will also address the development of impulse control, inhibition, and self-regulation in normative populations, as measured by neuropsychological testing batteries, self-report measures, and behavioral observation, and discuss the correlation of these behavioral changes in the context of our current understanding of the neurobiological changes that occur during development.

Nosology of Impulsivity and Developmental Theories

The word *impulse* originated in the 1500s and stems from the latin *impulsus*, meaning a “pressure to move or act” (<http://dictionary.reference.com/browse/impulse>). Theories on impulsivity and the development of impulse control have existed for over a century. The concept of impulse control is also central to psychoanalytic theory and to models of emotion and emotional regulation.

Psychodynamic Models

Psychoanalytic models view development as a series of epochs that are cumulative and that place strong emphasis on the mother–child dyad and other early relationships (Freud, 1965). Much of developmental psychoanalytic theory can be traced back to three core concepts: Freud’s psychosexual developmental stages (oral, anal, phallic, latency, and genital), the tripartite or structural model of the mind (id, ego, and superego), and object-relations theory (the concept of pleasure and unpleasure in relation to objects and the mother–child unit as a way of developing notions of the self, internal and external environments, and reality). Many of the early psychodynamic theorists viewed impulsive behavior and self-regulation within the context of id-based

instinctual drives and ego control (Pulkkinen, 1986). The id represents instinctual drives and follows what Freud referred to as the *pleasure principle*, striving for immediate gratification of all desires, wants, and needs (Freud, 1923). The superego is considered an internalization of parental authority. The ego is a structure independent of these pressures oriented to adaptation, both internal and external. Hartman et al. (1946) describe an undifferentiated matrix with constitutional contributions from which the id and ego emerge. They postulate a gradual differentiation of the self from the world and subsequent identification of relationships between one’s own body and objects in the first and second 6 months of life. According to Hartman et al., it is in the second year of life that an ego-id differentiation phase emerges from the undifferentiated matrix and the reality principle begins to overpower the pleasure principle and the primary regulatory/drive of behavioral outputs.

Freud and his contemporaries viewed impulse control as one of many ego functions. Rene Spitz (1959) described self-regulation as an important function of the ego and stated that constitutional, early environment, and interactional factors contributed to the self-regulatory process leading to adaptation or maladaptation. In psychodynamic terms, it is the conflict between different substructures of the psychic system, id-based instinctual drives, perceived external conforming pressures of the superego, and the dynamic ego that contribute to the ego function of impulse control. According to Freud, “an instinctual drive, libidinal or aggressive, expressed as a wish, an impulse, or a fantasy, seeks gratification” (Beres, 1999, p. 478). The ego function of impulse control may either allow or disallow gratification. If gratification of the drive is allowed, the result is pleasure, discharge of the impulse in an act of love or hate, as the impulse dictates. If impulse control prevents gratification, it does so by consciously or unconsciously presenting a dangerous situation and, instead of pleasure, the individual experiences “unpleasure” manifested by an affective state such as anxiety, guilt, or depression. During development, ego–superego conflicts can occur prior to the internalization of moral attitudes, ideals, and prohibitions that comprise the superego’s functions. Before structural solidification of superego functions in children, the conflict is between a child’s wish to gratify his or her impulses and the danger of losing parental (and, in adults, social) approval (Beres, 1999). As self-regulation and self-control become more possible, a new sense

of mastery and pride in overcoming drive demands appears, which substitutes for immediate gratification. It is important to note that there are marked differences in impulse/ego control between individuals and that the relative strengths of the psychic structures and drives influence the capacity of the individual to control his or her impulses. Impulse control increases over the lifespan as ego consolidation occurs (Buxbaum, 1991). During the latency period, ego consolidation aids in inhibition and control of drives, while in a mutually influential way, a biologically determined drive diminution facilitates ego consolidation (Buxbaum, 1991). Psychoanalytic analyses of patients with antisocial personality traits suggest that impulsivity and lack of functional impulse control are associated with malformation of the superego (Aichhorn, 1935) or with the ego keeping the superego at a distance and thus unable to prevent the individual from yielding to an impulse (Reich, 1933). In Freud's paper "The Ego and the Id," he describes the ego's purposes as assisting the id by "mastering the tensions" (Freud, 1923). Freud felt that, over time, the ego "develops from perceiving instincts to controlling them and from obeying instincts to inhibiting them" (Freud, 1923, p. 54).

Object relations theorists such as Hartman and Mahler viewed an individual's psychic structure in terms of his or her relation to early objects, their internal representations, and the interactional nature of their developmental processes (Tyson & Tyson, 1995). Among the object relationists, Margaret Mahler offered a developmental model in which object relations and the self are seen as outgrowths of the instinctual vicissitudes (Mahler & Furer, 1968). She described a *psychological birth* temporally separate from the biological birth and occurring in infancy with the sense/awareness of a self separate from others, with internal and external influences occurring in a process that she called *separation-individuation*. Mahler's developmental model presents development starting with what she termed the *normal autistic state* (internally focused), followed by a *symbiotic period* (in which the mother-child unit functions and is perceived by the infant as one). Separation-individuation proceeds through a series of four sequential subphases: (1) differentiation (4–9 months of age), during which the infant differentiates himself or herself from the mother; (2) practicing (9–15 months of age), during which the child is actively exploring the world through locomotion, establishing a sense of bodily boundaries and awareness; (3) rapprochement

(15–24 months of age), during which the infant becomes aware of the separateness and has the ambivalence to reach for the mother but also develops a sense of independent functioning; and (4) consolidation (at around 36 months of age), during which emotional object constancy begins. According to Mahler, it is in the separation-individuation subphase of rapprochement that self-regulation begins to emerge. Settlage (1977) identifies the developmental tasks of the rapprochement subphase as mastery of the intense feeling of separation anxiety, affirmation of basic trust, increased autonomy, firming up of the core sense of self, and establishment of affect and drive regulation (Settlage, 1977). Mahlerian analysts view the rapprochement subphase as the critical period of character formation crucial to the conflict between autonomy and dependence, separateness and closeness, and self-regulation and impulsivity (Kramer & Akhtar, 1988).

In Eric Erikson's stages of psychosocial development, eight stages define progression across the lifespan with successive crises and tasks that must be mastered or resolved as individuals progress through life (Erikson, 1950). Erikson's psychosocial theory also addresses the construct of impulse control during development. According to Erikson, impulse control emerges in early childhood (2–3 years of age) during the stage of autonomy versus shame and doubt. During this stage, the child develops a sense of control over physical urges, exemplified by toilet training, and with this control comes a sense of independence and autonomy. Erikson felt that a failure to navigate this stage leads to persistent impulsive behaviors.

Loevinger and other psychoanalytic theorists provided stages for the development of ego functions: presocial, impulsive, self-protective, conformist, conscientious, and autonomous (Loevinger, 1973). Unlike Erikson's stages, these psychodynamic stages are not related to age-specific tasks and thus are more plastic in terms of their developmental ranges. The *impulsive stage* begins around the developmental period when language develops (the second year) and is characterized by impulsive, egocentric, concrete behaviors. The next stage, known as the *self-protective stage*, is more ritualistic and is considered the first step toward control of impulses. In this stage, the child calculates his or her self-interests, weighing external rewards and punishments rather than acting on impulse. This stage progresses throughout childhood and sometimes into adolescence before the transition to the

conformist stage (in which rules are obeyed because they are the rules). Another psychodynamic theorist, Getsinger (1977), proposed ego delay as a construct to explain individual differences, with individuals with low ego delay being impulsive and having difficulty inhibiting responses and individuals with high ego delay being restrictive and able to inhibit impulses.

Taken as a whole, the different psychodynamic developmental theories view impulsive behaviors as predominantly occurring normatively during early childhood. As the ego and superego develop and strengthen throughout childhood and into adolescence, individuals become better able to control need-driven behaviors.

Cognitive and Developmental Theories

In the cognitive and developmental psychology literature, numerous theories on the construct of impulse control and its development have been postulated. One construct with significant overlap with impulsivity is that of *effortful control*. Rothbart and colleagues developed the theory of effortful control to denote a class of self-regulatory mechanisms, and effortful control is defined as the ability to suppress a dominant response and to instead perform a subdominant response (Rothbart, 1989). Effortful control emerges between 6 and 12 months of age, developing in concert with the maturation of attentional mechanisms (Rothbart et al., 1994). Another model of impulse control, developed by Maccoby and colleagues, includes two major components: (1) inhibition of action (motoric and cognitive) and (2) control of emotions (tolerating frustration, delaying gratification, and controlling excitement; Maccoby, 1980). Maccoby also integrates parent-child relationships into his developmental model and has demonstrated that adaptive, sensitive parenting fosters impulse control, ego control, and self-regulation (Maccoby, 1980). Kopp (1982) presented a developmental model of self-regulation as a complex multidomain construct of behavior and emotion with an emphasis of socialization of the child to everyday norms. Silverman and Ragusa (1992) viewed self-regulation as a superordinate construct encompassing activity, impulsivity, attention span, delay, and response inhibition. Milich and Kramer (1984) conceptualized impulsivity by examining tasks of impulse control typically used to assess this construct and discovered a common thread: the varied definitions of impulsivity all involve speed of response (rapid responding) accompanied by errors or undesirable behavior (i.e., the tendency to assert

insufficient control). Olson (1989) viewed impulsivity as a multidimensional construct influencing both social as well as cognitive functioning. Buss and Plomin (1975, 1984) viewed impulsivity as involving inhibitory control, decision time, sensation seeking, and persistence and as the “brakes” on temperament dimensions of emotionality, activity, and sociability.

It is important to note that across all of the developmental theories addressed in this chapter, the precursors of impulse control and self-regulative behaviors appear in early childhood. And conversely, with delayed or impaired development of the early developmental precursors, the individual will have difficulty with impulsive behavior throughout life (Tyson & Tyson, 1995). Also, as will be explored later in this chapter, inhibition and delay of gratification, which we may consider to be the antecedents or precursors of impulse control as measured by neuropsychological testing, also appear to develop within the first 3 years of life.

Cognitive Developmental Approaches to the Assessment of Impulse Control

The scientific community has approached the measurement of impulsivity and impulse control in at least three ways: (1) neuropsychological tests of impulse/cognitive control and inhibition; (2) behavioral observation; and (3) self-report measures. Each of these approaches is associated with specific advantages and disadvantages that enhance and limit the utility, generalization, and validity of the studies in which they have been used. The construct of impulse control can be further divided into component features that, across different developmental models, include inhibition of motor action (motor impulse control), inhibition of cognitive action (cognitive/planning impulse control), response inhibition, delaying gratification, and speed of processing/response time (Maccoby, 1980; Milich & Kramer, 1984; Silverman & Ragusa, 1992). Many of these components have been examined empirically, as will be described below.

Neuropsychological Tests of Cognitive Control and Inhibition

Tests of impulse control and its correlates have emerged from both the developmental psychology and adult neuropsychology literature. In this section, we address the neuropsychological tests of impulse control and inhibition across four developmental age ranges: 0–3, 3–7, 8–12, and 13–18 years.

Arguably the earliest precursors of impulse control emerge in the form of the ability to inhibit a prepotent response. This ability begins to develop on simple tasks within the first year of life. A *prepotent response tendency* may be defined as the tendency to repeat a positively reinforced response to a stimulus (Diamond, 2002). For example, in the Piagetian *object permanence task*, also known as the *A-not-B task*, an infant is shown a ball placed under one of two boxes (A to the left and B to the right) and, after a set time delay, must reach for the box with the ball in it (Piaget, 1954). In a second trial, when the ball is placed in the other location (box B instead of box A), the infant must inhibit his or her prepotent response to search for the ball in the previously known location. Two simple tasks that assess the ability to inhibit a prepotent response, the object permanence task and the object retrieval task, both show significant improvement from 6 to 12 months of age (Diamond, 1985, 1988). The psychoanalytic object relation theorists viewed Piaget's findings as support of early differentiation of self from other objects. Since the time of these initial studies, other groups have explored the early child's (less than 3 years of age) effortful control and ability to inhibit prepotent response tendencies. In a longitudinal study by Kochanska and colleagues (2000), children were followed from 9 to 33 months of age and evaluated at ages 22 and 33 months on five measures of effortful control (delaying, slowing down motor activity, suppressing/initiating activity to signal, effortful attention, and lowering the voice). The five measures of effortful control cohered at 22 months of age and improved markedly from 22 to 33 months. These measures correlated positively with parents' rating scale scores of their child's self-regulation. In this study there was a gender effect, with girls performing better than boys on tasks of effortful control at both ages. Another study by Gerardi-Caulton (2000) used a spatial incompatibility test to demonstrate that by 30 months children were able to inhibit a prepotent response and by 36 months the subjects were correct 90% of the time. Performance on this spatial incompatibility task correlated positively with behavioral measures of self-regulation including a snack delay, a whisper game, and a tower-building task.

The developmental window from 3 to 7 years of age is marked by large improvements in a variety of tasks of inhibition that also require holding information in mind, including tapping, day-night, color-object Stroop, simple card-sorting, and go/no-go tasks. The day-night task is a task of inhibition in

which a child must hold two rules in mind, inhibiting saying what the stimulus actually represents and instead saying the opposite (i.e., saying "night" when shown a white card with a picture of the sun; Gerstadt, 1994). Significant improvements in the speed of response occur from 3.5 to 7 years of age, with the most dramatic changes occurring between 3.5 and 4.5 years of age. This finding is congruent with findings from Diamond's color-object Stroop task, during which a child must inhibit giving a dominant response (saying the name of the object, e.g., "carrot") and instead give a subdominant response (color of the object, i.e., "orange"; Prevor & Diamond, 2005). In this task, reaction time decreased significantly from 3.5 to 6.5 years of age. Luria's (1966) tapping test also requires remembering two rules, inhibiting a prepotent response and providing the opposite response instead (i.e., "tap once when the experimenter taps twice and tap twice when the experimenter taps once"). It also requires appropriate motor coordination to successfully complete. In this task as well, the greatest improvements in correct responses occur from 3.5 to 4.5 years of age (Passler et al., 1985). The most significant improvements in tapping speed on Luria's test occur from 4.5 to 5.5 years of age.

The go/no-go task is one of the classic neuropsychological measures of inhibition and frontal lobe function and has been widely used in clinical as well as research settings and in animal models (Diamond, 2002). In this task, the child is expected to respond to one stimulus (e.g., press a button when shown an O) but do nothing when shown another (e.g., not press the button when shown an X). It is another example of a task in which children are able to restate the instructions correctly but cannot act according to the rules for the task (Tikhomirov, 1978). Multiple studies exploring variants of the go/no-go paradigm have consistently demonstrated that children do not succeed at the task until they reach 4.5 years of age and that there is continued improvement with age throughout adolescence and into young adulthood (Casey et al., 1997; Dowsett & Livesey, 2000; Garber & Ross, 1968; Jeffry, 1961; Luria, 1961; Tikhomirov, 1978; Van der Meet & Stemerdink, 1999). In a functional magnetic resonance imaging (fMRI) study, Casey et al. (1997) found increased activity in the inferior frontal gyrus (inb ventrolateral and dorsolateral prefrontal cortices), middle frontal gyrus, and orbital frontal gyrus on no-go trials compared with that on go trials (Casey et al., 1997). As improvement on all of these tasks of inhibition occurs from 3 to 7 years of age, this period

appears to be an important stage during which the neural circuits of inhibitory control develop.

From 8 to 12 years of age, significant improvements occur in speed of processing, the ability to use strategies, and working memory or the ability to hold information in mind while manipulating it, as well as in developing inhibitory control. During this time period, as children's working memories improve, they become able to hold information in mind while resisting interference, attentional inertia, and prepotent responses. Engle and Kane have defined *working memory* as the ability to actively hold information in mind while concurrently blocking or inhibiting other information from entering the active state (Kane & Engle, 2000). Working memory function is inversely related to impulsivity, and working memory is often assessed using task-switching paradigms (Romer et al., 2009). While children from 3 to 7 years of age can begin to perform such tasks, they have difficulty with the increasing complexity of the tasks as their working memory load is taxed. Tasks such as the pattern, counting, and spatial span tasks, the Wisconsin Card Sorting Test (WCST), the directed forgetting task, the directional Stroop task, and the antisaccade task all improve significantly from 8 to 12 years of age. Some of these tasks assess cognitive functions that follow a more protracted developmental course that continues through adolescence and into adulthood. Children reach an adult level of skill on the compound stimulus visual information task, the pattern span task, the counting and spatial span tasks, and the WCST between 10 and 11 years of age (Case, 1995; Case et al., 1992; Welsh, 1991; Wilson et al., 1987). Using a directed forgetting paradigm, Harnishfeger and Pope (1996) discovered that children 11 years of age have significantly more intrusions of to-be-forgotten words than do matched adults. Diamond's directional Stroop task is a spatial incompatibility task and requires inhibition of the tendency to make a prepotent response (i.e., the tendency to respond by pressing the button on the same side as the stimulus; Diamond et al., 1998). The ability to inhibit the natural tendency to respond on the same side as the stimulus, as measured by accuracy and response time on task, follows a protracted developmental course, improving linearly from 4 to 26 years of age (Davidson et al., 2006). The antisaccade task requires the subject to inhibit a prepotent response and suppress the tendency to look reflexively at (saccade to) a visual stimulus in the periphery, and instead look in the opposite direction (Fischer et al., 1997). Performance

on this task improves from 8 to 25 years of age. These tasks, which have a protracted developmental course, have a common thread: they all require subjects to hold information in mind while they inhibit responses.

Developmental studies of impulse control and executive function to date have provided additional support for different developmental trajectories of component features of impulse control (Klenberg et al., 2001; Welsh et al., 1991). Welsh and colleagues studied a normative sample of 100 subjects ranging from 3 to 12 years of age and 10 young adults on a variety of measures of executive function to ascertain when adult-level performance was achieved (Welsh et al., 1991). In their study, visual search efficiency and simple planning (as measured by a three-disk tower of Hanoi task) reached adult-level performance by age 6, followed by the WCST and the matching familiar figures test by age 10. Lastly, motor sequencing, verbal fluency, and a complex spatial planning task (four-disk tower of Hanoi task) did not reach adult-level performance until the age of 12. Tasks that were more complex, requiring larger cognitive loading and greater impulse control, reached adult-level performance at later ages when children had developed the ability to hold greater amounts of information in working memory and create more sophisticated strategies for efficiently and successfully performing the tasks. In another normative developmental study, Klenberg and colleagues tested 400 Finnish children ranging from 3 to 12 years old on age-appropriate subtests of impulse control and other executive functions from the NEPSY (a series of psychometric normed tests developed by Korkman, Kirk, and Kemp to assess neuropsychological development of children 3–16 years of age; Klenberg et al., 2001). The results of this study replicate the findings of Welsh et al. and suggest that cognitive development appears to proceed sequentially from motor inhibition to functions of selective and sustained attention and finally to language/fluency-related executive functions. Different inhibition and impulse control-related functions such as motor inhibition, set-shifting, and simple and complex planning come online at different time points as assistive skills and supportive functions emerge (i.e., the ability to sustain attention, to imagine and utilize increasingly complex strategies, and to increase reaction speed). Cumulatively, this literature suggests that impulse control and self-regulation, as measured by neuropsychological tasks of response inhibition, reaction time/speed of processing, and ability to inhibit a

prepotent response, begin to emerge as early as 24–36 months and become increasingly accurate and efficient with increasing age across normal development. This age-related unfolding of self-regulatory behavior parallels many of the developmental models of impulse control from both the psychoanalytic and cognitive/developmental psychology frameworks.

Behavioral Observations Delay of Gratification Paradigm

Another way of assessing the development of impulse control and self-regulation is to observe behaviors and choices of subjects when placed in controlled environments. While we previously discussed the behavioral observations of Kochanska in his studies of effortful control in young children, Michel's longitudinal delay-of-gratification work is arguably the best-known of these behavioral observation studies. Mischel and colleagues, in a series of experiments, used a delay-of-gratification paradigm to test 3- and 4-year-old children's ability to self-regulate, demonstrating meaningful individual differences in impulse control and its correlates (Mischel, 1989). Typical delay-of-gratification tasks require choice decisions in which an individual must postpone an immediately available reward in order to attain a delayed but more valuable one, usually in the form of a toy or food (i.e., receiving one cookie now versus two cookies later). When the children were categorized into those who were able to delay gratification versus those who were not, the children who selected the delay were found to be more intelligent, higher achieving, and more likely to resist temptation. In addition, on follow-up studies more than 10 years later, those who had selected the delay were more academically and socially competent, with higher scholastic aptitude test (SAT) scores, and were less likely to use alcohol or other substances of abuse (Ayduk et al., 2000; Mischel, 1989). Attentional allocation was found to be a factor in children's performance on this task. As well, the study revealed that those children who chose delayed gratification were able to use strategies such as distraction and verbal self-encouragement when instructed to, and that the ability to develop internal strategies emerged at around 6 years of age. Over the period of child development, the complexity of strategies improves, as does the ability to delay gratification. Interestingly, in a longitudinal study by Eigsti et al. (2006), the ability to delay gratification as preschoolers was positively associated with efficiency on a go/no-go task 10 years

later, suggesting that these individual differences in self-regulation and impulse control begin early in life and have some stability or predictive power with respect to developmental trajectory. Wilson et al. (2009) demonstrated that individual differences in physiological response, including heart rate and electrodermal response, during the delay-of-gratification task can help predict self-regulation. Subsequent studies have also correlated delay of gratification at 4 years of age with an increased risk of being overweight as an adolescent (Seeyave et al., 2009; Tsukayama et al., 2010).

Delay Discounting Paradigm

Delay discounting paradigms, used in behavioral economic studies, assess a subject's preference for future versus immediate outcomes by asking the respondent to choose between a small immediate reward and a delayed reward of greater value (e.g., would you rather have \$200 today, \$300 1 week from today, or \$500 1 month from today?). By repeating the trials of reward choice under varying the reward value and delay time conditions, individual hyperbolic discounting functions can be found. Across studies, comparative data used from delay discounting paradigms include the relative steepness/slope of the discounting curve and the indifference point. The relative steepness/slope of the discounting curve indicates the subject's preference for immediate, small rewards over delayed, larger ones, with steeper discounting functions (reflecting a preference for smaller, immediate rewards) being found in risk-taking and substance-abusing populations (Bickel et al., 1999). If enough trials are administered, subjects' responses converge and preferences for immediate and delayed rewards are equal. The intersection of responses is referred to as the *indifference point* and reflects the subjective value of the delayed reward if it were offered immediately (i.e., the discounted value of the delayed reward; Green et al., 2005). Paralleling steeper discounting functions, a subject with a lower indifference point values smaller immediate rewards. Delay discounting task performance involves both impulse control and future orientation. A functional neuroimaging study by McClure and colleagues (2004) suggests that delay discounting decisions reflect the engagement of two separate neural systems: a reward-seeking system encompassing the paralimbic cortex and midbrain dopaminergic system and a future-orienting, reasoning, and planning system involving the lateral prefrontal and parietal cortices. In this study, the cross-talk between these two

systems was directly associated with each subject's choice, with greater parietofrontal relative to limbic activity when subjects chose the delayed larger reward. As well, in a recent longitudinal study of twins, delay discounting showed significant heritability (30% and 51% at two different ages) and correlated with measures of conduct disorder, attention deficit hyperactivity disorder (ADHD), substance use, novelty seeking, and poor self-regulation (Anokhin et al., 2010). The literature on age-related changes in delay discounting appears limited and incongruent. In a study by Scheres et al. (2006), children (6–11 years of age) had a significantly steeper discounting curve than adolescents (12–17 years of age). Another study by Green and colleagues (1994) compared early adolescents (average age 12 years), young adults (average age 20 years), and older adults (average age 68 years) and found a significantly steeper discounting function in adolescents compared to young adults and a significantly steeper discounting function in younger compared to older adults. A follow-up study by Green's research group in an attempt to replicate their previous findings did not find a significant difference in the discounting function slope between a second group of younger and older adults (Green et al., 1996). Studies to date have consistently shown hyperbolic temporal discounting functions, leading researchers to conclude that differences in discounting are quantitative rather than qualitative factors (Green et al., 1999).

Adding to the data on discounting is Steinberg and colleague's (2009) large cross-sectional study in which 935 subjects between the ages of 10 and 30 years completed future orientation scales and a delay discounting task. While no significant correlation was found between age and discounting variables of rate and indifference point, when the sample was separated into subgroups during post-hoc analyses, subjects under 13 years of age had significantly steeper discounting rates than subjects aged 16 years and older, although no differences were noted in subjects who fell between the two age ranges. These data suggest that a person's tendency to discount (i.e., prefer/pursue a large but delayed reward over a smaller immediate reward) develops from childhood to young adulthood and that the period of time from 13 to 16 years of age is an important developmental period for reward discounting.

Self-report Measures of Impulsivity

While numerous impulsivity rating scales have been developed and normed, some for children and

adolescents, few studies have examined age-related changes of self-reported impulsivity. Some data can be gleaned from the standardization data in developing these scales. Fossati and colleagues (2002) adapted the Barratt Impulsiveness Scale-11 for adolescents (BIS-11-A) and administered it to a group of 563 Italian high school students of ages 13–19 years. In this group they found no significant correlation between age and BIS-11-A total score, but compared to a cohort of adults, the average total impulsiveness score for the adolescent population was significantly higher than that of a previously reported sample of Italian college students. As well, Eysenck's impulsivity questionnaire (Junior I_o) administered to 1504 children of ages 7–15 years demonstrated a slight but nonsignificant upward trend on scores of impulsivity, venturesomeness, and empathy (Eysenck et al., 1984). In a factor analytic study by Leshem and Glicksohn (2007) on 182 subjects ranging from 14 to 22 years of age who were separated into a younger group (ages 14–16) and an older group (ages 20–22), there was a significant decline with age on self-rated impulsivity scores (the Eysenck impulsiveness subscale and the Barratt Impulsiveness Scale) that correlated with decreased scores on neuropsychological tests of impulsivity. Another study by Stanford and colleagues (1996) showed higher self-reported impulsivity in high school students relative to college students. Two cross-sectional studies of self-reported impulsivity in adults found different results, one showing a decline in impulsivity with age (using the Eysenck impulsivity scale) and one showing no change in impulsive behavior across adulthood (using the Dickman Impulsivity Inventory; Claes et al., 2000; Eysenck et al., 1985). In both studies, males were found to be more impulsive than females.

Two studies have examined self-reported impulsivity across a broad range of ages. In an imaging study by Galvan et al. (2007), scores on the Connor Impulsivity Scale-Revised correlated negatively with chronological age in a group of evenly distributed children, adolescents, and young adults of ages 7–29 years. The largest study to examine multiple measures of impulsivity across a broad age span was done by Steinberg and colleagues (2008), who completed a cross-sectional study of 935 individuals between the ages of 10 and 30 years. They noted a linear trajectory of impulsivity over the lifespan decreasing from age 10 to 30. The self-reported impulsivity literature as a whole suggests that impulsivity is highest in preadolescence and early adolescence and declines thereafter.

Impulsivity Correlates and Risk Taking in Adolescents: Developmental Trajectories

While some studies suggest that impulsivity may follow a linear pattern, declining from age 12 years on, other related constructs, namely, sensation seeking, future orientation, and risk taking, may follow nonlinear trajectories (Steinberg et al., 2008). Impulsivity and risk taking represent distinct yet related constructs. Considerable evidence suggests that risk taking and suboptimal choice behavior are higher during adolescence than during preadolescence and adulthood, as evidenced by a variety of risk-taking scales and increases in risky behaviors (e.g., unprotected sex, binge drinking, reckless driving, and criminal behavior; Eaton et al., 2006; Steinberg et al., 2008). Multiple hypotheses have been generated to explain why the transitional period of adolescence is marked by a higher degree of suboptimal choice behavior. From a decision-making standpoint, an inability to conceptualize and make logical decisions does not appear to account for this phenomenon. While adolescents have difficulty modifying their behaviors and reducing their risk taking, they are able to reason about hypothetical dilemmas and weigh the risks and benefits of their behaviors (Reyna & Farley, 2006). As also stated previously, impulsivity may follow a linear trajectory and decrease with chronological age. In contrast, self-reports of sensation seeking follow a curvilinear pattern (similar to that of risk-taking behaviors), increasing during the first half of adolescence and then steadily declining from age 16 on (Steinberg et al., 2008). In Steinberg et al.'s study, puberty was associated with sensation seeking but not with impulsivity in male subjects. This suggests a role for sex hormones in sensation-seeking, reward sensitivity, and risk-taking behaviors, and subsequent studies have correlated levels of testosterone and other endogenous steroids with risk-taking behaviors in both adolescent and adult males (Coates & Herbert, 2008; Vermeersch et al., 2008). Future orientation also plays a role in suboptimal choice behaviors, as the weighing of short-term versus long-term consequences, their salience, and their probability strongly contribute to choice behavior. In the study by Steinberg and colleagues (2009), orientation to the future was measured by a variety of rating scales. Future orientation was found to increase with age on measures of time perspective and anticipation of consequences, while self-reported planning followed a curvilinear (quadratic) trend, with a decline in planning from 10 to 15 years of age followed by an increase thereafter

(Steinberg et al., 2009). Taken together, these findings indicate that adolescence is a period of (1) elevated but declining levels of impulsivity, (2) increased novelty/sensation seeking, and (3) decreased planning. The combination of these cognitive elements contributes to suboptimal or risky choice behaviors. Further complicating this picture are the group dynamic and environmental influences that may increase risk-taking behavior in adolescence. A study by Lewis and Lewis (1984) on peer-peer interactions and peer pressure demonstrated that peer pressure and "dares" encouraged problematic behaviors including a high likelihood of injury risk behaviors, substance experimentation, permissive sexuality, and a cluster of deviant behaviors (vandalism, lying, stealing; Lewis & Lewis, 1984). Another study of young men participating in competitive skateboarding demonstrated that the presence of an attractive female increased serum testosterone and physical risk taking (Ronay & von Hippel, 2010). These findings suggest that increased physical risk taking and increases in testosterone may be involved in aspects of sexual courtship and, perhaps by extension, risky sexual behaviors (Lewis & Lewis, 1984; Ronay & von Hippel, 2010).

Neurobiological Correlates of Impulsivity

Data suggest that the neural circuitry of motivation and impulse control involves a series of parallel cortical-striatal-thalamic-cortical circuits with interconnections between the prefrontal cortex and ventral striatum (nucleus accumbens; Chambers et al., 2003). Neural development begins in utero and with some brain regions continues throughout life. From 7.5 to 12 months of age, there is substantial growth in the dendritic branches of pyramidal neurons in layer III of the dorsolateral prefrontal cortex (DLPFC) and by 12 months of age those dendrites have reached their full mature extensions (Koenderink et al., 1994). Dendritic branching (in the total length of uncut terminal segments and in radial distance) appears to plateau at 1 year of age—a plateau that extends as far as 27 years of age. As well, levels of glucose metabolism as measured by positron emission tomography (PET) imaging in DLPFC reaches approximate adult levels by 1 year of age (Chugani et al., 1987). These initial neural adaptations in the frontal lobes coincide temporally with the development of early precursors of impulse control defined as the ability to inhibit prepotent responses on simple tasks. Following this childhood peak in synaptic number, arborization, glucose utilization, and gray matter (on volumetric MRI

studies), there is during adolescence synaptic pruning during refinement of neuronal networks (Giedd et al., 2008). These changes follow a caudal-to-rostral pattern and progress with the developmental maturation of higher-order association cortices only after lower-order somatosensory and visual cortices have matured (Gotay et al., 2004). Thus, the frontal lobes do not reach full maturation until young adulthood. In contrast, the subcortical structures and limbic system mature earlier. Casey et al. (2008) suggested that the interface between these two systems in the adolescent brain—the relatively mature nucleus accumbens/midbrain dopaminergic system and the relatively immature prefrontal lobes—leads to increased risk-taking behaviors. Galvan et al. (2007) found that nucleus accumbens activity measured during a reward task on fMRI correlated positively with risk-taking behavior but not with impulsivity.

While the midbrain dopaminergic system and its interconnections with the frontal lobes appear relevant to impulse control behaviors, other neurotransmitters and neuronal circuits have also been implicated. Soubrie's (1986) work in animal models suggests that interruption of serotonergic neural circuitry is associated with an increase in behavioral disinhibition and impulsivity. In addition, a study by Robinson and colleagues (2008) showed that a selective noradrenergic reuptake inhibitor, atomoxetine, decreased impulsive behavior in a rat model, suggesting a role for norepinephrine in reducing impulsivity.

Conclusions

The construct of impulsivity reflects multiple models and developmental theories postulated to explain its unfolding in early childhood. Psychoanalytic theorists have viewed impulse control and self-regulation primarily as ego functions, developing as the ego strengthens/fortifies in relation to the instinctual drives of the id and the pressures of the superego. Object relationists built on the work of structural theorists and have explored the interactional nature of developmental processes (including impulse control), focusing specifically on the early mother-child relationship and the process of separation and individuation within the infant. Cognitive and developmental psychological theorists have viewed the development of impulse control and self-regulation as additive cognitive functions emerging at different time points in early childhood. They have focused on concurrent development of attentional systems and the ability to inhibit a prepotent response,

which is thought to be a precursor to regulatory behavioral control. While differing in their explanations and valuations of the importance of environmental factors, most developmental theories of impulsivity have a common thread in describing the development of impulse control as being cumulative and emerging in early life, with the age range of 24–36 months being a formative period. As well, all of the developmental theories note that impulsivity, while associated with various neuropsychiatric diseases, is also a part of normal development of the healthy child. Emerging empirical data support the conclusion that impulse control and self-regulatory behaviors develop over the course of childhood, with full maturation of the neural circuitry of impulse control not being achieved until young adulthood or possibly later. Interestingly, different components of impulse control appear to come online at different time points during development. The antecedents of self-regulatory behaviors begin to emerge from 24 to 36 months of age as the ability to inhibit a prepotent response. Self-regulation and impulse control as measured by increasingly complex tasks of inhibition continue to improve during childhood, adolescence, and into young adulthood, with motor impulse control coming online prior to cognitive/planning impulse control. While there are limited and arguably somewhat incongruent data on the topic, delay discounting also appears to change from adolescence to young adulthood, with individuals demonstrating greater ability to delay gratification for larger rewards as they grow older. Although it is presently unclear at which points different aspects of impulsivity peak, data suggest that self-reported impulsivity is high at 10–12 years of age and then decreases linearly during adolescence and young adulthood. Conversely, sensation seeking and reward responsiveness appear to follow different developmental trajectories than impulsivity, peaking in adolescence and following an inverted U-shaped trajectory across the lifespan. These different trajectories may underlie the increase in risk taking that is noted to occur during adolescence in comparison to preadolescence and adulthood. This normative development of impulse control and other theoretically related constructs (sensation seeking and risk taking) coincides with the caudal-to-rostral developmental brain changes and early maturation of the nucleus accumbens and other subcortical regions in relation to the frontal lobes. Most studies on development of impulsivity have been cross-sectional in nature. Future studies on the development of impulse control should investigate

further the genetic, epigenetic, neurobiological, and environmental factors that contribute to this process.

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Impulsivity and Affective Regulation

Alan C. Swann

Abstract

Impulsivity and affect share important neurobehavioral mechanisms. Impulsivity is a pattern of responses to stimuli without the ability to conform the responses to their context, usually representing either inability to adequately evaluate a stimulus before responding to it or inability to delay the response for a reward. Mechanisms underlying impulsivity overlap substantially with constructs like arousal, attention, motivation, and reward, which are also prominent in regulation of affect. Both impulsivity and affect share relationships with regulation of monoaminergic and amino acid transmitter function. For example, activity of the locus coeruleus is sensitive to unexpected, intense, noxious, or stress-related stimuli. Impulsivity and affective dysregulation are increased by exaggerated or poorly modulated responses in this system. The course of the illness interacts with context-dependent effects on behavior via behavioral sensitization. Repeated exposure to stressors, drugs of abuse, or endogenous norepinephrine release in affective episodes leads to behavioral sensitization with increased impulsivity, affective dysregulation, and substance use. Impulsivity predisposes to, and is increased by, behavioral sensitization. In this context, we discuss impulsivity in depressive, manic, anxious, and mixed states, including suicidal behavior and characteristics of the course of illness that are related to behavioral sensitization.

Keywords: affect; anxiety; arousal; bipolar disorder; depressive disorder; impulsive behavior; motivation; stress; psychological; recurrence

Introduction

Impulsivity and affect share common mechanisms that are part of the initiation of action (Barratt & Patton, 1983). Impulsivity and affect overlap in the manner in which they are related to behavioral processes regulating the initiation of action. Their interaction bridges attentive and preattentive aspects of behavior (Barratt & Patton, 1983; Bechara et al., 1997; Moeller et al., 2001).

Properties of Impulsivity Related to Affective Regulation

Impulsivity can be defined as “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative

consequences” (Moeller et al., 2001, p. 1783.) resulting from impaired information processing outside of conscious deliberation (Barratt et al., 1997). Impulsivity involves preattentional and early attentional processing of information and is part of the initiation of action, which requires a balance between generation and screening of potential acts (Barratt & Patton, 1983). Impulsive behavior occurs when this balance fails, with excessive spontaneous behavior or stimulation (e.g., mania; Swann et al., 1987, 2001a) or deficient screening of potential behavior (as in frontal lobe trauma; Bechara et al., 1997), increasing the probability of action without conscious reflection. Impulsivity is prominent in affective disorders (Swann et al., 2001a, 2004, 2008)

and in related problems that often co-occur with affective disorders, including addictive disorders (Bickel et al., 1999; Sarramon et al., 1999; Swann et al., 2004), behavioral effects of abused drugs (Jentsch & Taylor, 1999; Moeller et al., 2002), personality disorders (Anderson & Revelle, 1994; Steinberg et al., 1997), stress-related disorders (Rinne et al., 2000; Southwick et al., 1999), high-risk psychiatric outpatients (Crean et al., 2000), and intermittent explosive disorder (Virkkunen & Linnoila, 1993). Impulsivity confers an increased risk for rapid behavioral responses with severe long-term consequences, most notably suicide (Cremniter et al., 1999; Gut-Fayand et al., 2001; Mann et al., 1999; Soloff et al., 2000; Swann et al., 2005b).

Mechanisms of impulsivity are related to those of arousal and of physiological responses to stressors (Arnsten et al., 1999; Cameron et al., 2000). These relationships have important overlaps with the regulation of affect. Changes in affect can potentially influence impulsivity, and vice versa. This chapter will focus on these relationships.

BEHAVIORAL MODELS OF IMPULSIVITY

As discussed in more detail elsewhere in this volume, impulsivity is complex, with models essentially derived from empirical observation of behavior; this behavior can be measured by behavioral tasks or questionnaires and has analogues in animal behavior (Evenden, 1999). These models are useful for delineating the behavioral mechanisms involved in impulsive behavior. Most impulsive behavior is a hybrid of more than one of these models, though one model may dominate.

Rapid-response impulsivity is based on responding without reflection or adequate screening of the stimulus and its context (Evenden, 1999). This model requires a filtering or screening function, served by the amygdala, prefrontal cortex, and other structures, and can be assessed with continuous performance, go/no-go, or stop-signal tasks (Dougherty et al., 2000; Halperin et al., 1988; Logan, 1994; Winstanley et al., 2006).

Reward delay or delay discounting impulsivity is based on the preference for a small immediate reward over a larger delayed one, an exaggeration of the normal hyperbolic loss in value of a future reward with increased time (Evenden, 2000; Swann et al., 2002a). Two-choice tests or other measures comparing the preference for immediate versus delayed rewards are validated measures in humans (Ho et al., 1999; Hyten et al., 1994).

INTEGRATED MEASURES

OF TRAIT-LIKE IMPULSIVITY

Integrated impulsivity is measured by questionnaires that integrate behavioral and cognitive aspects of impulsivity with their consequences. It is important to understand that, while impulsivity is often regarded in terms of *trait* and *state* characteristics, a more useful categorization may be context dependence versus context independence. High trait impulsivity may be expressed only in certain contexts. The Barratt Impulsiveness Scale (BIS-11) identifies three oblique factors associated with impulsivity as an enduring characteristic: attentional impulsivity, or lack of cognitive persistence with inability to tolerate complexity; motor impulsivity, or acting on the spur of the moment; and nonplanning impulsivity, or lack of a sense of the future (or the past; Patton et al., 1995). The BIS-11 may capture a context-independent measure of integrated impulsivity that predicts its context-dependent expression. As we will discuss below, the three BIS-11 subscales have differential but overlapping relationships to the affective state.

Another scale, Urgency-(lack of) Perseverance-(lack of) Premeditation-Sensation seeking (UPPS), measures aspects of behavior germane to relationships between impulsivity and affect. The Urgency component of the scale addresses the fact that any strong affect can elicit rash behavioral responses. It refers to positive urgency as rash responses to positive affect or to positive stimuli (like winning a bet; Cyders et al., 2007) and negative urgency as rash responses to negative affect or aversive stimuli (d'Acremont & Van der Linden, 2007). As will be discussed below, this scale addresses the important point that impulsivity can be increased in manic, depressive, or anxious states.

BEHAVIORAL CONSTRUCTS RELATED

TO IMPULSIVITY AND AFFECT

Impulsivity and Decision Making

Impulsivity can short-circuit conscious decision making, but impaired decision making could be related to insensitivity to the consequences of behavior in a manner related to impulsivity (Bechara et al., 1997). This is important in terms of impulsivity and affect because the affective state can strongly bias decision making (Bubier & Drabick, 2008; Franken et al., 2008; Moffoot et al., 1994). The Iowa Gambling Task (IGT) measures the ability to learn to make advantageous choices relative to shifting contingencies (Bechara et al., 1997). Performance on the IGT is impaired in substance

use disorders and with lesions of the ventromedial prefrontal cortex (Bechara et al., 2001). Performance on the IGT correlates with BIS-11 impulsivity scores in minimally symptomatic subjects with bipolar disorder (Christodoulou et al., 2006) but is not increased during mania (Clark et al., 2001). Decision making, as measured by the IGT, may therefore be at least partially independent of, and possibly complementary to, impulsivity.

Impulsivity and Venturesomeness

Impulsivity is an action-oriented trait related to increased stimulus orientation (Dickman, 1985). Constructs like venturesomeness and sensation seeking may share this characteristic; the combination has been formulated as a *supertrait* (Carrillo-de-la-Pena MT, 1992; Zuckerman, 1991) in the risk for potentially destructive patterns of behavior, including aggression, suicide attempts, substance use disorders, and impulse control disorders (Carrillo-de-la-Pena MT, 1992; Zuckerman, 1979, 1991). Impulsivity is related to preattentional responses to stimuli, however, while venturesomeness and similar constructs are more closely related to planful action (Barratt et al., 1997). This distinction may have clinical and physiological consequences (Magid et al., 2007). However, exaggerated responses to positive or negative stimuli, as occur in affective disorder, probably have both preattentional and attentional aspects.

Operational Definition of Affect

Affect is the experience of mood and can be viewed as the integrated experience of the current state of the central nervous system. We will classify affects as mania, depression, anxiety, and mixed states. Affect results from, and influences, basic behavioral processes including motivation, reward, and attention (Carroll, 1983; van Praag et al., 1990). Symptomatic affective states occur in the context of a course of illness defined by previous episodes and exposure to stressors or drugs of abuse (Post et al., 1986).

Common Mechanisms between Affect and Impulsivity

Impulsivity results from a failure in the balance between the generation and inhibition of action. The likelihood of impulsive behavior can therefore be influenced by many factors. Examples are arousal, motivation, and reward.

AROUSAL AND ATTENTION

Impulsivity is closely related to arousal and attention (Barratt & Patton, 1983). Efficient initiation of

action requires the ability to screen the environmental context of stimuli. Failure to accomplish this effectively increases the likelihood of rash, impulsive acts. Therefore, disruption of attention increases impulsivity, and impulsivity is prominent in disorders of attention (Johansson et al., 2005).

Either over- or underarousal can lead to impulsive behavior (Arnsten, 2007). Increases in impulsive behavior and cognitive impairment that occur with overstimulation or with excessively complex stimuli are exaggerated in psychiatric disorders in which impulsivity is increased (Shishida et al., 2006). Underarousal can also increase the likelihood of impulsive behavior, apparently due to compensatory efforts to increase sensory stimulation (Eckhoff et al., 2009). Affective disorders are characterized by increased and poorly modulated arousal (Fish et al., 2002). Patients with bipolar disorder, especially when in manic or mixed states, are particularly susceptible to overstimulation.

Environmental factors can increase impulsivity in patients with affective disorders. Environmental overstimulation or time pressure can impair response inhibition, especially in susceptible individuals (Chen et al., 2008). Sleep deprivation was reported to increase risk-taking behavior (Acheson et al., 2007). Acute stressors increase impulsive task performance (Chen et al., 2008; White et al., 2009); in addition, severe stressors in the past, common in affective disorders, may increase impulsivity throughout life (Garno et al., 2005b). These effects are mediated at least partially through poorly modulated catecholamine system function (Arnsten, 2000).

MOTIVATION

Impulsive acts may be drive-related and apparently goal-directed (Dickman, 1990). However, unlike more adaptive motivated behavior, impulsive acts occur without the opportunity to reflect on the goal involved or the means for achieving it. Goal-directed behavior is a result of a balance between activation and inhibition (Barratt & Patton, 1983). Impulsivity can result from a relative failure of behavioral inhibition (Evenden, 2000), resulting in essentially unopposed motivation.

Not all affectively influenced motivated behavior is impulsive. For example, in mania, impulsive behavior is increased (Swann, 2009), but some of the most troublesome behavior in manic episodes is planned behavior carried out in response to grandiose or paranoid cognitive distortions (Janowsky et al., 1974).

REWARD

Normal behavior, in all animals, has evolved to affect a balance between the need to wait for an optimal reward and the need to obtain a reward while it is needed or still available. The result is a phenomenon called *delay discounting*, in which the apparent value of a reward decreases as a hyperbolic function of time; exaggerated discounting is considered a form of impulsivity (Evenden, 2000; Stephens, 2002; Williams & Dayan, 2005). Impulsivity may also be a component of excessive reward-seeking behavior. One example is the addiction-like behavior of extreme risk seekers, who appear to use risk as a way to combat anhedonia (Franken et al., 2006).

Longitudinal Mechanisms

DEVELOPMENT

Changes in impulsivity occur over normal development. In general, activating behavioral mechanisms develop before inhibitory mechanisms do, beginning in the preschool years (Bell & Deater-Deckard, 2007; Kerr & Zelazo, 2004). For example, gamma aminobutyric acid (GABA) and serotonergic systems undergo active and experience-dependent development throughout childhood (Palomo et al., 2004). Neural inhibitory pathways involving the prefrontal cortex continue to develop throughout the late teens and early 20s (Olson et al., 2009). These processes are influenced by genetic characteristics (Kinnally et al., 2009) interacting with environmental factors, notably severe early stressors (Kinnally et al., 2009; McEwen, 2003). Early manifestations of psychiatric disorders and exposure (prescribed or nonprescribed) to pharmacological agents also have long-lasting consequences (Garno et al., 2005b).

BEHAVIORAL SENSITIZATION

Repeated exposure to stimuli that increase central catecholaminergic function leads to enhanced behavioral and cognitive responses to these stimuli (Robinson & Becker, 1986). Examples are stressors (Brake et al., 1997) and stimulants (Bonate et al., 1997a; Gaytan et al., 2000). There is cross-sensitization across stimulants (Bonate et al., 1997b; Yang et al., 2003) and between stimulants and behavioral stressors (Kita et al., 1999; Pacchioni et al., 2002; Phillips et al., 1997). In addition to exogenous stimuli such as stressors or stimulants, similar responses, and apparently sensitization (Gresch et al., 1994), can be elicited by endogenous activation of the same systems, such as manic or mixed states (Swann et al., 1987, 1994).

Behavioral sensitization is a complex phenomenon involving many systems that are basic to the regulation of affect. These include involvement of dopaminergic (Vanderschuren & Kalivas, 2000), noradrenergic (Drouin et al., 2002), corticotropin releasing factor (CRF; Cole et al., 1990), and excitatory amino acid (Wolf, 1998) systems.

Chronic stress leads to long-term changes in locus coeruleus function (Bremner et al., 1996a). Repeated stress activates the CRF–locus coeruleus system (Curtis et al., 1999). An apparent feed-forward loop of locus coeruleus and forebrain norepinephrine (NE) systems involves NE and CRF (Koob, 1999). Consistent with this and with behavioral sensitization, the response of the locus coeruleus to stress is enhanced by prior stressors without habituation (Sabban & Serova, 2007).

Animals with more behavioral impulsivity or spontaneous exploratory behavior appear more susceptible to behavioral sensitization (Alttoa et al., 2007; Anisman et al., 2000; Mitchell et al., 2006). Once sensitization has occurred, the animal is more impulsive (Paine et al., 2003). As will be discussed below, this relationship between behavioral sensitization and impulsivity appears analogous to the course and clinical characteristics of recurrent affective disorders (Post et al., 1986).

Behavioral sensitization to repeated amphetamine administration has been studied in healthy controls. Repeated amphetamine use increased the subjective response to subsequent amphetamine administration (Sax & Strakowski, 2001; Strakowski et al., 2001). In a positron emission tomography study that measured dopamine (DA) receptor occupancy by displacement of ^{11}C -raclopride, repeated amphetamine increased changes in DA receptor occupancy and psychomotor responses following subsequent acute amphetamine administration, especially in impulsive individuals (Boileau et al., 2006).

State-Dependent Aspects of Impulsivity and Affect

Neurotransmitter Systems in Affect and Impulsivity

Many of the same transmitter systems are involved in impulsivity and affect. In general, these systems do not have a unitary role in either impulsivity or affect but instead have biphasic or heterogeneous interactions, depending on receptor subtypes or location.

Impulsive behavior has been reported with elevated dopaminergic (Bergh et al., 1997; King et al., 1986) or noradrenergic function (Comings et al.,

2000; Gerra et al., 1999; Roy et al., 1988, 1989). Kindling (Anisman et al., 2000), or behavioral sensitization to catecholamines, whether through episodes of illness (Post, 2007), drugs of abuse (Yang et al., 2003), or stressors (Bremner et al., 1996b), may increase sensitivity to acute noradrenergic effects.

DOPAMINE AND IMPULSIVITY

Dopamine is related to the initiation of action and responses to rewarding stimuli (Wise & Rompre, 1989). It is increased in manic states but does not correlate with their severity (Swann et al., 1987). It has heterogeneous effects on prefrontal cortex function and interactions with NE, depending on the level of stimulation (Antelman & Caggiula, 1977). Dopaminergic stimulation may increase impulsivity (Evenden, 1998; Evenden & Ryan, 1996) due to its role in motivation and the initiation of action. Serotonin (5-HT) depletion in rats (Harrison et al., 1997; Mobini et al., 2000) or deletion of 5-HT receptors (Brunner & Hen, 1997) increases impulsivity only if DA is intact, so trait impulsivity may reflect a balance between DA and 5-HT.

NOREPINEPHRINE AND IMPULSIVITY

The locus coeruleus provides diffuse noradrenergic innervation to the cerebral cortex, limbic system, cerebellum, and spinal cord (Berridge & Waterhouse, 2003). It is activated by novel, unexpected, or noxious stimuli (Birnbaum et al., 1999; Koob, 1999; Singewald et al., 1999).

Noradrenergic activation can enhance attentive and self-monitoring functions of the prefrontal cortex via postsynaptic alpha-2A receptors (Arnsten & Li, 2005). However, at the higher levels associated with severe stressors or mania, noradrenergic stimulation impairs normally inhibitory functions of the amygdala (Braga et al., 2004) and prefrontal cortex (Arnsten, 2000) and contributes to behavioral sensitization (Drouin et al., 2002), largely through stimulation of alpha-1B receptors. Yohimbine increases rapid-response impulsivity in normal humans, parallel to increased behavioral activation (Swann et al., 2005a). These findings are consistent with parallel increases in impulsivity and NE reported in manic states (Swann et al., 1987, 2003) and with precipitation of hypomania by yohimbine in bipolar depressed subjects (Price et al., 1984).

SEROTONIN AND IMPULSIVITY

Serotonin has complex genetic and neurochemical regulation, far-ranging roles in affect and behavior,

and extensive interaction with other transmitter systems (Kreek et al., 2005). Studies measuring indices of 5-HT synthesis or turnover, or of 5-HT reduction by depletion of its precursor, tryptophan, show that 5-HT is involved in affect and social behavior, including increased dominance and reduced aggression (Young & Leyton, 2002). Turnover of 5-HT is reduced in patients with impulsive-aggressive or severe suicidal behavior (Linnoila et al., 1983; Virkkunen et al., 1995). Acute depletion of tryptophan results in increased rapid-response (Dougherty et al., 2007) and reward-delay (Schweighofer et al., 2008) impulsivity in healthy subjects, increased aggression in susceptible subject groups (Bjork et al., 1999; Booij et al., 2006), and relapse of symptoms in treated depressive or anxious subjects (Bell et al., 2001). Activation of presynaptic or postsynaptic 5HT1A and 5HT2 receptors can have either anti-impulsive or anxiolytic effects (Schreiber & De Vry, 1993). In nonhuman primates, low 5-HT metabolite levels are associated with increased impulsive behavior under naturalistic conditions (Mehlman et al., 1994).

Serotonin acts in balance with DA to oppose drive-related behavior. For example, as noted above, depletion of 5-HT in animals results in increased impulsive behavior, but only if DA systems are intact (Harrison et al., 1997).

ACETYLCHOLINE AND IMPULSIVITY

Acetylcholine (Ach) potentially influences affect and impulsivity via both muscarinic and nicotinic receptors. Cholinergic and noradrenergic systems may interact in the regulation of affect and in bipolar disorder (Janowsky et al., 1983; Yeomans et al., 2001). Activation of nicotinic receptors enhances attention and potentially reduces impulsivity (Dawkins et al., 2007; Levin & Simon, 1998); impulsivity predisposes to smoking, possibly as self-treatment (Dawkins et al., 2007; Mitchell, 1999). Smoking can also influence affect because nicotine stimulates DA and NE release (Brazell et al., 1991; Mitchell, 1993).

AMINO ACID TRANSMITTER SYSTEMS

AND IMPULSIVITY

Excitatory Amino Acids

Glutamate receptor systems facilitate stress-induced NE release (Singewald et al., 1995; Van Bockstaele, 1998; Van Gaalen et al., 1997) and have important roles in behavioral plasticity, including the role of both *N*-methyl-D-aspartate (NMDA) and alpha-amino-3-hydroxy-5-methyl-4-isoxazolepropionate

(AMPA) receptors in behavioral sensitization to stimulants and stressors (Bell & Kalivas, 1996; Sripada et al., 2001; Wolf, 1998).

Gamma Aminobutyric Acid

Gamma aminobutyric acid can reduce impulsivity by opposing stress-induced release of NE or DA (Gervasoni et al., 1998; Pudovkina et al., 2001; Singewald et al., 1995; Van Bockstaele, 1998). In excess, GABA can increase impulsivity through impaired attention (Shannon & Love, 2005).

Relationships between Impulsivity and Affective State

INTEGRATED MEASURES OF IMPULSIVITY

Questionnaires, including the BIS-11, measure multiple facets of trait-like impulsivity, described above. Another scale, the UPPS, is designed to measure urgency, sensation seeking, lack of premeditation, and lack of perseverance. *Urgency* is defined as acting in a driven manner, without reflection (Anestis et al., 2007). Urgency can be positive or negative, depending on the affective state (Cyders et al., 2007). This is consistent with the fact that NE release by the locus coeruleus is increased by either aversive or appetitive stimuli (Berridge & Waterhouse, 2003).

IMPULSIVITY AND MANIA

Impulsivity is a central component of manic behavior, predicts susceptibility to mania, and contributes to its behavioral complications like substance use disorders (Swann, 2009). Impulsive behavior is a requirement for manic episodes. Rapid-response impulsivity is increased in manic episodes (Strakowski et al., 2009; Swann et al., 2003) and correlates with severity of mania (Swann et al., 2001a), though these relationships are obscured if a substance use disorder is also present (Swann et al., 2004, 2008). Motor and attentional impulsivity on the BIS-11 are increased during manic episodes (Swann et al., 2008). Lack of longitudinal data makes it difficult to determine the extent to which these characteristics reflect properties of the individual having the episode rather than the episode itself.

In addition to being a component of manic behavior, impulsivity may predispose to mania. One study found a tendency to act before thinking (rapid-response impulsivity) and exaggerated rash behavioral responses to success (positive urgency; Cyders et al., 2007) in subjects at risk for bipolar disorder (Johnson & Jones, 2009). Impulsivity, affective

instability, hyperactivity, and increased energy, combined with either euphoria or dysphoria, were identified as premorbid symptoms in bipolar disorder (Gudiene et al., 2008).

Impulsivity in mania appears to be related to increased noradrenergic activity. Noradrenergic activity is increased during mania and correlates with the severity of manic symptoms (Swann et al., 1987). The exaggerated responses to success (Bijttebier et al., 2009) or to negative stimuli that are associated with impulsivity in manic episodes are consistent with enhanced physiological responses to noradrenergic stimulation (Koob, 1999; Singewald et al., 1999; Tanaka et al., 2000). The exaggerated response to success in mania resembles that in pathological gambling (Cummins et al., 2009). Like bipolar disorder, pathological gambling is associated with increased noradrenergic and dopaminergic function (Bergh et al., 1997; Roy et al., 1988); noradrenergic function is related to extraversion in pathological gambling (Roy et al., 1989).

Affective effects of noradrenergic stimulation depend on state and individual characteristics. Pharmacologically increasing noradrenergic activity increases rapid-response impulsivity and subjective activation in healthy subjects (Swann et al., 2005a); increases anxiety and somatic symptoms, with the potential for panic attacks, in subjects with major depressive or anxiety disorders (Albus et al., 1992; Guthrie et al., 1993; Heninger et al., 1988); and can precipitate hypomania in bipolar depressed subjects (Price et al., 1984).

IMPULSIVITY AND DEPRESSION

At first glance, one might not expect impulsivity to be increased during depression. However, impulsivity can be increased during either unipolar or bipolar depressive states, consistent with increased autonomic activation in these states (Koslow et al., 1983) and with negative urgency (d'Acremont & Van der Linden, 2007).

In unipolar major depressive episodes, impulsivity is expressed as increased negative urgency on the UPPS scale (d'Acremont & Van der Linden, 2007). Impulsivity in depressive episodes is associated with suicidal behavior (Corruble et al., 1999) and with loss of control, including elevated BIS-11 nonplanning and attentional impulsivity scores (Corruble et al., 2003a, 2003b). Impulsivity was associated with negative affect in “pre-symptomatic” depressed individuals (Palomo et al., 2008). In a prospective study of patients with major depressive

disorder, increased impulsivity predicted depression at follow-up (Grano et al., 2007).

In bipolar depressive disorder, impulsivity is related to depression in two ways. First, attentional and nonplanning impulsivity are increased in bipolar depressive episodes; these increases are associated with hopelessness, anhedonia, and suicidal ideation but not with subjective sadness (Swann et al., 2008). Second, when manic symptoms are present during bipolar depressive episodes, the severity of manic symptoms correlates with both rapid-response impulsivity and BIS-11 scores (Swann et al., 2007).

IMPULSIVITY AND ANXIETY

Anxiety is considered to be an inhibitory mechanism and therefore might not appear consistent with impulsivity. Anxiety can protect against impulsive behavior (Carver, 2005). However, anxiety can also be related to impulsivity, with increased scores for UPPS negative urgency (Taylor et al., 2008) and increased arousal (Cameron et al., 2000). In a sample of young adults, impulsivity correlated with depression scores only if anxiety was elevated (Farmer, 1998). These findings are consistent with relationships between impulsivity and activation associated with increased noradrenergic function (Swann et al., 2005a). In bipolar disorder, anxiety is highly heritable (Contreras et al., 2009) and is related to impulsive behavior (Taylor et al., 2008) and suicide risk (Apter et al., 1993). Barratt and his coworkers described anxiety and impulsivity as orthogonal; individuals in whom both anxiety and impulsivity were elevated had more severe general psychopathology than other subjects (Barratt, 1967; Barratt & White, 1969).

IMPULSIVITY AND AFFECTIVE LABILITY

Impulsivity may be more closely related to affective lability than to the severity of any specific affective state. Most studies of this association involved subjects with personality disorders or eating disorders rather than primary affective disorders. Affective instability predicted impulsivity in bulimia nervosa (Anestis et al., 2009). Affective instability and intensity were related to impulsivity in borderline personality disorder (Herpertz et al., 1997). Affective lability correlated with impulsivity in suicide attempters with borderline personality disorder (Links et al., 2000). Decision-making impairment correlated with affective instability, but not with impulsivity, in suicide attempters (Jollant et al., 2005, 2007), consistent with differences in the time course of impulsivity and decision making

(Barratt & Patton, 1983). In a nonclinical sample, affective instability and impulsivity appeared to be separate dimensions predicting borderline personality characteristics (Tragesser & Robinson, 2009). Taken together, these results suggest that impulsivity and affective instability have different mechanisms but have mutual predispositions based on increased activation or arousal.

IMPULSIVITY AND MIXED STATES

Mixed states, in the current diagnostic system, are defined as manic episodes in which syndromal major depression is also present for at least a week (American Psychiatric Association, 1995). Mixed states combine symptoms of mania, depression, and anxiety (Swann et al., 1986, 2009c). Clinically, it may be more useful to regard mixed states as a continuum, ranging from manic episodes with subsyndromal depression (Swann et al., 1997), to depressive episodes with subsyndromal mania (Swann et al., 2007), to combinations of full manic and major depressive states (Akiskal & Benazzi, 2004; Akiskal et al., 1998; McElroy et al., 1992; Swann et al., 2009c). Manic episodes with at least two depressive symptoms, or depressive episodes with at least two manic symptoms, already differ from pure manic or depressive episodes with respect to clinical characteristics, impulsivity, and course of illness (Akiskal et al., 1998; Swann et al., 1997, 2001b, 2007). Consistent with increased activation and impulsivity, noradrenergic activity is increased in mixed states (Swann et al., 1994). Behavioral characteristics of these patients are consistent with both positive and negative urgency. Impulsivity appears additive with depressive and manic states; subjects in mixed states have increased attentional, motor, and nonplanning impulsivity (Swann et al., 2008).

In manic episodes, mixed symptoms are most likely to include anhedonia, hopelessness, and suicidal ideation (Henry et al., 2003; Swann et al., 1986). Unlike other manic episodes, anxiety can be severe (Swann et al., 1986, 2009c). In depressive episodes, flight of ideas/racing thoughts, agitation/hyperactivity, and irritability are the most consistent hypomanic symptoms identifying mixed depressive states (Akiskal & Benazzi, 2004; Benazzi, 2003; Benazzi & Akiskal, 2006; Goldberg et al., 2009; Swann et al., 2009c).

Mixed States and Agitated Depression

Agitation can consist of excited goal-directed hyperactivity or painful inner tension (Akiskal & Benazzi, 2005; Swann et al., 1993). Excited goal-directed

hyperactivity occurs in all manic episodes (mixed or not) and painful inner tension occurs in all depressive episodes (mixed or not). Mixed states combine both kinds of agitation (Swann et al., 1993). Noradrenergic function is higher in mixed states than in agitated depression without hypomanic symptoms (Swann et al., 1994). Early onset, risky behaviors, addictive disorders, and a family history of bipolar disorder are all increased in agitated, depressed patients who have hypomanic symptoms compared to those who do not (Akiskal et al., 2005; Maj et al., 2006; Zimmerman et al., 2009).

IMPULSIVITY AND SUICIDAL BEHAVIOR IN AFFECTIVE DISORDERS

General Conditions

Suicidal behavior is associated with a combination of the wish to die, represented by depression and especially by hopelessness, and behavioral activation, represented by impulsivity (Mann et al., 1999). Therefore, the combination of depression and activation/impulsivity carries a high risk for suicidal behavior.

Suicide attempts can be predominantly impulsive or premeditated, but they usually have components of both (Oquendo et al., 2004). In fact, due to the increased impulsivity associated with moderate doses of alcohol (Dougherty et al., 2008), individuals can essentially plan an impulsive suicide attempt by using alcohol to facilitate behavior that would otherwise have been inhibited (Powell et al., 2001).

Among medically severe suicide attempts, an epidemiological study found that approximately one-third were predominantly impulsive (Simon et al., 2001). A study of people who survived self-inflicted gunshot wounds found a similar proportion of impulsive attempts (Peterson et al., 1985). These results show that, while many predominantly impulsive suicide attempts may have low lethality, a substantial proportion of potentially lethal suicide attempts are impulsive. In both studies, predominantly impulsive suicide attempts differed from predominantly premeditated attempts with respect to (1) less severe depressive symptoms, (2) relatively trivial precipitants, and (3) greater likelihood of using a violent method, despite (4) less expectation or intention of death. The combination of the third and fourth features is consistent with the manner in which impulsive behavior cannot be conformed to the context or intent.

Despite having lower depression scores, impulsive suicide attempters were as likely to be hopeless

as nonimpulsive attempters were (Simon et al., 2001). This is consistent with characteristics of impulsivity in depressed individuals (Swann et al., 2008): (1) increased attentional impulsivity, associated with giving up easily in the face of even minor unexpected adversity, and (2) increased nonplanning impulsivity, associated with a lack of sense of the future (Patton et al., 1995).

Bipolar Disorder

In subjects with bipolar disorder, a history of a potentially lethal suicide attempt was associated with increased rapid-response impulsivity (Swann et al., 2005b). This relationship persisted with correction for affective state and history of addictive disorder. Interestingly, the suicide attempts themselves were not necessarily impulsive. This implies, as suggested above, that a more impulsive person is more likely to be able to go through with a planned suicide attempt.

The combination of depression and activation is dangerous in terms of suicidal behavior, and it can arise quickly. Depressive, manic, and anxious states are all associated with impulsivity, especially positive or negative urgency. Perhaps the most susceptible group of patients is those who are experiencing mixed states, whether predominantly manic or depressed (Swann et al., 2009c). As mixed states combine hopelessness with behavioral activation (Akiskal & Benazzi, 2005), they are associated with substantial risk for suicidal behavior (Balazs et al., 2006; Berk & Dodd, 2005; Dilsaver et al., 1995; Goldberg et al., 1998, 2009; Strakowski et al., 1996).

In manic patients, consistent with the results in impulsive suicide attempts described above, emergence of apparently minor depressive symptoms or adversity can result in an extreme behavioral response, whose severity may be increased by high state- and trait-related aggressiveness, leading to suicidal behavior (Dilsaver et al., 1995; Goldberg et al., 1998). Accordingly, Strakowski et al. (1996) reported a correlation between suicidality and depressive symptoms in manic patients.

In depressed patients, even minimal activation, including racing thoughts, hyperactivity, or mood lability, can overcome the threshold of inhibition that might normally protect the patient against suicide and increase the risk for suicidal behavior (Akiskal & Benazzi, 2005). This activation can arise from environmental stimulation, from the natural history of the illness (Bronisch et al., 2005;

Swann et al., 2005b), or from treatment with anti-depressive or other potentially activating agents (Colom et al., 2006). Clinicians must be aware that bipolar depressed patients who have not yet been diagnosed with bipolar disorder (because mania or hypomania has not yet occurred or been recognized) are susceptible to this activation (Akiskal et al., 2005; O'Donovan et al., 2007).

Behavioral activation by antidepressive agents is associated with history of a substance use disorder (Goldberg & Whiteside, 2002) and an unstable illness course (O'Donovan et al., 2007). This susceptibility to behavioral activation may be related to treatment-emergent suicidal behavior in mixed depressive patients (Berk & Dodd, 2005).

Trait-Dependent Aspects of Impulsivity and Affective Regulation

Impulsivity and the Course of Affective Disorders

Patients with bipolar disorder vary substantially in their course of illness. Early onset and more frequent episodes are associated with susceptibility to mixed states (Swann et al., 2007) and with resistance to lithium treatment (Duffy et al., 2002; Swann et al., 1997, 2002b). Impulsivity is potentially related to the long-term course of bipolar disorder, whether as a result of unstable illness or as an expression of biological factors predisposing to a severe course (Swann et al., 2007). Impulsivity may worsen the course of the illness by contributing to substance abuse (Swann et al., 2004) and nonadherence to the treatment regimen (Dunayevich et al., 2000). Early-onset or highly recurrent bipolar disorder is associated with substance use disorders, aggression, and suicide attempts (Cate Carter et al., 2003; Fergus et al., 2003; Grunebaum et al., 2006). The BIS-11 scores, rapid-response impulsivity, and reward-delay impulsivity were increased, regardless of the current clinical state or the pharmacological treatment, in subjects with early onset, many previous episodes, and substance or alcohol use disorders (Swann et al., 2009a, 2009b).

Impulsivity and Early Stressors in Bipolar Disorder

Patients with mood disorders who experienced early stressors have more severe symptoms of personality disorders, suicidal behavior, an unstable bipolar course, and substance use (Garno et al., 2005a, 2005b), consistent with behavioral sensitization (Brake et al., 1997) and increased impulsivity.

Behavioral Sensitization to Stimulants and Stressors: A Bridge between Trait and State?

These findings are consistent with a relationship linking impulsivity, affect, and behavioral sensitization, based on (1) noradrenergic stimulation increasing impulsivity and (2) repeated noradrenergic stimulation associated with frequent episodes of illness or exposure to stressors, manic or mixed episodes, or drugs of abuse. In bipolar disorder, impulsivity has a strong relationship to the course of the illness. The BIS-11 scores and rapid-response impulsivity are increased in subjects with early onset of illness and with many episodes of illness, especially manic or mixed episodes (Swann et al., 2007, 2009a, 2009b). Similar measures of impulsivity are also increased in subjects with history of substance or alcohol use disorders (Swann et al., 2004), mild central nervous system trauma (Swann et al., 2007), or suicide attempts (Swann et al., 2007, 2009a, 2009b), especially medically severe suicide attempts (Murnan et al., 2004). Mixed affective features are related to these same characteristics (Akiskal et al., 1998; Swann et al., 2007).

Conclusions

Impulsivity and affect are strongly interrelated in a manner that combines acute, context-sensitive mechanisms with long-term trait characteristics. Both are related to basic aspects of the initiation of action, including mechanisms of arousal, motivation, and reward. The strong relationship between impulsivity and stimulus reactivity results in a two-way relationship between impulsivity and affective state in which each can influence the other. An example is the construct of behavioral urgency, which can be associated with either positive or negative affects.

The neurobiology underlying relationships between impulsivity and affect is complex. The response to NE release by the locus coeruleus is an important component. In terms of acute ongoing behavior, this system responds to unexpected or intensive stimuli, regardless of their affective valence. In terms of the life course, locus coeruleus activation is involved in behavioral stimulation to stressors and stimulants.

Behavioral sensitization to stimulants and stressors bridges state- and trait-related aspects of impulsivity and affect. Direct evidence in animals and indirect evidence in humans links impulsivity and behavioral sensitization. Sensitization requires activation of the same catecholaminergic systems that are involved in context-dependent impulsivity,

responses to reward or stressors, and episodes of affective disorders. Affective and related psychiatric disorders may be related to increased susceptibility to behavioral sensitization. Animal experiments show that this susceptibility is associated with increased trait-like impulsivity; further, impulsive behaviors are likely to contribute to behavioral sensitization.

Clinically, impulsivity is increased in depression, mania, and anxiety. Consistent with behavioral sensitization, this increase is amplified by coexisting substance use disorders, early stressors, or a highly recurrent course of illness. Impulsivity contributes to potentially destructive affect-related behaviors, most notably suicidal behavior associated with combined hopelessness and activation. Clinical management of the important relationship between impulsivity and affect requires combined pharmacological and nonpharmacological strategies stabilizing responses to internal and external stimuli and reducing the recurrence of sensitizing stimuli or events.

Acknowledgements

This work was supported in part by the Pat R. Rutherford, Jr., chair in Psychiatry- and by NIH Grant R01 MH069944.

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Psychology of Impulsivity

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Abstract

Impulsive behavior is generally viewed as counterproductive by society, and individual differences in impulsivity have been found to be related to a number of socially relevant behaviors. Yet, there are times when acting quickly and without thinking may seem desirable, even adaptive. With the possible exception of intelligence, no other personality dimension or trait so broadly influences various areas of human endeavor: interpersonal relationships, education, fiscal responsibility, personal moral behavior, business ethics and entrepreneurship, aggression, and criminality. This chapter gives an overview of impulsivity from a personality theory perspective. Topics discussed include the historical development of the construct, the place of impulsivity in a broader personality theory, self-report and behavioral assessment, and the role of impulsiveness in impulse control disorders.

Keywords: impulse control, impulsivity self-report measures, behavioral disinhibition, nonplanning, Barratt, Eysenck, delayed discounting, impulsive personality

The notion of impulsivity and acting impulsively has become so common in our culture that it is now integrated into every element of our culture. The lyrics to the popular song “Impulsive” by the vocal group Wilson-Phillips capture the essence of impulsivity and its connotation of acting recklessly, without thinking and regard for consequences. In that song the troubadour sings about longing to be free from thinking of consequences, not wanting to overanalyze the situation, and wanting to just be impulsive and to fall into a romantic embrace. Expressing the same sentiment in a different context, Nike, the sportswear manufacturer, has a bumper sticker that reads: “Just do it!” Yet, while there are times when acting quickly and without thinking may seem desirable and adaptive, there are other times when such action is maladaptive. And therein lies the difficulty with the concept of impulsiveness. Is it desirable or not, behavior due to state or trait, does it constitute pathology, and is it even definable? These are questions that reach far

back in time, but in modern psychological literature *impulsivity* is defined as “a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others” (Moeller et al., 2001, p. 1784).

Plato addressed issues such as impulsive behavior when he wrote about what constitutes ethical behavior. He was not alone among ancient ethicists, as Socrates, Aristotle, and Epicurus, all ethicists and moralists, considered the topic of how to define ethical behavior. The assumption made by Plato was that drive, volition, and action were the result of encountering stimulation from without and thought from within regarding the nature of the stimulation. A balanced individual would demonstrate his fitness when rational behavior overcame the tendency to act impulsively (Bain, 2006). In the writings of Epicurus, one finds the Greek term *protēnoia*, meaning literally “primal thought” but translated as “impulse.” Socrates believed that leaders should be

drawn from the cohort of citizens who demonstrated training in athletics as well as scholarship and then further demonstrated emotional stability (McCown & DeSimone, 1993). These individuals would, of course, reflect the antithesis of impulsive individuals.

Impulsive behavior has been with us as a manifestation of the human condition since before the recording of human history. There are a number of instances of impulsive behavior cataloged in Genesis, one of the oldest preserved descriptions of human nature. Chief among them is the impulsive act of Lot's wife as she turned back to look at the destruction of Sodom. Acting in defiance of Yahweh, she was turned into a pillar of salt (Genesis 19:23). Later, in Numbers 35:22–25, the author, Moses, draws a distinction between someone who acts impulsively without enmity and without malice of forethought and one who acts out of anger and instrumentally. Those who act impulsively are not to be delivered into the hands of avengers. There are few references to impulsive behavior in the New Testament, but one of the most important comes from the Gospels of Matthew and Mark, where Jesus seemingly impulsively curses a barren fig tree (Matthew 21:18–19; Mark 12:12–14).

McCown and DeSimone (1993) observe that early Christians may not have addressed the concept of impulsiveness until Augustine in his *Confessions* (AD 354–430) and not again until the time of the Reformation because they believed in a corporeal devil and impulsive thoughts and behaviors were derivative of the influence of the devil. It was, of course, typical of this time for cognition, the mind, to be attributed to the action of spirits and it remained so until the seventeenth century, when William Willis and the Oxford Circle began to demystify the causes of behavior (Zimmer, 2004).

McCown and DeSimone (1993) provide an excellent history of the use of the English noun *impulse*. According to them, it came into use in the sixteenth century and derives from the Latin *impulsus*, the stem of which is *impellere*, “to move.” By the seventeenth century, *impulsive* came into use as a description of a consistent pattern of dysfunctional behaviors. John Knox, a Scottish clergyman, who was one of the founders of the Presbyterian denomination and a leader in the Reformation, was a believer in predestination and he, among others, thought that those who were not predestined for salvation came to their ruin by virtue of their susceptibility to impulsive behavior: “given over to

Impulse in every deed” and “bound over to the Evil one” (McCown & DeSimone, 1993, p. 8).

At the end of the nineteenth century Walter Dill Scott, an American student of psychology and a graduate of Illinois State Normal University, traveled to Germany to study with Wundt. He completed a dissertation at Leipzig on the psychology of impulses in 1900. In this dissertation, he sought to develop a definition of the concept of impulse and to divorce it from the theological and moral connotations it held outside of science. Scott went on to be recognized as one of the first applied psychologists and became professor and chair at Northwestern University as well as professor of advertising in the School of Commerce at Northwestern. He developed a number of tests to measure characteristics important in job applicants (Schultz & Schultz, 2004).

Philosophers in the twentieth century also dealt with impulsive behavior. For instance, John Dewey wrote that impulsive activity lacks conscious purpose and is not directed consciously toward a specific end. Dewey considered impulsive behavior as nearly reflexive in the sense that the behavior might achieve a purpose but that the person who displayed the behavior orchestrated the beginning of the behavior without due consideration of the end—acting quickly without thinking (Dewey, 1936).

Personality Theory

Hippocrates (460–377 BC) formulated one of the first theories of personality. He hypothesized that there were four humors associated with various body fluids and these resulted in temperaments based on which was more abundant: choleric (yellow bile), sanguine (blood), melancholic (black bile), and phlegmatic (phlegm; Adams, 1886). Of these, both the choleric and sanguine types had elements of impulsivity, the sanguine type having elements of gregariousness and extroversion and the choleric type having elements of easy anger and bad temper. Kant, working under the still powerful influence of both Hippocrates and Galen, came to believe that human personality could be divided into types (Wood, 2001). Modern medicine had not yet been born in the Oxford Circle (Zimmer, 2004), and it was therefore easy to adopt the four-humors prototype prevalent at the time. While the labels may have changed, the paradigm of naming dimensions of personality has not. In the late nineteenth century, theorists were still focused on clinical observation and idiographic approaches to personality. As the new century opened, theorists like Kraepelin,

Kahn, and Freud, and later Fenishel, recognized the importance of impulsivity in predicting the risk of mental illness. These theorists, while acute observers on the human condition and strategic thinkers in terms of nomenclature, had little scientific evidence to support most of their conclusions.

Modern personality theory can be traced to Wundt (1907), who began to think dimensionally about behavior. This approach, combined with correlational techniques developed by Spearman, a student of Wundt and a mentor to Cattell, became the first empirical and therefore nomothetic approach to personality. Spearman's (1904) work with intelligence began to lay the basis for classical test theory. His use of rudimentary factor analysis laid the foundation for others to make important empirical contributions to personality theory. Galton's notion that personality characteristics were so fundamental to human relationships that they would be encoded into language was also influential. Thurstone (1933) noted that adjectives he used to describe people could be collapsed into coherent categories. Allport and Odbert (1936) developed an extensive list of adjectives that are used to describe personality, and this list formed the basis for researchers like Catell and Tuples and Christal.

In the 1940s, Cattell began to use computational methods to analyze the list formulated by Allport and Odbert. After clustering items, he asked people to rate others using adjectives on the list. Using the new technique of factor analysis to analyze this list, Cattell (1946, 1957) formulated 16 independent dimensions that people used to classify each other. Tuples and Christal (1961) used young inductees in the U.S. Air Force who had reported for basic training at Lackland Air Force Base to systematically replicate the work of Cattell. Norman (1963) essentially replicated Cattell's finding and the 16 Personality Factors (16PF). Cattell's test, is still available and being used today (Conn & Rieke, 1994)). Cattell found that personality is hierarchical and consists of both "primary" (his 16 traits) and secondary or higher-order "secondary" traits. This line of research firmly established factor analysis as a method that could be used to discover and organize underlying personality characteristics. Contemporary notions of evolutionary psychology owe their existence to this line of thinking, in which evolutionarily important dimensions are used for judging others and are so critical to relationships that they are incorporated into language.

Tuples and Christal (1961) realized that the more numerous factors that first emerged from their

analysis would collapse into fewer factors, essentially congruent with those of Cattell, that grouped the larger set into five coherent higher-order or secondary trait domains as well. The field remained static for two decades until Goldberg (1981) independently took a language-based lexical approach, found individual factors approximating those of Cattell, and discovered that they collapsed into five molar or secondary factors. Goldberg coined the term *Big Five* to describe these factors. Shortly thereafter, Costa and McCrae published the Neuroticism-Extroversion-Openness Inventory (NEO-I) five-factor personality inventory (Costa & McCrae, 1985) and validated the five-factor model across a number of domains (McCrae & Costa, 1987).

What separates the development of the 16PF and the five-factor model of personality is that their development was entirely empirical and, rather than being based on the theoretical musings of any one theorist, their development depended on the structuralist views of the scientists who developed them. This approach would not have been possible without newly developed analytical methods using correlational data to define *factors* composed of related descriptive items. Contemporary views of personality as an expression of a mechanism to solve evolutionary problems related to interpersonal relationships predicts that these factors would be stable across cultures and perhaps different between the sexes, and this in fact seems to be the case (e.g., Costa et al., 2001). In fact, all five factors demonstrate roughly equal contributions from genetic and environmental sources of variance (with h^2 values between 0.42 and 0.57; Bouchard & McGue, 2003; Jang et al., 1996).

The five factors are Openness to Experience, Conscientiousness, Extraversion, Agreeableness, and Neuroticism. Openness to Experience characterizes people who are open to experience as intellectually curious, flexible, and holding unconventional beliefs. Those less open are more conventional and traditional and prefer what they know to new experiences. Conscientiousness describes people who are disciplined and achievement oriented. Conscientious individuals control their impulses, while those less conscientious are less strategic in their planning and are seen as less reliable. Extraverts are people who tend to seek stimulation and the company of others. Extraverted people are not shy and are perceived as being energetic and action-oriented. Introverts have a tendency to be more quiet, deliberate, and less social. They seem to need less stimulation for optimal arousal than extraverts. Agreeableness is a tendency

to be cooperative rather than agonistic in group settings. People who score high on agreeableness strive for social harmony and are more likely to be optimistic, considerate, helpful, and willing to compromise their interests in the service of group harmony. Those less agreeable are likely to be self-centered and less concerned with the welfare of others, and they can be unfriendly and uncooperative. Neuroticism indicates a high probability of experiencing negative emotions including anxiety and depression. These individuals are seen as emotionally vulnerable and reactive, making them less likely to cope well with life's stressful circumstances. Those who score low on Neuroticism are more likely to be calm and stable and less inclined to suffer from frequent negative affect. Impulsivity is one of two closely associated traits that make up the personality trait of Extraversion (McCrae & Costa, 1987).

Researchers have found these five factors to be a reliable outcome of factor analytic studies (e.g., Digman, 1990) and fairly stable in adulthood (McCrae & Costa, 1990) but subject to maturational effects as well (McCrae & Costa, 1990; Roberts & Mroczek, 2008; Srivastava et al., 2003). Although these factors seem to be reliable molar descriptors of personality and helpful as an organizing heuristic for the primary personality traits, they have not been proven to be exceptionally powerful for predicting specific behavior (e.g., Mershon & Gorsuch, 1988). Impulsivity is one of those primary traits that is linked to specific behavioral tendencies.

Impulsivity as a Trait

Although impulsivity is a component of the Big Five's Extraversion factor, two researchers are primarily responsible for today's understanding of impulsivity as an independent personality characteristic: Hans J. Eysenck and Ernest S. Barratt. It is important to keep in mind, however, that impulsivity itself is seen by both of these scientists as a higher-order personality variable itself composed of more primary components.

Hans J. Eysenck, like Cattell, is a structuralist theoretician who conceptualizes primary traits such as impulsivity, sociability, persistence, and activity as being grouped into higher-order factors such as Extraversion (E), Neuroticism (N), or Psychoticism (P; Eysenck, 1947; Eysenck & Eysenck, 1975, 1985). Note, however, that while Eysenck's approach to personality is similar to that of Cattell and Costa and McCrae, his ultimate structure for personality is quite different. While Cattell as well as Costa and McCrae provide empirical support for five major

personality factors, Eysenck provides analyses that favor only three. Over the years, Eysenck has been a critic of the Big Five (Eysenck, 1991, 1992a, 1992b). Regardless of his position on the larger dimensions of personality, because of his clinical background and interest in psychopathology, Eysenck has focused intently on impulsivity as a major determinant of behavior in general and abnormal behavior in particular.

Eysenck believes that impulsivity can be separated into at least two types, one related to dysfunctional impulsivity, as most often described, and one related to functional impulsivity that might better be named *spontaneity* (Eysenck, 1993a). Eysenck observes that impulsivity is increased by brain damage (in particular, damage to right frontal and orbitofrontal areas) and the ingestion of ethanol, both of which he believes diminish cortical arousal (by acting on the reticular activating system) and therefore decrease anxiety and subsequently behavioral inhibition (Eysenck, 1993a). At the same time, variables that increase cortical arousal, such as stimulant drugs, decrease impulsivity (Eysenck, 1963). So, for Eysenck, impulsivity in the narrow sense is properly viewed as a dysfunctional personality characteristic that results in defective behavioral inhibition and subsequent behavioral inefficiency or abnormality.

Eysenck argues that three levels of analysis are needed—subtraits, traits, and dimensions—in agreement with more global theorists such as Costa and McCrae and Cattell. Early in his career, Eysenck formulated, standardized, and used a personality instrument he called the Eysenck Personality Inventory (EPI; Eysenck & Eysenck, 1964). Within the factor structure of the EPI, impulsivity was a subfactor of Extraversion. Extraversion was considered a mixture of impulsivity and sociability, and Eysenck was more closely focused on inhibition or lack thereof. In these analyses, Eysenck depended heavily on analyses from data sets including hysterics. Later, however, as Eysenck began to develop his arousal theory, his data set came to include many more psychopaths. He changed the nature and content of his inventory and developed the Eysenck Personality Questionnaire (EPQ; Eysenck & Eysenck, 1975). The new formulation expanded his factor structure to include Psychoticism, and impulsivity was included in that new dimension rather than in Extraversion. That is the factor structure that endures today. As Eysenck and his colleagues worked, they developed a specific instrument for impulsivity.

Eysenck and other researchers (e.g., Barratt, Zuckerman, Dickman) have different perspectives on what subtraits compose impulsivity and what determines those subtraits. Over the course of more than a decade the Eysencks developed a series of impulsivity scales: the I₅, I₆, and I₇ (for a review of this developmental history, see Eysenck, 1993b). Throughout this developmental process, the Eysencks used these instruments to relate their evolving measurement and conceptualization to delinquency and criminality, and they experienced a modicum of success in this enterprise as they related the item clusters on these instruments to behavioral outcomes and to other variables in their conceptual personality structure. The final instrument, the I₇, gives a measure of impulsivity that they claim loads on both P and N of their personality structure and is a measure of pathology, dysfunctional impulsive behavior, that predicts difficulty in life and problems in living, including delinquency and criminality (e.g., drug abuse, psychopathology, poor academic achievement, and attrition from psychotherapy; Eysenck, 1993b). They are not alone in adopting this perspective, as the reader will see as this chapter progresses.

Using data from the I₇, for analysis Eysenck breaks down impulsivity into four subfactors or subtraits: Impulsivity Narrow, Risk-taking, Non-Planning, and Liveliness (Eysenck, 1993b; Eysenck & Eysenck, 1975). These subfactors show only moderate intercorrelations, but they clearly are related and far from orthogonal. While this scheme decomposes impulsivity into what the Eysencks believe are coherent and stable subtraits, empirical data support the notion that a more global measure of impulsivity achieved by obtaining a total impulsivity score better predicts dysfunction and psychopathology. Eysenck (1993b) cites Dickman's work, where Eysenck's Impulsivity Narrow correlates 0.73 with dysfunctional impulsivity. Eysenck has consistently been concerned with the notion that impulsivity seems to run in families, and he has attempted to investigate this observation and quantify it. Based on studies by Eysenck (Eaves et al., 1989; Eysenck, 1983), the heritability of Impulsiveness Narrow is 0.57 for males and 0.60 for females.

Clearly, the more narrow aspect of impulsivity, Impulsiveness Narrow, is the most predictive of delinquency and predictive of the relationship between extraversion and cognitive functioning (Dickman, 1993). As the reader will see from Barratt's work, it is often of marginal utility to separate impulsivity into subcomponents, as the overall

measure of impulsivity seems to predict cognitive deficits as well as psychopathology better than correlations with its subcomponents.

Ernest S. Barratt, early in his career, with a background in experimental/clinical psychology, was interested in anxiety and personality structure. He noticed a cluster of items that suggested an impulsivity trait, which consists of acting without thinking. Barratt conceptualized this lack of thinking as variability in cognitive performance born of variability of intraindividual inhibition while performing perceptual-motor or cognitive/learning tasks (Barratt, 1993). While teaching a laboratory course in clinical psychology, Barratt would give his students anxiety and impulsivity scale items from self-report inventories such as the Thurstone Temperament Schedule (Thurstone, 1953) and the Guilford-Zimmerman Temperament Survey (Guilford & Zimmerman, 1949) and correlate their responses with their performance on various psychomotor and cognitive laboratory tasks. His initial experiments confirmed the effect of anxiety on task performance and also convinced him that impulsivity was related to task performance as well. He also noticed that the impulsiveness and anxiety subscales had only low-order correlations with each other, and he theorized that these personality dimensions were orthogonal. He began to focus exclusively on the domain of impulsivity. The result was the development of his first impulsiveness scale (Barratt, 1959). He named his contribution the Barratt Impulsiveness Scale (BIS) and over the next five decades Barratt, in cooperation with colleagues, students, and postdoctoral fellows, would conduct research aimed at refinement of his definition and measurement of impulsivity and attempting to relate it to cognitive, behavioral, and psychophysiological variables.

Over a broad range of laboratory tasks, Barratt found that subjects who scored highest on impulsivity and low on anxiety performed most inefficiently on his tasks (e.g., Barratt, 1967). He further confirmed that impulsiveness was not significantly correlated with anxiety (Barratt, 1959, 1965, 1967). He found that poor performance was often related to task complexity and that high-impulsive subjects exhibited problems with planning (Barratt, 1967), response set, and accuracy of fine perceptual-motor performance (Barratt et al., 1981). Barratt also attempted to relate impulsiveness to biological and psychophysiological variables (Barratt, 1972; Barratt & Patton, 1983; Barratt et al., 1987). He often wondered if impulsiveness was merely correlated

with psychophysiological variables or if those variables were manifestations of processes that determined impulsive behavior (J. H. Patton, personal communication, 1980).

Ultimately, Barratt, like Eysenck, viewed impulsivity as a first-order personality trait that had a biological basis (Barratt & Patton, 1983). Barratt was intimately familiar with Eysenck's work and frequently cited Eysenck's publications as informing his own thought and work (see Barratt, 1993). However, Barratt did not participate in the larger debate between Five Factor advocates (Costa & McCrae, 1992) and their critics (Eysenck, 1991, 1992a; Zuckerman, 1992). Rather, Barratt focused

entirely on refinement of the BIS and its relationship with performance and psychophysiological variables. During his lengthy career, the BIS underwent constant revision as his conceptualization of impulsivity was shaped by his research and refined by factor analytic study. The developmental history of the instrument is presented in Table 21.1.

Earlier versions of the BIS, such as the BIS-6, contained more subscales relating to subcategories of impulsive behavior. For the BIS-6 there were six subscales: motor control, intraindividual variability, impulsive interests, risk taking, interpersonal relationships, and impulse control (Barratt, 1993). However, as Barratt continued to develop his theory

Table 21.1 Developmental History of the BIS

BIS Version	Reference	# of Items	Scoring	Subscales
1	Barratt (1959)	45 impulsiveness items	True / False	None
		35 "filler" items		
2	Barratt (1961)	?	True / False	Lack of Persistence
				Social Optimism
				Lack of Motor Inhibition
				Aggression – Autonomy
				Action Oriented
3	Unpublished	?	True / False	?
4	Unpublished	?	True / False	?
5	Barratt (1965)	26 items	True / False	Speed of Cognitive Response
				Impulsiveness
				Adventure Seeking (Extraversion)
				Risk Taking
6	Barratt (1972)	?	True / False	Motor Control
				Inter-Individual Variability
				Impulsive Interest
				Risk Taking
				Interpersonal Relationships
				Impulse Control

(continued)

Table 21.1 Developmental History of the BIS (continued)

BIS Version	Reference	# of Items	Scoring	Subscales
7A	Discussed in Barratt & Patton (1983)	48 items	Rarely/Never	?
			Occasionally	
			Often	
			Usually	
7B	Barratt et al. (1981)	48 items	Rarely/Never	Sensory Stimulation
			Occasionally	Motor Impulsivity
			Often	Interpersonal Behavior
			Usually	Self-Assessment of Impulsiveness
				Risk Taking
8	Barratt & Patton (1983)	48 items	Rarely/Never	?
			Occasionally	
			Often	
			Usually	
10	Barratt (1985b)	34 items	Rarely/Never	Motor Impulsiveness
			Occasionally	Cognitive Impulsiveness
			Often	Nonplanning Impulsiveness
				Almost Always/Always
11	Patton et al. (1995)	30 items	Rarely/Never	Attentional Impulsiveness
			Occasionally	Motor Impulsiveness
			Often	Nonplanning Impulsiveness
				Almost Always/Always

Note. The BIS has been revised 11 times, counting both versions 7A and 7B; there was, however, never a BIS-9.

of impulsiveness, he became more and more convinced that in addition to failure to plan ahead, impulsive people have a fast cognitive tempo and rapidity of thought (Barratt, 1985a). In this regard, Barratt falls into the category of researchers who believe that impulsivity is reflective of making decisions too quickly and therefore often in error. Barratt conceptualized impulsivity as being part of an action-oriented personality that included extraversion, sensation seeking, and lack of inhibition that is separate from and orthogonal to anxiety per se. Behaviors associated with this personality would be response speed, risk taking, acting without thinking, and lack of planning. Barratt provides his clearest

statement and his outline of this theoretical position in his chapter in Zuckerman's book on the biology of sensation seeking (Barratt & Patton, 1983).

Late in his career, Barratt focused on three subtraits of impulsiveness: cognitive impulsiveness, motor impulsiveness, and nonplanning impulsiveness. Thirty-four items purporting to measure these three subtraits composed the BIS-10 (Barratt, 1985b). Speed of cognitive decision making was captured by "cognitive impulsiveness," acting without thinking was conceptualized as "motor impulsiveness," and "nonplanning" impulsiveness referred to acting without forethought. A number of studies have consistently demonstrated and replicated this

three-subtrait structure (Luengo et al., 1991; Miller et al., 2004; Parker et al., 1993; Patton et al., 1995). Gerbing et al. (1987) administered 373 impulsivity items from the BIS-8 and BIS-10, Eysenck's I_s and I_d, the 16PF, the Minnesota Multiphasic Personality Interview (MMPI), the Guilford-Zimmerman Temperament Scale, the Personality Research Form, and Buss and Plomin's EASI-II (Emotionality, Activity, Sociability and Impulsivity Temperament Survey) to 379 undergraduate students and submitted the results to a factor analytic analysis. The analysis yielded 15 factors that were submitted to a multiple-groups confirmatory factor analysis. This analysis revealed clear evidence of second-order factors corresponding to Barratt's Motor and Nonplanning Impulsiveness. The third factor contained items that related to cognitive activity, but it was not precisely the same as the Cognitive Impulsiveness subtrait Barratt had originally conceptualized. The Gerbing et al. (1987) study also included a battery of laboratory measures of impulsiveness. None of these measures were related to the self-report data in any reliable way. By and large, then, the Gerbing et al. study added confirmatory weight to Barratt's conception that impulsivity was multifactorial and that the major factors related to acting hastily and failing to plan for the future, while there was a third factor that was related to cognitive impulsiveness but not as Barratt had originally conceived it. In this study, Barratt's Motor Impulsiveness appeared closely related to Eysenck's Impulsiveness Narrow (Eysenck & Eysenck, 1977).

Barratt made one final attempt to revise and refine the BIS (Patton et al., 1995). In this study, the BIS-10 was administered to 412 college undergraduates, 248 psychiatric inpatients, and 73 male prison inmates. An exploratory principal components analysis identified six primary factors and three second-order factors. The three second-order factors were labeled Attentional Impulsiveness, Motor Impulsiveness, and Nonplanning Impulsiveness. Two of the three, Motor Impulsiveness and Nonplanning Impulsiveness, were consistent with earlier research (Barratt, 1985; Luengo et al., 1991), but cognitive impulsiveness was not identified in this analysis. Attentional Impulsiveness was not consistent with cognitive impulsiveness, whose items loaded on all of the factors. The second-order factors Motor Impulsiveness and Nonplanning Impulsiveness seemed similar to the Eysencks' (Eysenck & Eysenck, 1977) impulsiveness subtraits of Impulsiveness Narrow and Nonplanning. The three factors had significant intercorrelations, which suggests that the

instrument is a measure of a general impulsivity trait. The BIS was reduced to the 30 items that loaded on the second-order factors, and this instrument was labeled the BIS-11. The total score for impulsiveness, obtained by adding the subscale scores, was found to be a very consistent measure of overall impulsiveness and had significant utility for measuring impulsiveness among patient and inmate populations.

Stanford et al. (2009) state that the BIS-11 is arguably the most commonly administered self-report measure for the assessment of impulsiveness in both research and clinical settings. Stanford and his colleagues gathered new psychometric data using the BIS-11 and several other self-report measures of impulsivity to look at the validity and reliability of the BIS-11 factors and their relationship to other measures in a sample of 1577 adults. The sample included individuals from university populations and nonuniversity healthy adults from two geographically dispersed locations. As in the Gerbing et al. study (1987), a subset of these subjects also completed a battery of laboratory behavioral measures of impulsivity. Consistent with previous research, the BIS-11 is highly correlated with similar self-report measures of impulsiveness—that is, it exhibits convergent validity—but it is not significantly correlated with behavioral measures of impulsiveness (Barratt & Patton, 1983; Gerbing et al., 1987; Lane et al., 2003). Stanford and his colleagues reason that this failure to relate self-report measures of impulsiveness to behavioral measures is due to the fact that instruments like the BIS-11 assess stable personality traits that develop over long periods of time (i.e., they are trait measures), while behavioral measures depend heavily on state-dependent factors (Dougherty et al., 2003; Stanford, et al., 2009).

Between 1995 and November 2009 there were 689 citations of the BIS-11 paper (Patton et al., 1995). Clearly, the BIS-11 has generated considerable interest both as a research instrument and as a clinical measure. Therefore, the Stanford et al. (2009) paper included an update of normative data for classification of someone as "highly impulsive." The authors also observe that individuals who exhibit high levels of impulsiveness also demonstrate more aggression, greater variability of performance on tasks, a faster conceptual tempo, and physiological differences indicative of a low baseline arousal level. Additionally, college students who scored in the high impulsiveness range were more than twice as likely as nonscorers in this range to

have shoplifted an item over \$10 in value and more than twice as likely to have been involved in self-mutilation. Finally, the Stanford et al. paper included a fairly diverse review of the literature, examining studies that have used the BIS-11 in clinical as well as a number of nonclinical populations.

Criticisms of Self-report Measures of Impulsivity

Aside from the criticism that at times the different self-report measures do not correlate well with each other and therefore lack convergent validity, the other major criticism is that the self-report measures do not predict divergent performance on behavioral tasks. Dickman (1990, 1993) took a cognitive approach to impulsiveness. He studied impulsiveness within the context of a complex card-sorting task in which subjects were asked to perform under different conditions and time constraints. His research led to his proposal of two types of impulsiveness, functional and dysfunctional. Dickman characterized both types by a tendency to act with relatively little forethought. The difference is that functionally impulsive individuals act without thinking because they have been reinforced for doing so. In Dickman's card-sorting task, subjects were required to either sort cards containing complex stimuli rapidly, with minimal regard for the number of errors, or to sort the same stimulus cards with a high degree of accuracy (1990). When subjects are required to sort rapidly with minimal penalty for error, the number of correct responses may exceed the number of incorrect responses and overall, the outcome may be positive and compensate for the errors. Within this framework, quick responding could be viewed as adaptive; while errors were made, the overall outcome was positive. This view may relate to Barratt's notion of a response set (Barratt & Patton, 1993).

Using his cognitive perspective, Dickman (1993) also criticizes both Eysenck's and Barratt's self-report-based trait theories of impulsiveness for not producing behavioral data that are consonant with their theoretical underpinnings and assumptions. Eysenck's theory is based on arousal, and Dickman criticizes him because multiple studies of the effects of arousal on performance do not correlate with the behavior of high- and low-impulsive subjects. Dickman observes that Barratt's theory is based on subjects' individual differences in cognitive tempo (e.g., Barratt & Patton, 1983). Yet, Dickman observes that data from a number of studies using reaction time tasks and rhythmic timing tasks do

not support Barratt's hypothesis (1993). This was especially the case when tasks became complex.

Therefore, while Dickman acknowledges that there is some agreement among theorists such as Eysenck and Barratt regarding the underlying nature of the traits derived from self-report measures of impulsivity, he is critical of the ability of these trait differences to predict performance on laboratory tasks. In response, Dickman proposes an attentional model of impulsiveness (Dickman, 1993). Dickman observes that tasks that are either very simple or very familiar do not discriminate between high- and low-impulsive subjects, nor does impulsiveness reliably discriminate the ability to solve complex problems. As an alternative, Dickman proposes that high and low impulsives will only differ on tasks that are especially demanding in terms of requiring intense focus, where low-impulsive subjects will excel, or on tasks requiring shifting attention, where high-impulsive subjects will excel. The key here is that impulsivity demands more than just attention to arousal and cognitive tempo. Rather, predicting performance differences requires understanding the nature of the cognitive task in terms of sustained attention. Other researchers have used a different laboratory approach to investigating differences in decision making among high- and low-impulsiveness subjects.

Swann and colleagues (2002) define two dominant models of impulsivity as related to psychiatric disorders: the rapid-response model, where impulsivity is defined as responding quickly without adequate assessment of the context or consequences (similar to Barratt's Motor Impulsivity and Eysenck's Impulsivity Narrow), and the reward-discounting model, where impulsivity is defined as the inability to wait for a larger reward (similar to Barratt's Nonplanning Impulsiveness and Eysenck's non-planning). Swann and his colleagues have used two types of task: a continuous performance task, the Immediate Memory-Delayed Memory Task (rapid-response model), and tasks where subjects were asked to choose between a smaller immediate reward and a larger delayed reward (reward-discounting model). Swann and his colleagues found that errors of commission on the rapid-response measure correlated with BIS-11 scores, while reward-delay measures did not correlate very strongly (Swann, et al., 2002). These authors also found that the rapid-response tasks also correlated with the presence of an Axis I or Axis II diagnosis and with symptoms of personality disorder. This confirms earlier research where high impulsiveness scores on the BIS predict

psychopathology. Swann and his colleagues reasoned that the rapid-response tests are designed to measure the tendency to act without assessing the context and consequence, and this type of measure correlates more highly with trait-type impulsivity. They also reasoned that humans may be more amenable to developing strategies that circumvent or defeat reward-discounting tasks. Swann et al. also noted that their attention score was more strongly related to the presence of an Axis I or Axis II diagnosis and this, to some degree, affirms Dickman's assertion concerning the importance of attention.

In a separate study, Dougherty and his colleagues (2009) restate a definition of impulsivity proposed earlier—"a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others"—and criticize experiments that measure only limited behavioral approaches to the laboratory measurement of impulsivity (Moeller et al., 2001, p. 1784). Dougherty proposes a three-component model that includes response initiation, response inhibition, and consequence sensitivity. He and his colleagues then suggest using continuous performance tasks to require subjects to respond selectively to particular categories of stimuli in order to measure rapid but incorrect responding as well as the ability to respond to a stop signal as a measure of failed inhibition. Finally, they suggest reward or delay-discounting tasks for the assessment of preference for smaller-sooner over larger-later rewards. Using his own paradigm, Dougherty examined the effect of ethanol and marijuana ingestion on these tasks. Using factor analytic techniques, he and his colleagues found that performance on these tasks did cluster into categories supporting his proposed model, with limited intercorrelation between the three aspects of the model. In this same article, he also reports the results of a recent study where ethanol produced degraded performance on the continuous performance task, the stop-signal task, and the delay-discounting task, while marijuana ingestion produced increased impulsive responding on the stop-signal task but not on the continuous performance task or the delay-discounting task.

Delay discounting has become a model for cognitive aspects of impulsivity. Madden and Bickel (2010) have produced a monograph detailing delay-discounting paradigms and their application to risk taking, substance abuse disorders, health decision making, attention deficit/hyperactivity disorder, and the implications of this work for theories of

impulsivity. A detailed review of this field is beyond the scope of this chapter, but it is important to realize that impulsivity is not solely comprised of delay discounting and delay discounting does not in most instances correlate highly with trait measures of impulsivity. Part of the problem may be the artificial nature of many delay-discounting paradigms, where rewards are either imagined or trivial given the state of the subjects employed. What is clear is that there appears to be significant interest in developing new behavioral paradigms to measure impulsivity in the laboratory and that some of these paradigms seem to have significant promise, as they appear to relate to various forms of drug ingestion and psychopathology. This approach therefore holds the promise that it may not always be necessary to rely on self-report measures of impulsivity for assessing impulsiveness and informing clinical judgment.

Impulsivity and the DSM-IV-TR Impulse Control Disorders

As a personality trait normally distributed in the population, impulsivity may occur comorbidly with any psychiatric disorder (Moeller et al., 2001). Traditionally, the presence of heightened impulsiveness in a psychiatric patient has been viewed clinically as a sign of a more severe pathology and/or predictive of a poor treatment outcome (Matsunaga et al., 2005; Swann et al., 2009). The DSM-IV-TR (APA, 2000) lists impulsivity as a core characteristic or diagnostic criterion of several psychiatric disorders often referred to as *impulse control disorders* (Table 21.2). The DSM-IV-TR defines an impulse control disorder (ICD) as the failure to resist an impulse, a drive, or a temptation to engage in some harmful act. The impulsive behavior tends to be preceded by increasing tension or arousal and followed by pleasure, gratification, or relief (APA, 2000).

While the DSM-IV-TR definition above holds true for all the ICDs, even a cursory review of the clinical characteristics of these disorders suggests that they can be separated into two distinct subcategories. The first subcategory might be called *impulsive ICDs*. This would include Attention Deficit/Hyperactivity Disorder, Conduct Disorder, Substance Abuse/Dependence, Intermittent Explosive Disorder, Eating Disorders, Borderline Personality Disorder and Antisocial Personality Disorder (Table 21.2).

A majority of the clinical research on impulsivity to date has been done with the impulsive ICDs. Trait impulsiveness has been extensively assessed in each of these disordered groups using both self-report instruments and behavioral measures of

Table 21.2 DSM-IV-TR ICDs

Impulsive
Attention Deficit/Hyperactivity Disorder
Conduct Disorder
Substance Abuse/Dependence
Intermittent Explosive Disorder
Eating Disorders
Borderline Personality Disorder
Antisocial Personality Disorder
Compulsive
Kleptomania
Pyromania
Pathological Gambling
Trichotillomania
Impulse-Control Disorder Not Otherwise Specified
Paraphilic disorders

response inhibition (e.g., go/no-go tasks). As would be expected, high scores on self-report measures of impulsivity as well as diminished response inhibition have been consistently demonstrated for all the impulsive ICDs (Chamberlain & Sahakian, 2007; Moeller et al., 2001; Stanford et al., 2009). Studies looking at the relationship between the comorbidity of these disorders and impulsivity have found a significant positive association, with higher impulsivity resulting in the presence of multiple ICDs in a single individual (Kennedy & Grubin, 1990; Lacey & Evans, 1986; Stanford & Barratt, 1992). It has been suggested that the impulsive ICDs are part of an externalizing spectrum of psychopathology that is linked etiologically by an underlying dysfunction in general behavioral control (Krueger et al., 2005). Clinically, the comorbid diagnosis of any of these disorders in a psychiatric patient is often seen as evidence for heightened impulsiveness.

The second subcategory of the impulse control disorders could be labeled *compulsive ICDs*. This would include Kleptomania, Pyromania, Pathological Gambling, Trichotillomania, Impulse Control Disorder NOS (e.g., Compulsive Sexual Behavior, Compulsive Buying), and the Paraphilic disorders. Unlike the impulsive ICDs, which show a general deficit in impulse control, the failure to resist impulses in this set of disorders is highly compulsive and ritualized.

Published studies of trait impulsivity in the compulsive ICDs are limited, with the exception of pathological gambling, for which there is a wealth of research. While few in number, the results of self-report and response inhibition studies of the compulsive ICDs have consistently shown high trait impulsivity in this clinical population (Bayle et al., 2003; Billieux et al., 2008; Chamberlain et al., 2006; Maccallum et al., 2007). As in the impulsive ICDs, high trait impulsivity appears to be a core feature of the compulsive ICDs despite the compulsive nature of the problem behavior. The compulsive ICDs are thought to be part of an obsessive-compulsive spectrum of disorders and etiologically related to obsessive-compulsive disorder (Rossi, 2006).

Treating Impulsivity

Heightened trait impulsiveness appears to be a preexisting vulnerability marker for both the impulsive and compulsive ICDs (Verdejo-Garcia et al., 2007), possibly mediated by some underlying central serotonergic dysfunction (Stein et al., 1993). Pharmacotherapy for these disorders has tended to focus on two types of medications, selective serotonin reuptake inhibitors (SSRIs) and antiepileptic drugs (AEDs). Both types of drugs have been shown to be effective in the treatment of both impulsive and compulsive ICDs (Stein et al., 2006). More recent studies using naltrexone, an opioid antagonist shown to be effective in the treatment of certain substance use disorders and pathological gambling, have shown clinical efficacy across the ICDs (Grant & Potenza, 2007). Psychotherapeutic interventions that have proven effective in the treatment of impulsivity and the ICDs (both impulsive and compulsive ICDs) include cognitive-behavioral therapy (Oddyke & Olasov-Rothbaum, 1998) and dialectical behavioral therapy (Robins, 2002).

Concluding Remarks

Impulsive behavior has captured the attention of thinkers since the beginning of recorded history. Yet, after more than five decades of focused research, there is clearly more to be learned about both the place of impulsivity in a trait theory of personality and about the nature and expression of the trait itself. Interest in impulsivity is certain not to wane. With the possible exception of intelligence, no other personality dimension or trait so broadly influences various areas of human endeavor: interpersonal relationships, education, fiscal responsibility, personal moral behavior, business ethics and entrepreneurship,

aggression, and many others. Impulsivity clearly modulates the expression of other personality traits. Additionally, the individual impulsive acts of our fellow citizens continue on a steady basis to astonish us. How can such intelligent and competent people make such foolish and predictably destructive errors in personal judgment? How is it that the well-known politician comes to be involved with a member of his campaign staff or a student intern, leading to the destruction of his marriage and the loss of public trust? How can an accomplished and highly regarded investment advisor come to steal millions of dollars, thus impoverishing many of those who trusted him as well as, ultimately, his own family? Why does a particular young person choose to risk using a highly dependency producing drug, thus setting herself on a lifetime course of addiction? These and other questions relating to fundamental definitions, as well as the experiential and neurobiological antecedents of impulsivity, will occupy researchers and clinicians well into the foreseeable future.

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PART **4**

Assessment and
Treatment of
Impulse Control
Disorders

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Assessment and Treatment of Pathological Gambling

Hermano Tavares

Abstract

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

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

Introduction

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

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

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

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

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

PGambling Assessment

Screening for Problem Gambling and PG

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

The South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987) is a screening tool for PG based on the DSM-III criteria. Because it was one of the first gambling scales and is flexible in nature (it can be administered as a semistructured interview or as a self-report scale), the SOGS is still the most popular instrument for the assessment of problematic gambling behavior. It can be easily administered while patients are seated in the waiting room for a consultation. Lesieur and Blume (1993) revised the instrument for application in different settings, and

concluded that the initial questions about gambling preferences can be modified to suit the games available in a particular jurisdiction and that instructions may undergo slight modifications to cover specific time frames, such as the past 12 months.

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

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

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

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

A small fraction of pathological gamblers seek help for their gambling problems. It is estimated that between 7% and 10% of pathological gamblers have ever sought treatment for PG in North America (Suurvali et al., 2009). However, problem gambling is related to poor health, and the presence of problem and pathological gamblers in general practice settings is disproportionate to the frequency of PG in the general population (Pasternak & Fleming, 1999). This means that they do seek medical attention for other problems likely related to the distress caused by gambling and an unhealthy lifestyle (Morasco et al., 2006; Potenza et al., 2002). Thus, general practitioners (GPs) are well positioned to recognize and approach problem and pathological gamblers. Nevertheless, GPs rarely ask about betting habits and may not be comfortable with the idea of asking patients if they gamble "out of the blue." Tolchard and colleagues (2007) have emphasized the role of primary care professionals in early intervention to prevent a habit from escalating to a serious gambling problem. They pictured such intervention in a flowchart that begins with the

patient's report of stress-related complaints (headaches, depression, anxiety, and sleeping problems). This gives the GP an opportunity to ask about stressors, including alcohol and drug intake. In this context, questions about gambling will not seem awkward or misplaced. If the patient reports regular gambling, the GP can suggest applying the Early Intervention Gambling Health Test (EIGHT; Sullivan, 2000). The EIGHT is an eight-item self- or clinician-administered questionnaire with yes/no answers. Each positive answer adds 1 point to the score. If the score is 4 or more, gambling is affecting the patient's well-being and he or she should be referred for specialized treatment. If the score is below 4, the patient should receive information to raise his or her awareness of gambling risks and how to minimize them. The GP will then take a note to reassess the patient's gambling status at the next meeting.

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

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

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

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

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

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

to gambling. When investigating gambling harm in the elderly, clinicians should keep in mind the potential compromise between the use of retirement savings and the narrowing of leisure and coping strategies, which at the moment of assessment may not be perceived as a threat by the patient (Ladd et al., 2003).

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

Diagnosing PG and Psychiatric Comorbidities

In the DSM-IV-TR (APA, 2000) PG is classified as a disorder of impulse control, but its diagnostic criteria are modeled on substance addiction (Cusack et al., 1993). It is defined as recurrent maladaptive gambling behavior that is not better accounted for by a manic or hypomanic episode. In order to be diagnosed as a pathological gambler, the subject has to fulfill at least 5 out of the 10 following criteria:

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

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

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

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

4. “[I]s restless or irritable when attempting to cut down or stop gambling.” This phenomenon parallels the withdrawal symptoms seen in substance dependence. Recently, Cunningham-Williams and colleagues (2009) studied a sample of 312 gamblers from the community. They verified that restlessness and irritability were positively associated with gambling pathology. However, three additional withdrawal-like symptoms were experienced by 41% of the sample

when attempting to quit or control gambling: feelings of anger, guilt, and disappointment. This association held true even when the overlap with comorbid substance withdrawal and depressive symptoms were considered. Besides these emotional withdrawal phenomena, one study described several bodily ones such as insomnia, headaches, upset stomach, loss of appetite, physical weakness, racing heart, muscle aches, difficulty breathing, sweating, and chills among 65% of pathological gamblers attempting to stop gambling (Rosenthal & Lesieur, 1992).

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

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

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

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

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

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

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

As described above, the diagnosis of PG follows an addiction's general rationale. It is preferable to understand that this rationale that has not changed over the last 40 years (Edwards, 1986) than to memorize the DSM criteria for PG, which are often revised in the next edition of the DSM. The fitness of PG in the addiction model has been questioned, but so far, no alternative model has proved better (Tavares & Gentil, 2007). The addiction model predicts that the potentially addictive stimulus changes the individual's subjective state by altering brain activity, in general terms stimulating or depressing

neuronal activity. Gambling is regarded as a stimulating activity that in many ways can mimic the effects of stimulant abuse (Zack & Poulos, 2009b). When such action meets an individual's need to compensate for arousal that is either above or below the ideal level, and/or to restore a damaged self-image by means of temporary emotional empowerment and alienation, the contact with the stimulus is strongly reinforced (Brown, 1987; Jacobs, 1986). Recurrent exposure to the stimulus will cause two types of brain adaptation: compensatory mechanisms that lessen the stimulus impact in order to protect the brain activity and sensitization that prompts readily available behavioral responses to the stimulus and other related stimuli. The sensitization process establishes an association with environmental cueing signs, making the stimulus-seeking behavior progressively dominant in the individual's conduct.

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

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

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

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

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

social milieus of drinking and gambling, since both are nowadays legal and normative behaviors; alternatively, both addictions may share specific genetic and biological features yet to be discovered.

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

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

Psychopathology of PG and Assessment of Related Phenomena

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

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

of play even with continued loss (Young & Wohl, 2009).

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

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

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

But lack of reflection and time perspective may not fully account for bad decisions. Another possibility is that pathological gamblers observe gambling outcomes and jump to conclusions based on false cognitive premises. In the mid-1970s, researchers began to point out how chance events might influence cognitions. Langer (1975) demonstrated how a game as simple as coin tossing could yield an illusion of control by means of specific sequences of outcomes. Recently, Monaghan and colleagues (2009) found that undergraduates playing at an electronic gambling machine (EGM) developed irrational thoughts, erroneous perceptions of chance, and superstitious beliefs, depending on the outcome; those who lost showed less distorted thinking than those who won. Sharpe and Tarrier (1993) propose that cognitive distortions are the bridge between the conditioning processes at play in recurrent gambling. First, operant conditioning factors (winning, excitement, relief from a negative mood, etc.) prompt initial persistence at gambling. Through recurring betting, occasional winnings will be associated with events that occurred simultaneously or close in time (e.g., a certain hour of the day, a specific combination of betting numbers, or pressing the machine's button with the thumb). Such an association can be strengthened by a larger than usual financial reward or by a few chance repetitions that establish two kinds of distorted thinking (Toneatto & Gunaratne, 2009): primary illusion of control (the gambler thinks he or she can manipulate gambling and environmental features in order to produce the desired outcome) and secondary illusion of control (the gambler thinks he or she can read gambling and environmental cues in order to predict the outcome). These cognitive distortions foster unrealistic expectations of winning and more

investment in gambling. Finally, the frequent reexperience of the psychophysiological effects of gambling consolidates a classical conditioning process establishing environmental gambling cues that will trigger the cravings, as mentioned above. However, if such a model was infallible, any frequent gambler would have the misfortune of once being fortunate and experiencing a "lucky streak." Then, sooner or later, he or she would turn into a pathological gambler. The truth is that there are regular gamblers who will never develop significant gambling problems. Compared to social gamblers, problem gamblers develop more superstitious beliefs, expectancies of winning, and attribution biases (i.e., positive outcomes are due to the gambler's control and predictive abilities, while negative ones are due to chance or the undesired interference of others; Joukhador et al. 2003). Thus, the question remains whether cognitive distortions are the result of one's history of a gambling chain of reinforcements, or if there could be a trait predisposition to develop superstitions and a wishful pseudorational logic.

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

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

Natural History and Progress of PG

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

In the 1970s, Dr. Robert Custer established the first treatment unit for pathological gamblers at the Brecksville, Ohio, Veterans Administration Hospital (Custer & Milt, 1985). Based on his experience at that unit, he proposed a progressive course of PG. Gambling progression was divided into three stages: winning, losing, and desperation. Later, Rosenthal added a fourth stage: giving up (Lesieur & Rosenthal, 1991). Usually, a pathological gambler reports frequent wins at the beginning of his or her gambling career. In the case of Custer's patients, this could be explained partly by the fact that almost all of them were male, and many betted on horse races or card games in which experience and skill played a small part. Skill-based wins enhanced the enthusiasm for gambling and the gambler's confidence in his or her ability to predict or produce desired outcomes, thus boosting the betting that afterward would no longer follow any rationale. Another possibility is that pathological gamblers are bettors who experienced a big win or several small wins by sheer luck at the beginning of their gambling, predisposing them to increase their commitment to gambling (Lesieur, 1984).

Finally, losing or defeat may prevail in the gambler's early gambling career, but several small losses will not lead to unbearable debts in this initial stage and are likely to be shadowed by fewer but bigger wins, which will create a memory bias toward the gambler's luck and ability at wagering. However, PG is considered a heterogeneous disorder with at least two types of gamblers: the action seeker, who is more likely to be a male, and the escape seeker, who is more likely to be a female (Ledgerwood & Petry, 2006a). Escape gamblers are not oriented to winning, but rather relief and alienation, which again are more easily achieved in the initial stage of gambling, when financial losses have not accumulated to the point where worries start rivaling oblivion. In both cases, the typical pathological gambler will refer to the initial wagering as a profitable and/or pleasurable activity, with a sense of nostalgia and a desire to go back to the time when betting was a positive experience.

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

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

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

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

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

Not only is the diagnosis of PG unstable, but the diagnostic criteria also vary in nature. In a large

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

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

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

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

Besides gender, types of games have been related to the addictive potential of gambling and to faster progression, with the suggestion that games that sustain continuous arousal are more likely to provoke careless gambling. This can be achieved by associating gambling with an ongoing event in which the outcome is known at the end—the case of horse racing (Coventry & Norman, 1997)—or by shortening the time between placing the bet and observing the outcome, which allows faster repeated betting and continuous play—the case of EGMs (Dickerson & O'Connor, 2006). Indeed, in two studies, preference

for an EGM was associated with faster gambling progression (Breen & Zimmerman, 2002; Tavares et al., 2003a). Thus, female gamblers may be at particular risk for rapid progression to gambling problems because of factors related to gender itself and because they have a greater preference for EGMs than men.

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

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

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

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

Suurvali and colleagues (2010) conducted a review on motivations for ending gambling by oneself and with specific help. They surveyed 10 studies addressing reasons for quitting gambling. Help seekers differed from those who solved their own problem in that among help seekers, gambling-related harm (financial and relationship issues and negative emotions including hitting rock bottom) was the most frequent reason for trying to quit. Self-help gamblers more frequently reported that gambling became incompatible with recent changes in the environment or with their lifestyle. They were also more likely to stop by using self-appraisal and decision making. It is worth noting that treatment seekers are very much aware of the negative consequences of gambling, but they are unlikely to acknowledge the slim chances of making money through gambling and reaching a decision based on this evaluation.

This fact gives support to cognitive and motivational enhancement techniques applied in different gambling therapies and suggests that treatment is especially indicated for those pathological gamblers who may have biases in assessing the pros and cons of risk-taking behaviors that may compromise the decision-making process. Studies applying open-ended questions also identified incompatibility with self-image as a frequent reason to quit gambling (Hodgins et al., 2002; Toneatto et al., 2008). Indeed, during the first sessions when a patient is not entirely convinced of his or her desire or capacity to stop gambling, it is much more fruitful to focus on regaining self-control and restoring family ties as objectives shared by the therapist and patient, and then proceeding to the analysis of what is needed to achieve such goals. Usually, in time, gambling patients will realize that the image of the will-powered man or the caring, responsible woman they pursue is not compatible with gambling.

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

PG and Personality

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

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

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

Indeed, results from studies using different dimensional models of personality assessment display a fair amount of convergence. Usually problem and pathological gamblers have higher scores on dimensions representing negative emotionality and lower scores on dimensions representing constraint

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

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

PG and Related Medical Conditions

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

hepatic diseases. Since many of these diseases are caused by sexually transmitted viruses, it is feasible to speculate that impulsivity pervades other aspects of pathological gamblers' lives, including sexual behavior, which exposes them to further threats to physical health (Martins et al., 2004).

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

circumstances, and overwhelming financial or gambling losses.

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

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

PG and Psychosocial and Environmental Stressors

Pathological gambling is clearly influenced by contextual factors, however little is known about the impact of psychosocial and environmental stressors in gambling behavior. For instance, attendance, participation, and group and family support are all important factors in abstinence maintenance for members of GA (Oei & Gordon, 2008). Epidemiological surveys clearly correlate PG and indicators of loneliness (divorce, separation, widowhood, etc.), shallow community ties (migration, unemployment, etc.), financial burdens, and problems with the legal system (Blaszczynski & Silove, 1996; Mazzoleni et al., 2009; Petry et al., 2005). But the

causality direction in these cases is not yet defined. One study has investigated prior traumatic events and a lifetime history of PG (Scherrer et al., 2007) in a twin cohort. After controlling for psychiatric disorders, genetic factors, and family environmental influences, PG was still significantly associated with child abuse, child neglect, witnessing domestic violence, and physical attack. Moreover, genetic factors and family environment partly mediated the movement from trauma exposure to development of gambling symptoms. In adolescent gambling, the role of psychosocial factors is even more dramatic. In a sample of students from grades 7 to 13, it was found that problem gambling was associated with poor perceived family and peer support, substance misuse, conduct problems, family problems, and parental involvement in gambling and substance use (Harroon et al., 2004). For adolescents and other patients with limited autonomy, some degree of intervention to lessen environmental adversity is warranted (e.g., family or couples therapy, foster homes, day-hospital care, or other forms of extended therapeutic support).

PG, Associated Risky Behaviors, and Global Functioning

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

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

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

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

Treatment

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

and physical) and a good quality of life (Walker et al., 2006).

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

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

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

First Measures: Comorbidity, Impulsivity, and Craving Pharmacology

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

Currently, there is no medication approved by the U.S. Food and Drug Administration for the treatment of PG. But the psychopharmacology of impulsivity and gambling craving is a promising field. In a recent meta-analysis of pharmacological treatments for PG, Pallesen and colleagues (2007) described an overall effect size of 0.78. Cohen (1977) proposed that, for the behavioral sciences, effect sizes between 0.5 and 0.8 should be considered a medium

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

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

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

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

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

carried on by the same group (Grant et al., 2003). Sertraline was also tested in an RCT with negative results (Saiz-Ruiz et al., 2005). Citalopram (Zimmerman et al., 2002) and escitalopram (Black et al., 2007b; Grant & Potenza, 2006) were tested only in open-label studies with small samples; despite the preliminary auspicious results, they still await further validation.

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

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

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

Opiate antagonists are probably the best-studied class of drugs in PG treatment. Their use is based on the several similarities between PG, alcohol, and other substance addictions. Naltrexone is a mu-opioid receptor antagonist that modulates the release of dopamine in the ventral tegmental area/nucleus accumbens/medial orbitofrontal cortex circuit, the so-called brain reward system. It has proven efficacious in the treatment of alcohol dependence syndrome, mostly by reducing the reinforcing properties of drinking and the intensity of craving episodes (Volpicelli et al., 1992). Naltrexone was superior to placebo for the treatment of PG in two methodologically sound RCTs. The first study applied a flexible dose design (Kim et al., 2001). Interestingly, the mean dose prescribed was 187.5 mg/day, which is much larger than the dose prescribed for alcohol dependence (50 mg/day). The second study investigated the specific effect of three dose regimens (50, 100, and 150 mg/day) against placebo on gambling cravings (Grant et al., 2008a). All three naltrexone groups were superior to placebo, but no difference in craving reduction was found between them. Naltrexone is well tolerated and safe, provided that the patient takes no acetaminophen, aspirin, or other nonsteroidal anti-inflammatory drugs; otherwise, liver enzyme levels may become elevated (Kim et al., 2006). In the same line, Grant and colleagues (2006) conducted an RCT of nalmefene in PG.

Nalmefene is also a mu-opioid receptor antagonist. Compared to naltrexone, its advantages were a longer half-life, superior oral bioavailability, and no dose-dependent association with liver toxicity. It was also more effective than placebo, but although no direct comparison was carried out, in contrast to naltrexone, nalmefene was associated with more adverse effects (nausea, dizziness, and insomnia), and doses above 25 mg/day were hardly tolerated. Examining a combined sample from the three RCTs just described, Grant and colleagues (2008b) found that patients reported intense craving, and taking higher doses of an opioid antagonist was associated with a treatment response but only on a trend level, whereas a family history of alcoholism was a strong predictor of response. Nonetheless, a recent RCT for pathological gamblers demonstrating comorbidity with either alcohol abuse or dependence showed no significant action of naltrexone on gambling or drinking behavior (Toneatto et al., 2009). The fact that both groups received CBT counseling during the trial may have clouded potential differences, but it also underscores the importance of psychological treatments in PG. The majority of patients from both groups retained the treatment effects at the 1-year follow-up, demonstrating the strong impact of the therapeutic program.

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

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

First Measures: Psychoeducation, Motivation Enhancement, and Contingency Control

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

gambling in comparison to the control condition between baseline and week 6, relating to a significant decrease in gambling at month 9.

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

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

Pros (+) of Gambling	Cons (-) of Gambling
Pros (+) of Not Gambling	Cons (-) of Not Gambling

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

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

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

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

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

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

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

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

Early attempts from the mid-1960s to apply behavioral principals to PG treatment focused mostly on aversive conditioning methods, but case reports and small, uncontrolled studies failed to provide consistent evidence of their usefulness (Tavares et al., 2003b). Later, covert sensitization, imaginal desensitization, systematic desensitization, relaxation training, and alternative sources of satisfaction were described, either alone or in combination in multimodal behavioral treatments (Hodgins & Peden, 2008) in small case series. The best-studied behavioral technique so far is imaginal desensitization. In a quiet room, patients are taught gradual relaxation. Next, the therapist asks them to visualize a circumstance in which gambling is likely to occur, and then the therapist suggests that they avoid this situation and engage in alternative activity. The format of imaginal desensitization therapy has varied across studies, from 14 sessions delivered twice a day for 1 week in an inpatient program (McConaghy

et al., 1991) to 2 or 3 exposures to audiotaped imaginal exposure over 5 weeks (Grant et al., 2009). Three controlled trials compared imaginal desensitization to other techniques. The first study compared imaginal desensitization to aversion therapy (delivering an electric shock to the finger tips). Both therapies were considered effective 1 month after treatment, but only imaginal desensitization retained significant reduction of gambling (70%) 12 months after treatment (McConaghy et al., 1983). The second study compared imaginal desensitization to aversion therapy, imaginal relaxation, and in vivo exposure to gambling situations (McConaghy et al., 1991). Imaginal desensitization exceeded all other modalities, with close to an 80% response rate. Finally, in a recent study, Grant and colleagues (2009) compared imaginal desensitization combined with motivational, cognitive, and other behavioral techniques delivered in an 8-week program to GA referral. The combined program was significantly superior to GA referral, with a 64% abstinence rate at treatment completion. However, the multimodal format of the program precludes appreciation of the specific contribution of imaginal desensitization to the treatment outcome. The only other behavioral method tested in an RCT was in vivo exposure. In this technique, the patient is involved in sessions of progressive exposure to gambling-related stimuli, but only after a period of abstinence when stabilization has been obtained. The first sessions may include standing outside a gambling venue with no money while being coached by the therapist to prevent any behavioral response. The patient must feel the rise of anxiety and arousal, and the exposure can be terminated when both have clearly declined. The following sessions provide progressive exposure by manipulating environmental contingencies (outside/inside the gambling venue, back turned to the machine/front turned to the machine, with/without the therapist, and carrying no money/with money). Echeburúa and colleagues (1996) compared in vivo exposure only, cognitive restructuring, and a combination of both techniques to a waiting list control condition. At treatment completion, the exposure-only condition had a 69% abstinence rate compared to 38% for the other treatment groups and 25% for the control group. The problem, as in aversion therapy, was the steep decline in the abstinence rate a few months after treatment completion. In vivo exposure should be particularly considered when the environment precludes avoidance of gambling stimuli or when avoiding them would cost the patient a great deal of social seclusion. For instance, in some areas of Spain,

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

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

containing observations about response patterns; hints specific to each patient on how to deal with gambling urges; and, always, praise for any progress recorded. Often the patient comes eagerly to the session just to enjoy the pleasure of reading what the therapist wrote. Besides the journal review, it is advisable to supply handouts about how games of chance operate (including information on real probabilities of winning, negative rates of financial return, the house edge, etc.) and the usual cognitive distortions that come to mind when gambling. Time should be taken to discuss which topics in the handouts apply to the patient's actual experience with gambling.

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

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

Fig. 22.2 Weekly record of gambling urges.

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

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

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

Considering the pathological gambler's deficits in executive functions (i.e., control and planning) and vulnerability to negative affective states (anxiety and depression), almost all multimodal CBT programs include practice sessions on problem solving and affective coping methods. A detailed description of simple and efficient techniques for this purpose is presented in the cognitive-behavioral coping skills therapy manual by Kadden and colleagues (1995). With the aid of a five-question form, patients are guided through a stepped process in which they are expected to (1) recognize the problem; (2) describe its constituents; (3) formulate different approaches based on the previous appraisal; (4) balance the pros and cons of each option; and (5) evaluate the outcome of the chosen approach. For enhancement of affective coping, the therapist gives the patients a handout listing the different types of cognitive distortions that usually go along with anxiety and depression. These distortions are reviewed, and the patients are asked to provide examples related to gambling or other problems from their lives when appropriate. Then patients are

taught to identify and challenge the distortions in their own thinking. Kadden and colleagues have provided a set of 20 questions meant to guide the patients through this process. The questions aim at encouraging an unbiased evidence-based appraisal of the facts, avoiding exaggeration of consequences and "all-or-nothing" thinking (e.g., "What's the evidence? Would this thought hold up in a court of law or am I jumping to conclusions based on circumstantial evidence?").

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

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

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

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

In finishing this explanation of treatment methods for acute PG, it is important to reemphasize that the intensity of intervention has to match the level of gambling severity (Hodgins, 2005). Treatment must be tailored to the patient's needs. Severe gambling pathology will require a multimodal approach. Several components of the multimodal treatment packages for PG derive from clinical experience with other addictions (Tavares et al., 2003b). The field of gambling treatment is in dire need of dismantling studies, in which each component of multimodal approaches is tested in isolation so that its

specific contribution to treatment can be estimated, as well as for whom, when, and under what condition such methods are likely to ensure a positive outcome. Additionally, Pallesen and colleagues (2005) suggest that future clinical research concentrate on the selection of universal outcome measures and instruments to ease the comparison between treatment modalities. Meanwhile, clinicians will have to base treatment options on the patient's assessment, the methods they are acquainted with, and continuous reassessment to redirect treatment whenever needed.

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

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

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

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

Assessing Treatment Efficacy

The assessment of any therapeutic effort to treat PG is important for research and clinical purposes. Unfortunately, the field still lacks a consistent set of assessment tools that can reliably and comprehensively evaluate the effects of PG treatment. In this regard, pharmacological trials show greater uniformity. Most of them have applied the Yale Brown Obsessive Compulsive Scale adapted for PG (PG-YBOCS), the Clinical Global Impression (CGI), and direct measures of gambling behavior such as gambling frequency and amount of money gambled in a given period (Pallensen et al., 2007). This approach eases comparisons, but it is limited to craving, distress caused by gambling, and a few aspects of gambling behavior. Conversely, psychological trials have employed a greater variety of gambling variables to assess treatment, but variations on the assessment methods and variables adopted preclude comparisons. A group of experts, named the *Banff consensus*, proposed that the minimal required information to assess therapeutic results in PG should cover three domains: (1) measures of gambling

behavior including the monthly net expenditure, the frequency (in days per month), and the time spent thinking about or engaged in gambling per month; (2) measures of gambling-related problems (relationships, financial and legal problems, etc.); and (3) measures of the process of change related to treatment modality (e.g., measures of cognitive distortions for gambling cognitive restructuring programs, defense mechanisms for psychodynamic treatments). The authors also state that the assessment of gambling problems could be complemented by measures of the quality of life (Walker et al., 2006). However, since both constructs seem to tap into different realms that play different roles (gambling problems having a determinant role in distress and motivation for treatment, quality of life being important to relapse prevention and consolidation of therapeutic gains), it would be advisable to approach both as separate domains. In particular, quality of life is a multidimensional concept whose definition has been a bit elusive. Maybe the best approach to it would be to unpack the model and choose specific domains that should be important for recovering gamblers, like a sense of belonging to a community, satisfaction with one's attainments in relation to one's culture, and shared value systems, social adjustment, and mental and physical health (Magalhães et al., 2009).

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

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

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

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The Assessment and Treatment of Trichotillomania

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Abstract

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

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

Introduction

Although trichotillomania has been clinically recognized for over a century (Hallopeau, 1889), the literature on this disorder remains scant compared to that of other psychological, psychiatric, and behavioral disorders. Increased interest in trichotillomania arose in the late 1980s out of the consideration of its possible connection to obsessive-compulsive disorder (OCD; see the chapter "Phenomenology and Epidemiology of Trichotillomania") and the realization that trichotillomania is more prevalent than was previously recognized (Christenson et al., 1991d). The conceptualization of trichotillomania as a possible variant of OCD influenced both the initial approach to assessment as well as investigations of pharmacological treatment. In contrast, the preferred behavioral treatments for the two disorders diverged, with exposure/response-prevention having

been established as the most effective intervention for most manifestations of OCD (Abramowitz, 1997), whereas habit reversal therapy became the standard treatment for trichotillomania (Keuthen et al., 1999). However, further research has suggested that these two disorders may not be as closely related as was initially hypothesized and likely require different pharmacological as well as behavioral approaches. As with any condition, the effectiveness of any treatment intervention depends on a thorough clinical assessment as well as comprehension of the treatment literature.

Assessment

Clinical Assessment

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

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

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

The clinical assessment of hair pulling should include an inquiry into activities most likely to be associated with this practice. These are often activities that require sustained attention but limited physical activity such as reading, driving, watching television, or speaking on the phone (Christenson et al., 1995). However, hair pulling may also occur in the context of both physical and mental relaxation.

These attributes of hair pulling are particularly useful to note prior to embarking on behavioral interventions. As many hair pullers engage in some kind of oral manipulation of hair and some actually ingest their hair (trichophagy), it is important to inquire about this specific behavior. The presence of hair ingestion should prompt a review of gastrointestinal symptoms and appropriate additional evaluation if there is any suggestion of the presence of a trichobezoar (hair ball) in the stomach or intestines (see the chapter "Phenomenology and Epidemiology of Trichotillomania")

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

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

Assessment of Severity and Change

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

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

Instruments Measuring Trichotillomania Symptom Change

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

THE MASSACHUSETTS GENERAL HOSPITAL HAIRPULLING SCALE

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

For example, less progress over time on the resistance and control factor may reveal motivational issues that can then be more strongly addressed in therapy (Keuthen et al., 2007a).

NIMH SCALES

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

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

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

THE PSYCHIATRIC INSTITUTE TRICHOTILLOMANIA SCALE

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

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

TRICHOTILLOMANIA SCALE FOR CHILDREN

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

As documented above, the clinician or researcher has several scales to choose from. However, the MGH-HPS has become preferred over other instruments due to its systematic development, brevity,

and similarity of format to self-administered instruments used for other clinical conditions (Diefenbach et al., 2005).

Behavioral Treatment of Trichotillomania

Habit Reversal Therapy

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

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

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

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

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

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

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

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

Behavioral therapy has also been compared to the selective serotonin reuptake inhibitor (SSRI) fluoxetine for the treatment of trichotillomania utilizing a randomized design with a waiting list control (van Minnen et al., 2003). Forty subjects completed this 12-week study, with 14 completers randomized to behavioral therapy, 11 to medication, and 15 to waiting list control. Behavioral therapy was administered during six 45-minute sessions over the course of the 12 weeks. Behavioral treatment components included many of those characteristic of HRT but significantly modified from the original program of Azrin et al. (1980) in regard to selection of specific interventions (e.g., the competing response of making a fist was absent, and stimulus-response interventions consisted of more complex recreational or task-oriented activities). Behavioral strategies included behavioral chain identification and self-monitoring, both via logging of hair pulls and collecting hair in an envelope. Increased awareness of pulling was targeted via the introduction of sensory indicators, which varied by the individual. Targeted sensations included tactile (e.g., wearing bandages on the fingers), olfactory (e.g., placing heavy makeup on the wrist), and/or auditory (e.g., wearing tinkling bracelets on the hair-pulling arm) measures. Stimulus control was approached by organizing the environment to be less conducive to hair pulling. Incompatible behaviors were also introduced to interrupt the chain of hair-pulling behavior. Examples of substituted behaviors included going for a walk, going for a jog, calling a friend, and cleaning a room. Subjects were permitted to pull out hair if the urge to pull persisted provided that they had completed the substituted behavior and had experienced an associated delay in the hair-pulling behavioral chain. The majority of subjects also engaged in stimulus control

by wearing gloves in high-risk situations. Some also administered self-rewards, although specific details about the types of rewards were not mentioned. Results of this study favored behavioral therapy over both medication treatment and waiting list control in regard to decreased symptoms of trichotillomania as well as clinically meaningful improvements. Sixty-four percent of the behaviorally treated group were deemed to have achieved a clinically significant change compared to 20% of the waiting list controls and only 9% of those receiving medication treatment. The authors concluded that behavioral therapy was highly effective in the treatment of trichotillomania, whereas fluoxetine was not.

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

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

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

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

The above-mentioned studies primarily included adults with trichotillomania. The literature on the behavioral treatment of children has generally been limited to case reports and case series using a variety of behavioral approaches (reviewed below). An open trial of cognitive behavioral therapy in children suggests that approaches similar to those employed in treating adults may be useful in this younger population. Tolin et al. (2007) enrolled 22 children and adolescents (mean age, 12.6 years) in a two-phase study of manual-based cognitive behavioral therapy for trichotillomania. The first phase consisted of active treatment administered weekly over 2 months. Individual sessions focused on progressive acquisition of knowledge and skills and included psychoeducation, competing response training, stimulus control, progressive muscle relaxation, cognitive

restructuring, guided self-dialogue and relapse prevention strategies including covert modeling. The second phase concentrated on relapse prevention and consisted of four biweekly sessions and brief intersession phone contacts with the therapist. During this phase, children were reminded of the various strategies acquired during the active treatment phase. The strategies employed by the child or adolescent were also reviewed, with guidance for troubleshooting when needed. Fourteen children completed the study. Based on changes in NIMH trichotillomania scales and Clinical Global Improvement Scales, 77% of subjects were classified as responders. However, at 6-month follow-up, only 63% continued to be responders, representing a loss of one-quarter of the responders. Of interest, the researchers classified 32% of subjects as "excellent responders," and none of this group lost their response at follow-up.

HRT Group Therapy

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

Other Behavioral Techniques

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

to achieve clinical benefit in some individuals with trichotillomania.

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

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

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

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

Aversive consequences of hair pulling have also been utilized, such as self-administered rubber band snaps to the wrist (Mastellone, 1974) and rigorous exercise (Stevens, 1984). Therapist-administered aversive consequences have included faradic shock, hand slaps, and aromatic ammonia inhalation (Altman et al., 1978; Gray, 1979; Horne, 1977). For young children who pull out hair while sucking their thumbs, the application of an aversive-tasting substance to the thumb has been reported to reduce both behaviors (Altman et al., 1982; Friman & Hove, 1987; Knell & Moore, 1988). Response cost,

the denial of privileges, and removal of a valued possession or experience as a consequence of hair pulling have been also been employed but usually in the context of additional interventions (Cordle & Long, 1980; Epstein & Peterson, 1973).

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

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

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

A Flexible Multicomponent Behavioral Approach

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

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

Medication Treatment of Trichotillomania

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

Clomipramine

Although a limited number of reports of apparently effective pharmacological treatment of trichotillomania already existed in the literature, it wasn't until 1989 that the first comparison study of medication treatment of trichotillomania was published. Based on observations that trichotillomania had phenomenological similarities to obsessive-compulsive disorder (OCD), Swedo et al. (1989) compared the strongly serotonergic tricyclic antidepressant clomipramine to desipramine, a tricyclic primarily characterized by noradrenergic reuptake inhibition. Clomipramine had been demonstrated to be superior to less serotonergic antidepressants in studies of OCD (Leonard et al., 1989; Volavka et al., 1985). Thirteen subjects with severe hair pulling completed this double-blind, randomized study, which consisted of 5 weeks treatment with either

medication prior to immediate crossover to the alternative medication. Clomipramine (mean dose, 181 mg/day) proved statistically superior to desipramine, as measured by two of the three NIMH trichotillomania scales. These positive findings are impressive considering the short duration of treatment, initial medication titrations, and lack of a washout period in the study design.

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

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

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

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

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

Selective Serotonin Reuptake Inhibitors

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

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

was deemed ineffective in the short-term treatment of trichotillomania.

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

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

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

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

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

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

Selective Serotonin and Norepinephrine Reuptake Inhibitors

Serotonin and norepinephrine reuptake inhibitors (SNRIs) retain the strong serotonergic properties initially suspected to be of benefit for the treatment of trichotillomania while offering additional norenergic activity similar to, albeit stronger than, that of clomipramine. Considering the initial expectation that serotonergic agents would be of benefit for the treatment of trichotillomania and the data that continue to lend stronger support for the efficacy of clomipramine, it is not surprising that the SNRI venlafaxine would have been investigated as a possible treatment for trichotillomania. O'Sullivan et al (1998) reported on 10 patients who had been treated with venlafaxine (mean dose = 274 mg/day) for

8–28 weeks. Significant improvement was observed in an intent-to-treat analysis as measured by the MGH-HPS and the PITS. Ninan et al. (1998) treated 20 patients with venlafaxine (mean dose, 322.5 mg/day) for up to 12 weeks. Responders were defined as demonstrating a minimal 50% reduction in hair-pulling symptoms as well as being clinician rated as “much” to “very much” improved. Based on these criteria, 55% of subjects were deemed treatment responders. Eight responders were then entered into a double-blind discontinuation trial in which half were randomly assigned to continue on medication and half were switched to placebo. With the exception of one patient who received placebo, all subjects relapsed within the next 24 weeks.

Opiate Antagonists

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

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

Dopamine Antagonists

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

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

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

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

Although there is evidence supporting the effectiveness of olanzapine and other atypical drugs for the treatment of trichotillomania, the known potential side effects of these agents, including weight gain, hyperglycemia, hyperlipidemia, cardiac arrhythmia,

and tardive dyskinesia (Üçok & Gaebel, 2008), suggest judicial consideration of the risks and benefits, as well as consideration of whether the severity of trichotillomania in individual cases justifies treatment with this pharmacological class.

Mood Stabilizers

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

Topical Agents

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

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

Glutamate Modulators

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

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

Anxiolytics

Since SSRIs and SNRIs have anxiolytic properties and several have specific indications for generalized anxiety, traditional anxiolytics have also been

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

Other Agents

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

Combined Behavioral and Medication Treatment

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

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

Hypnosis

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

Robiner et al. (1999) also reported on an additional 10 patients with trichotillomania who participated in

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

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

Naturalistic Treatment Studies

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

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

Boughn and Holdom (2002) recorded semi-structured phone interviews of 44 self-identified hair pullers to assess their perceptions of their response to treatment. Treatment approaches were divided into five broad categories: medications, behavior modification, individual therapy, group therapy, and alternative therapy. Seventy-seven percent of subjects had received medication for trichotillomania or coexisting disorders such as anxiety and/or depression. Medications included four different SSRIs, clomipramine, venlafaxine, and other antidepressants, as well as lithium, olanzapine, buspirone, and several benzodiazepines. Only two subjects (6%) reported long-term efficacy with medication. Both subjects had four ineffective pharmacological interventions before they responded to either St. John’s wort or a combination of olanzapine and paroxetine. Seventy-three percent of subjects had been

treated with behavioral modification; 56% found this intervention to be effective. However, participants noted that maintaining the acquired techniques was challenging. Individual psychotherapy was reported to be utilized by 71% of subjects but was less effective than behavioral modification; only 32% of subjects treated with individual psychotherapy responded to this intervention. Group therapy was utilized by 68% of subjects. Five of eight (63%) women treated with group therapy designed to treat trichotillomania found it to be beneficial; in contrast, none of seven women treated with group therapy that did not specifically target hair pulling found this approach to be helpful. The authors used the term *nontraditional group therapy* to describe two other types of group involvement. One of them referred to Internet-based trichotillomania support groups. The other referred to involvement with various aspects of the Trichotillomania Learning Center (TLC), a national organization that provides support, information, referrals, and resources for individuals with trichotillomania. Eighteen (55%) subjects interacted with at least one of these two resources, and seventeen (94%) found them to be effective. Web-based support groups were noted to have the advantage of anonymity, and appeared to be less threatening than face-to-face participation. The authors noted that the existence of both the Web-based support groups and the TLC resources helped to emphasize that subjects were not alone, provided support from others dealing with trichotillomania, and provided resources that otherwise may not have been available in the subject’s community.

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

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

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

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

administered in the community, suggest that despite multiple treatment strategies, perhaps only 15%–20% of cases of trichotillomania will achieve acceptable sustained improvement.

Conclusion

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

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

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

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

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

Future Directions

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

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

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

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Assessment and Treatment of Kleptomania

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

Abstract

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

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

Introduction

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

The first *Diagnostic and Statistical Manual of Mental Disorders* (DSM-I) did not include kleptomania as a formal diagnostic illness but rather as a supplementary term (APA, 1952). Kleptomania was left out of the DSM-II altogether (APA, 1968). In 1980, the DSM-III categorized kleptomania as an impulse control disorder not elsewhere classified (APA, 1980), the same clinical diagnostic category it currently occupies in the DSM-IV-TR. As an impulse control disorder, kleptomania is currently

classified in DSM-IV-TR with pathological gambling, pyromania, intermittent explosive disorder, and trichotillomania (APA, 2000). Although included in DSM-IV, kleptomania is still a poorly understood disorder and has received very little empirical study.

Diagnosis

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

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

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

Failure to Diagnose

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

Shame and Secrecy

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

Patient Lack of Knowledge

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

Clinician Lack of Knowledge

Few health care professionals have education or training in kleptomania. In addition, clinicians may have many of the same biases about stealing as do patients. For example, clinicians may see themselves as potential victims of the illegal behaviors associated with impulse control disorders—that is, clinicians

pay more for items due to shoplifting—and therefore may not feel that the behavior is an illness and deserving of treatment.

Misdiagnosis

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

Mood Disorder

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

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

Obsessive-Compulsive Disorder

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

Substance Use Disorders

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

Personality Disorders

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

Distinguishing Kleptomania from Typical Shoplifting

Individuals with kleptomania differ from ordinary shoplifters in that they do not steal for personal gain, but rather for symptomatic relief. It is estimated that

more than \$13 billion worth of goods are stolen from retailers each year, which translates into more than \$35 million per day (National Association for Shoplifting Prevention, 2009). The vast majority of shoplifters are described as amateurs with sporadic activity, with no known history of criminal activity, and who steal for their own consumption rather than for resale.

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

Assessment of Stealing in Children and Adolescents

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

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

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

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

Treatment

Biological Theories

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

SEROTONIN AND INHIBITION

Evidence for serotonergic involvement in impulse control disorders comes in part from studies of platelet monoamine oxidase B activity, which correlates with cerebrospinal fluid levels of 5-hydroxyindoleacetic acid and is considered a peripheral marker of serotonin (5-HT) function (Coccaro et al., 1990; Linnoila et al., 1983). Patients with

kleptomania report significant increases in impulsivity and risk taking compared to normal controls (Baylé et al., 2003; Grant & Kim 2002d), and diminished inhibitory mechanisms may underlie the risk-taking behavior of kleptomania. Decreased levels of serotonin have long been associated with a variety of adult risk-taking behaviors (Moreno et al., 1991; Virkunnen et al., 1994). One study examined the platelet serotonin transporter in 20 patients with kleptomania. The level of the platelet 5-HT transporter, evaluated by means of binding of ³H-paroxetine, was lower in patients with kleptomania than in healthy control subjects (Marazziti et al., 2000), suggesting serotonergic dysfunction.

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

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

DOPAMINE AND REWARD DEFICIENCY

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

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

OPIOID SYSTEM, CRAVINGS, AND PLEASURE

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

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

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

Psychological Theories

The etiology of kleptomania is most likely multifactorial (biological, psychological, sociocultural). Why do kleptomania patients continue to engage in a behavior that results in unneeded items when the possible repercussions are so devastating? Attempting to address this complex question, many psychological theories of kleptomania have been postulated: for example, gratification of unconscious sexual impulses (Goldman, 1991; McElroy et al., 1991a), castration anxiety (Levy, 1934), low self-esteem (Goldman, 1991), unresolved dependency

(Allen, 1965), and masochism (Rado, 1933). There are no data, however, to confirm or refute these theories (Goldman, 1991). Focusing on the pleasure many patients derive from shoplifting, some have theorized that kleptomania results from an attempt to relieve feelings of depression through stimulation (Goldman, 1991; Gudjonsson, 1987; McElroy et al., 1991a). Risk-taking behavior may therefore produce an antidepressant effect for some patients (Fishbain, 1987; Goldman, 1991). It is possible that depressed individuals may engage in shoplifting to distract themselves from life stressors and unpleasant cognitions. Unlike drugs or alcohol, shoplifting leads to neither intoxication nor a directly impaired ability to function at work and, as such, may be an especially attractive means of escape. Persons who are depressed may also view the objects they steal as a means of achieving significant symptom relief and view the possibility of being apprehended as a relatively minor and theoretical setback. Ironically, problems resulting directly from shoplifting (e.g., embarrassment and shame from getting caught) may, in turn, lead to even more shoplifting in a misguided attempt at symptom management.

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

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

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

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

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

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

psychological and biological understanding of operant processes in the etiology and maintenance of kleptomania.

Pharmacotherapy

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

CASE REPORTS

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

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

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

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

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

OPEN-LABEL STUDIES

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

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

DOUBLE-BLIND, PLACEBO-CONTROLLED STUDIES

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

on placebo (Fisher's exact = .003) (Grant et al., 2009). Although the study was only 8 weeks long, a naturalistic outpatient study demonstrated that subjects treated with naltrexone monotherapy for up to 3 years continued to report overall improvement (Grant, 2005).

PSYCHOTHERAPY

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

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

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

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

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

PHARMACOTHERAPY AND COMBINATION TREATMENTS

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

Recommendations Based on Treatment Outcome Literature

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

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

Future Directions

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

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Abstract

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

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

Introduction

Intermittent explosive disorder (IED) is the DSM-IV-TR diagnostic category used to classify individuals who engage in repeated acts of impulsive aggression that are disproportionate to any provocation and are not better accounted for by the effects of a substance, medical condition, or other psychological disorder (APA, 2000). Importantly, IED is the only disorder in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; APA, 2000) for which affective aggressive behavior is the defining feature. Though historically IED was thought to be quite rare (APA, 2000), recent clinical and epidemiological findings support the notion that it is an underdiagnosed disorder with lifetime prevalence rates ranging from 4% to 7% (Coccaro et al., 2005; Kessler et al., 2006; Ortega et al., 2008). Furthermore, IED is associated with significant impairments in interpersonal and occupational functioning that can include loss of work, troubled

relationships, and legal problems (McElroy et al., 1998). This burden is compounded by the chronic nature of the disorder when untreated (Kessler et al., 2006; McElroy et al., 1998). In addition, IED is associated with significant medical problems including coronary heart disease, hypertension, and stroke (McCloskey et al., 2010). Despite its prevalence and severity, there is a relative dearth of information on the assessment and treatment of IED.

Assessment of IED

IED Diagnostic Issues

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

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

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

The final exclusionary criterion (C) specifies that the aggressive acts are not due to another mental disorder, a substance, or a general medical condition. This can be complicated, as individuals with IED often have a history of several other comorbid disorders, including an increased rate of substance use disorders (Coccaro et al., 2005; Kessler et al., 2006; Ortega et al., 2008). For more acute disorders,

a temporal comparison of the aggressive episodes and the comorbid condition is usually sufficient to determine if IED should be diagnosed. For example, if an individual being evaluated for IED also has a history of major depression with three depressive episodes, the assessor would determine if the level of aggression frequency and severity *outside* of the three depressive episodes was sufficient to meet criteria for IED. If not—that is, if the person's aggression occurred primarily when clinically depressed, the individual would not be given an additional diagnosis of IED. However, this becomes more complicated when the exclusionary disorders are chronic. For example, antisocial personality disorder and borderline personality disorder are explicitly listed in the DSM as two potential exclusionary diagnoses. However, both disorders (much like IED) become evident early in life and tend to follow a chronic course, making it extremely difficult to determine whether the aggression is better accounted for by the existing personality disorder or IED.

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

providing a separate, partially overlapping criteria set for IED.

IED Assessment

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

INTERMITTENT EXPLOSIVE DISORDER MODULE (IED-M)

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

The IED-M has been used in several published studies to diagnose IED (Coccaro et al., 2004; McCloskey et al., 2008a). These studies have provided indirect evidence of the measure’s construct validity. Specifically, individuals diagnosed as having IED based on the IED-M are more aggressive on self-report and behavioral aggression measures than comparison groups with other psychiatric disorders

(McCloskey et al., 2006; McCloskey et al., 2008a). Unpublished data from Coccaro also show that IED diagnoses using the IED-M have strong interrater reliability ($k = .84$) when used with adults as part of a full diagnostic battery that includes structured clinical interviews for other Axis I and Axis II disorders. The only published validation study of the IED-M showed the measure to have strong interrater reliability ($k = .76-.87$) but less impressive test-retest reliability ($k = .43-.63$) when administered to a group of 34 children and adolescents in treatment and community settings (Olvera et al., 2001). This study also provided evidence for the construct validity for the IED-M, with the IED group reporting higher levels of lifetime verbal and physical aggression than community or psychiatric controls (Olvera et al., 2001).

INTERMITTENT EXPLOSIVE DISORDER DIAGNOSTIC QUESTIONNAIRE (IED-DQ)

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

Aggression Assessment

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

LIFE HISTORY OF AGGRESSION (LHA)

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

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

BUSS PERRY AGGRESSION QUESTIONNAIRE (BPAQ)

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

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

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

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

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

Treatment of IED

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

Pharmacological IED Treatments

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

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

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

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

Mood stabilizers have also been evaluated for their antiaggressive effects. An early RCT showed that lithium reduced violent infractions relative to placebo among prisoners with chronic aggressive behavior (Sheard et al., 1976). More recently, studies have looked at anticonvulsant mood stabilizers such as carbamazepine and divalproex. A small RCT showed that divalproex reduced "explosive temper" relative to placebo in children with a disruptive behavior disorder (Donovan et al., 2000). These results paralleled an open-label trial of divalproex on

personality-disordered adults in which the medication was found to decrease irritability and aggressiveness over the e8-week trial period (Kavoussi & Coccaro, 1998), as well as a small crossover study comparing carbamazepine to placebo in 11 women with borderline personality disorder (Gardner & Cowdry, 1986). However, the antiaggressive effects of these mood stabilizers may not extend to all IED subjects. A large-scale RCT of divalproex in aggressive patients suggested that the medication was not more efficacious in reducing aggression than placebo (Hollander et al., 2003). A subanalysis showed that divalproex did reduce aggression among IED-IR subjects with a comorbid Cluster B personality disorder, but not among IED-IR subject without a comorbid Cluster B personality disorder (Hollander et al., 2003).

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

Behavioral IED Interventions

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

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

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

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

require a longer, more intensive therapy regimen than subclinical aggressive populations (Galovski & Blanchard, 2002).

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

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

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

Conclusions

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

Future Directions

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

A related area of importance will be to disentangle IED from other comorbid disorders. Intermittent explosive disorder is comorbid with several psychological disorders that are associated with an increased risk of aggression (e.g., substance use disorders, mood disorders, borderline personality disorder).

This distinction is most difficult for chronic disorders such as borderline personality disorder. Research looking into the neurobiological underpinnings of IED may help discriminate these disorders. Whereas borderline personality disorder is associated with generalized limbic hyperactivation to negative stimuli, early research suggests that patients with IED have limbic activity that is specific to anger stimuli (Coccaro et al., 2007).

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

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

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Assessment and Treatment of Pyromania

Jon E. Grant and Brian L. Odlaug

Abstract

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

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

Introduction

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

The first *Diagnostic and Statistical Manual of Mental Disorders* (DSM-I) included pyromania as a supplementary term rather than as a formal diagnostic entity (APA, 1952), and pyromania was omitted entirely from DSM-II (APA, 1968). DSM-III categorized pyromania as an impulse control disorder not elsewhere classified (APA, 1980), the same clinical diagnostic category it currently holds in the

DSM-IV-TR (APA, 2000). As an impulse control disorder, pyromania is currently classified with pathological gambling, kleptomania, intermittent explosive disorder, and trichotillomania (APA, 2000). Although included in DSM-IV-TR, pyromania is still a poorly understood disorder and has received very little empirical study.

Diagnosis

Pyromania, also referred to as *pathological fire setting*, is defined by the following criteria according to DSM-IV-TR: (1) deliberate and purposeful fire-setting that has occurred on more than one occasion; (2) feelings of tension or arousal preceding a fire-setting act; (3) pleasure, gratification, or relief when setting fires or when watching/participating in the aftermath of the fire; (5) the act of fire-setting is not done out of vengeance or for monetary gain; and (6) fire-setting cannot be directly attributed to another mental condition such as conduct or bipolar disorder or impairment due to substance use (APA, 2000, p. 671).

Only one published instrument is used to screen for pyromania: the Minnesota Impulse Disorders Interview (MIDI; Grant, 2008). The MIDI includes five questions that screen for pyromania. These questions have demonstrated excellent classification accuracy based on structured clinical interviews for pyromania in adults (sensitivity 100%, specificity 100%; Grant et al., 2005) and in adolescents (sensitivity 85.7%, specificity 98.9%; Grant et al., 2007). Using this instrument, researchers found lifetime pyromania rates of 5.9% in a sample of 204 adult psychiatric inpatients (Grant et al., 2005) and 6.9% in a sample of 102 adolescent psychiatric inpatients (Grant et al., 2007). Furthermore, a modified self-report version of the MIDI found a lifetime pyromania prevalence rate of 1.01% in a sample of 791 college students (Odlaug & Grant, 2010).

Failure to Diagnose

Although pyromania may be fairly common among certain populations (e.g., psychiatric patients), it usually goes undiagnosed. Many reasons exist for the failure to diagnose this disorder.

Shame and Secrecy

Shame and secrecy are aspects of pyromania, largely due to the illegal nature of the behavior. Many people are also embarrassed because of the lack of control inherent in pyromania. Shame and secrecy may explain to some extent why so few patients will volunteer information regarding this behavior unless specifically asked (Grant et al., 2005). Patients suffering from pyromania may also be worried that the clinician is required to report their behavior. Clinicians therefore may want to inform patients at the beginning of the evaluation about what they do and do not have to report.

Clinician Lack of Knowledge

Few health care professionals have education or training in pyromania. Clinicians may instead diagnose fire setting as antisocial personality disorder or as a symptom of mania. In addition, clinicians may have many biases regarding fire setting. For example, clinicians may see themselves as potential victims of the behavior. Therefore, they may see fire setting as purely criminal, without further assessment of the underlying motivations.

Misdiagnosis

Fire setting may be a symptom of other disorders. Some of the more common misdiagnoses are discussed below.

Bipolar Disorder

Bipolar manic episodes are characterized by impulsive behaviors (APA, 2000). These manic behaviors may include fire setting (Gunderson, 1974). In addition, individuals with pyromania have high rates of co-occurring bipolar disorder (14.3%; Grant & Kim, 2007). During a manic episode, however, people often exhibit multiple symptoms of mood dysregulation—excess energy, distractibility, mood elevation, or irritability (APA, 2000)—whereas the behavior of pyromania usually has none of these associated symptoms. The DSM-IV excludes a diagnosis of pyromania when the fire setting is due to another mental condition such as bipolar disorder (APA, 2000). When individuals with pyromania have co-occurring bipolar disorder, it is important for the clinician to determine if the fire setting exists only during mania or possibly worsens during a manic episode.

Substance Use Disorders

Substance use disorders frequently co-occur in individuals with pyromania (33.3%; Grant & Kim, 2007). It is important for clinicians to determine (1) whether the substance use problem results in the fire setting, perhaps through disinhibition (e.g., fire setting only when intoxicated), or (2) whether they are related but independent problems. One study of 90 arson recidivists found that only 3 (3.3%) met criteria for pyromania but that an additional 9 subjects met criteria for pyromania when intoxicated at the time of the fire setting (Lindberg et al., 2005). Additionally, a survey of 34 adult arsonists found that 100% were under the influence of alcohol prior to or during the act of setting fires (Jayaraman & Frazer, 2006).

Personality Disorders

One study found that the rate of borderline personality disorder is elevated in individuals with pyromania (9.5%; Grant & Kim, 2007) compared to the community at large (5.9%; Grant et al., 2008). The DSM-IV-TR diagnosis of pyromania excludes those who set fires out of anger (which will exclude fire setting only as a symptom of borderline personality disorder) or those who set fires for monetary gains or criminal purposes (which will exclude individuals with antisocial personality disorder who set fires only for criminal reasons; APA, 2000). The clinician must therefore determine if the fire setting merits an independent diagnosis of pyromania or is only secondary to a personality disorder.

Psychotic Disorders

In one study of 54 fire setters, psychotic disorders were seen in 21 (38.9%) of the cases (O'Sullivan & Kelleher, 1987). In a similar study, 20%–30% of arsonists reported symptoms consistent with psychosis (Taylor & Gunn, 1984). Fire setting that is performed only during a psychotic episode would not meet DSM-IV-TR criteria for pyromania (APA, 2000). Clinicians therefore need to screen for possible thought disorders at the time of the fire setting.

Distinguishing Pyromania from Criminal Fire Setting

Most individuals who set fires are not pyromaniacs. In fact, the most common motives for fire setting appear to be anger and revenge (Rix, 1994). People without pyromania who set fires have been found to have poor interpersonal skills, and their fire-setting behavior is thought to reflect their inability to cope with feelings of anger and frustration (Rix, 1994). For these individuals, there may be no psychiatric disorder driving the behavior. One study of 191 psychiatric inpatients found that approximately 26% had indulged in some kind of fire-setting behavior, although no relationship could be found between this behavior and a psychiatric illness (Geller & Bertsch, 1985). A review of 79 psychiatric outpatient children revealed that 18.9% were fire setters (Fitzgerald & O'Hanlon, 1991). Consequently, it is important that the clinical interview examine the motivations behind the fire setting and not simply assess the behavior. Individuals with pyromania have urges to set fires and experience a “rush” from this behavior (Grant & Kim, 2007).

There is often confusion about the terms used to describe the various types of fire setting. *Pyromania* is the proper term for fire-setting behavior only when the DSM-IV criteria have been met. In contrast to pyromania, arson is not a diagnosed psychiatric disorder or medical illness and has a distinct definition that differs from that of pyromania. Although state statutes may differ on the explicit language, *arson* is generally defined as a crime of maliciously, voluntarily, and willfully setting fire to a building or other property of another person or burning one's own property for an improper purpose (i.e., insurance fraud). Generally, an arsonist is motivated by some type of gain, whether it is revenge for a wrongdoing, for insurance fraud, or for socio-political reasons. Pyromania, on the other hand, is an impulse or urge-driven behavior that affects, among other things, the social and occupational lives of the individuals suffering from the illness.

These urges have been described as addictive, and the act of setting a fire produces a sense of calm for the individual (Grant & Kim, 2007).

Unlike pyromania, arson appears to be a common occurrence that affects many people financially and emotionally. In 2004 according to statistics from the Federal Bureau of Investigation, there were 68,245 reported arson offenses in the United States, with an average dollar loss per incident of \$12,017 (Federal Bureau of Investigation, 2004). Recent data provided through the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) found that 1.13% of a large community sample acknowledged having started a fire in their lifetime for the purpose of destroying someone else's property or just to see it burn (Vaughn et al., 2010). Very few arsonists, however, suffer from pyromania. One study found that only 3 (3.3%) of 90 arson recidivists had pure pyromania (Lindberg et al., 2005). In addition, not everyone who suffers from pyromania is an arsonist. A person meeting DSM-IV criteria for pyromania may also meet the legal definition of arson, but the DSM-IV criteria for pyromania do not require that a person ever set fire to another person's property.

Assessing Juvenile Fire Setting

Fascination with fire has been documented for centuries. Usually beginning at around 2 or 3 years of age, fire's attraction remains constant throughout an individual's life. In fact, behaviors such as playing with matches have been noted in 24.4% of child psychiatric outpatients, and a fire-setting rate of 19.4% has been found in this sample (Kolko & Kazdin, 1988). Another study of 99 elementary school boys found that 45% played with matches (Kafry, 1980). Although it is presently unknown how common this behavior is among children in the general population, its consequences are clear. Burn injuries account for 40% of accidental deaths in children under the age of 5, making these injuries the second leading cause of death in this age group (National Fire Data Center, 2001).

Juvenile fire setters are responsible for approximately 60% of all fires set in large cities (Mieszala, 1981). Local fire departments detect more children who set fires than do mental health professionals (Kolko, 1988), and on most occasions they are responsible for referring children suspected of concerning/serious fire-setting behavior to the various services for appropriate intervention. Therefore, various state fire departments have developed screening measures to help identify fire-setting

behavior in children (Fineman, 1980). Screening tools help fire department officials assess the severity of fire-setting behavior and then implement appropriate intervention and follow-up. Appropriate and adequate follow-up is important because the index fire-setting behavior is often preceded by rejection by the individual's care providers or social services departments (Koson & Dvoskin, 1982), and may contribute to high rates of suicide and self-injurious behavior among arsonists (O'Sullivan & Kelleher, 1987).

Based on an initial screening interview, a fire marshal may classify a child with fire-setting behavior into a category (Federal Emergency Management Agency, 1983) of either little (the fire department intervenes through an educational approach and provides information on safety), definite, or extreme concern. Children falling into the definite or extreme-risk category are referred to mental health services for further assessment and treatment of the behavior. Children with relatively less concerning fire-setting behavior (e.g., "curiosity" fire setters with little or no parental supervision) do not necessarily go through a mental health referral and benefit most from fire safety education provided by trained fire department officials (Fineman, 1995). This approach, along with educating the parents, has been shown to improve outcomes in children (with less concerning behavior) in terms of decreasing future fire-setting behavior (Fineman, 1995). Parents of children who set fires may find it difficult to accept such behavior as needing the attention of mental health professionals. This may be a huge barrier to the treatment of a child with serious/extreme fire-setting behavior. In such circumstances, it is vital to understand the social situation of the family (which itself may have contributed to the fire-setting behavior) and take aggressive steps to get the family involved in treatment and appropriate follow-up, even if that means forceful intervention on the part of the fire department by threatening a report to the Child Protection Services.

Mental health evaluations consist of a psychosocial assessment and a psychiatric diagnosis. A screening procedure developed by Sakheim and colleagues (1985, 1986) has helped identify certain variables that, when present in the assessment/interview of a child, correlate positively with the fire-setting behavior. Some of these variables are strong feelings of anger/resentment over neglect/abandonment and/or abuse by parents, strong feelings of revenge for the wrong done by the parents, poor impulse control and poor judgment, sexual maladjustment

(which may be the result of abuse itself), absence of the father, upbringing in a conflicted family (Fineman, 1980), and a history of previous fire setting (by matches or lighters). Other positive correlates of fire-setting behavior are a psychiatric diagnosis of conduct disorder and dysthymic disorder (Fineman, 1980) and a positive response to the test item "I get quite excited when I see a fire burning."

Factors that were found to decrease the risk of (future) fire setting were feelings of guilt and depression after setting the fire, the ability to verbally express frustration/anger, the ability to empathize with other people, and good relations with peers/desire to feel close and intimate (Sakheim & Osborn, 1986; Sakheim et al., 1985).

Both typical fire setting and pyromania may start at a relatively early age. While child and adolescent fire setting is fairly common, true pyromania is quite rare in this age group. Researchers have identified revenge on parents or other persons in authority as the primary motivator for juvenile fire setters (Räsänen et al., 1995). There is, however, little information on how pyromania presents in adolescents and whether it presents differently than in adults. Based on reports of adolescent pyromania (Grant, 2006), there is some evidence that the current DSM-IV criteria apply to adolescents who report being unable to control their fire setting. More research, however, is needed to understand the presentation of fire setting among adolescents.

Treatment

Etiology

Understanding the possible etiology or etiologies for pyromania may better target treatment options. The underlying biological mechanism of urge-based disorders may involve the processing of incoming reward inputs by the ventral tegmental area-nucleus accumbens-orbitofrontal cortex circuit. This circuit influences behavior by modulating animal and human motivation (e.g., urges, cravings; Kalivas et al., 1999). Dopamine and glutamate may play major roles in the regulation of this region's functioning. One hypothesis is that differences in this region may result in the urges seen in pyromania and other impulse control disorders. The efficacy of topiramate (see below) lends some support to this hypothesis. Topiramate is thought to modulate dopaminergic and glutamatergic neurons in this area.

Urges linked to the experience of reward and pleasure represent an important potential clinical target in pyromania. Patients with pyromania report urges to set fires. Most report fairly frequent urges

that result in fire setting. Many indicate that the act of setting a fire reduces the urges or the tension these urges produce. The act of fire setting also often provides a “rush” for some patients, producing a pleasurable feeling (Grant & Kim, 2007).

Frontal serotonergic systems have been implicated in impaired impulse regulation, and serotonin dysregulation may also be a clue to the pathophysiology of pyromania (as well as other impulse control disorders). Pharmacological intervention for pyromania has produced some promising results from selective serotonin reuptake inhibitors (SSRIs).

Some support for frontal inhibitory dysfunction comes from a study of a single individual with pyromania. Neuroimaging using single photon emission computed tomography (SPECT) found an association between left inferior frontal perfusion deficits and pyromania in a young man with pyromania (Grant, 2006). Other support comes from neuropsychological assessment of one subject with pyromania that found impairments in attention (Continuous Performance Test), verbal/visual memory (Rey Auditory Verbal Learning and Complex Figure Tests), and executive functions (Verbal Fluency); by contrast, visuospatial skills (Parietal Lobe Test) were intact (Parks et al., 2005).

New-onset fire setting has also been described in an individual with an arachnoid cyst of the cerebellar vermis (Heidrich et al., 1996). Neuroimaging studies examining other impulse control disorders, such as trichotillomania (TTM), have reported reduced volumes of the total, right, and left cerebellum cortex in TTM (Keuthen et al., 2007). Keuthen et al. (2007) found that these patients had significantly reduced volumes of the emotional functional cluster compared to controls, potentially underscoring the affective symptoms often reported in TTM subjects as well as those with other impulsive behaviors such as pyromania.

From a behavioral standpoint, fire setting may raise self-esteem by creating a feeling of power from the destruction of property, and this feeling may be particularly reinforcing when a person sets a fire without being apprehended. Some evidence supporting this theory may be found in the fact that individuals with pyromania report rates of abuse in childhood that are higher than those found in the population at large (Grant & Kim, 2007). Other behavioral models that have been used to explain other impulse control disorders may also apply to pyromania. Physiological arousal associated with fire setting (the “rush” reported by individuals with pyromania; Grant & Kim, 2007) may

be a reinforcer that initiates and perpetuates the behavior.

Treatment Options

There are no randomized, controlled clinical trials examining either pharmacotherapy or psychotherapy for the treatment of pyromania. No medications have been approved by the Food and Drug Administration (FDA) for the treatment of this disorder. Therefore, it is important to inform patients of any “off-label” use of medications for this condition, as well as about the empirical basis for considering medication treatment.

Several different classes of medications have been described in case reports as demonstrating benefit in the treatment of pyromania. These include SSRIs such as escitalopram, sertraline, and fluoxetine; the mood stabilizer lithium; the antiepileptic medication topiramate; and a combination of the neuroleptic olanzapine and the antiepileptic sodium valproate (Grant & Kim, 2007; Parks et al., 2005). An equal number of medications, however, have also shown no benefit in the treatment of pyromania in case reports: fluoxetine, valproic acid, lithium, sertraline, olanzapine, escitalopram, citalopram, and clonazepam.

One case report, illustrating the treatment of an 18-year-old male with pyromania, described the use of a combination of topiramate with 3 weeks of daily cognitive behavioral therapy (CBT). The CBT consisted of fifteen 1-hour sessions that included imaginal exposure with response prevention and cognitive restructuring of fire-setting urges (Grant, 2006).

Other treatments have been developed for fire-setting behavior without limiting the intervention to pyromania. One case report detailed the use of relaxation therapy, overt sensitization, and awareness training in a 10-year-old male who set fires. One-year follow-up results indicated successful abstinence from all fire-setting behavior (Koles & Jenson, 1985). Other studies describing behavioral treatments of fire setting include methods such as fire safety education, coping and relaxation skills, aversive therapy, positive reinforcement, stimulus satiation, and operant structured fantasies and prevention programs designed for pyromania (Bennett et al., 2004; Bumpass et al., 1983; Clare et al., 1992; Franklin et al., 2002; Kolko, 1988; Rice & Quinsey, 1980; Taylor et al., 2002).

A treatment intervention for children developed by Bumpass and colleagues (1983) using a line-graphing technique with 29 children who set fires

helped the children become aware of the factors that triggered the behavior and identify the feelings that culminated in their fire setting (Bumpass et al., 1983). An average 2.5-year follow-up reported that 27 (93.1%) of the 29 children continued to set fires.

Bennett and colleagues (2004) report the successful use of fire education provided by firefighters to 42 children (29 court-referred) with fire-setting behaviors. After an assessment and subsequent fire-setting consequence education were provided, the researchers found a 100% success rate in quelling fire-setting behavior.

Group therapy methods have also been utilized with some success. Rice and Chaplin (1979) describe the use of assertion training in a group therapy setting for 10 fire-setting patients detained in a maximum security psychiatric institution (Rice & Chaplin, 1979; Rice & Quinsey, 1980). Along with social skills training already provided in the hospital, this training consisted of teaching patients how to express anger appropriately and how to say "no" to unreasonable requests made by others. Role playing and modeling the behavior of both therapists and other group members were also employed. At an 18-month follow-up, researchers noted no fire-setting behavior among the nine completers of the therapy.

Taylor and colleagues (2002) reported the use of 40-session CBT-based group therapy for 14 subjects with below average- or borderline intelligence (IQ=64-84) convicted of arson. Through a focus on reducing interest in fire and changing beliefs and attitudes about fire setting, clinically significant improvements on fire setting attitudes and interest were found in 10 (71.4%) subjects at the conclusion of treatment (Taylor et al., 2002).

Using preventive methods, Franklin and colleagues (2002) found that after 132 juvenile subjects (ages 4-17) who were arrested and convicted of fire setting were subjected to a full day of fire education, including the various financial, legal, and societal impacts of fire setting and an emphasis on individual accountability for their actions, the rate of recidivism was less than 1% for those in the active treatment group. A control group of 102 offenders fared less well, with a recidivism rate of 36% (Franklin et al., 2002).

Although there is no standard treatment method for pyromania at this time, based on the very limited literature, an approach using both CBT and pharmacological treatment may be most beneficial.

Conclusions

Pyromania is a largely unrecognized disorder that causes significant psychological, social, and legal repercussions. Because few individuals volunteer information regarding their fire setting, it is important that clinicians recognize the disorder and screen patients appropriately. Various treatments have been helpful in case studies, but more research on etiology and treatment is needed.

Future Directions

In an effort to better understand and subsequently treat this often disabling disorder, future research should strive to identify the neurobiological underpinnings of pyromania. Neuroimaging, neuropsychological testing, and randomized, placebo-controlled clinical trials are sorely needed and widely absent from the current literature for pyromania.

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Assessment and Treatment of Pathological Skin Picking

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Abstract

Pathological skin picking (PSP) refers to chronic skin picking or scratching that causes tissue damage and distress. It is a heterogeneous category of behaviors and may be manifest in the context of various psychological disorders. This chapter presents an overview of the empirical literature on the assessment and treatment of PSP, including (1) a cognitive-behavioral model as heuristic for conceptualizing treatment, (2) assessment tools, (3) a review of the pharmacological and psychosocial treatment outcome literatures, (4) cognitive-behavioral treatment techniques, and (5) future directions. The chapter is intended to introduce the clinician to the assessment and psychological tools used to treat PSP, as well as to provide impetus to advance research in this understudied domain.

Keywords: skin picking; body-focused repetitive behavior; cognitive-behavior therapy; excoriation; habit reversal training

Assessment and Treatment of PSP

Introduction

Pathological skin picking (PSP) refers to chronic picking or scratching of skin lesions that causes tissue damage and distress, sometimes quite severe (Simeon et al., 1997). In the literature it is referred to in many ways, including *neurotic*, *psychogenic*, or *psychocutaneous excoriation*; *dermatillomania*; *neurodermatitis*; *neurotic or self-inflicted dermatosis*; and *acne excoriée*, all of which may or may not describe exactly the same phenomenon. Not currently classified as a unique psychiatric disorder (American Psychiatric Association, 2000), PSP is a heterogeneous condition that is similar to other body-focused repetitive behaviors (BFRBs) but is manifest in the context of numerous disorders and clinical presentations. Individuals engage in PSP for many reasons, and the behavior itself varies considerably from individual to individual (e.g., site and instrument of picking). The phenomenology and treatment of PSP are relatively understudied, and although there is budding evidence for the

efficacy of psychological and psychiatric interventions, the empirical literature is limited. The clinician wishing to develop a treatment approach may be flummoxed when consulting a small literature based on a heterogeneous patient population.

The purpose of this chapter is to provide an overview of the assessment and treatment of PSP and, in so doing, (1) to introduce the clinician to the assessment and psychological (particularly cognitive-behavioral) tools used to treat PSP and (2) to provide an impetus to advance research on PSP. We begin with a simplified model, as directly relevant to assessment and treatment, highlighting the diverse and multiply determined behaviors under the umbrella term *pathological skin picking*. With attention to the consequent importance of careful and functional assessment, we then present assessment tools with demonstrated utility for PSP. Thereafter, we review the treatment outcome literature and then describe cognitive-behavioral techniques in treating PSP. Finally, we end with directions for future research.

Cognitive-Behavioral Model of PSP

Historically, the model for understanding PSP has centered on the notion of urge reduction. That is, picking was thought to occur in direct response to an internal tension, urge, or discomfort, which was then relieved by picking. Thus, picking was negatively reinforced each time it successfully rid the individual of the unpleasant sensation. The growing literature on BFRBs, however, suggests that this contingency is likely only one of many factors involved in the development and maintenance of PSP. The current conceptualization incorporates a range of biological, psychological, and situational features that can play a role in the presentation of this phenomenon. This newer model importantly accounts for the heterogeneous presentation of PSP, which includes the varied pathways through which the behavior could result and be maintained. Figure 27.1 presents a simplified version of the models presented by Franklin and Tolin (2007) and Mansueti et al., (1997) for trichotillomania and other BFRBs. We delineate the different components of this model below and also illustrate how a careful functional analysis is imperative when selecting the

most helpful treatment avenue for any given individual. An important caveat to consider with regard to the current model is that it should be viewed as a work in progress, as future research will most certainly elucidate the specific factors involved, as well as the relationships that exist between these factors.

The model outlines three areas in which the factors that may contribute to the development and maintenance of PSP lie: general vulnerability factors, antecedents, and consequents. General vulnerability factors themselves do not necessarily lead to the onset of PSP, but rather function as a backdrop and occasionally help explicate reasons for initial bouts of the behavior. For example, biological factors, including a potential genetic predisposition (Bienvenu et al., 2000; Wilhelm et al., 1999), altered neurobiological circuits (Grachev, 1997; Swedo et al., 1991), and altered pain sensitivity (Christenson et al., 1991; Stanley et al., 1992) could all render an individual more vulnerable to the development of PSP. Family dynamics, such as learning or socialization, along with life experiences and comorbidity, may also increase an individual's

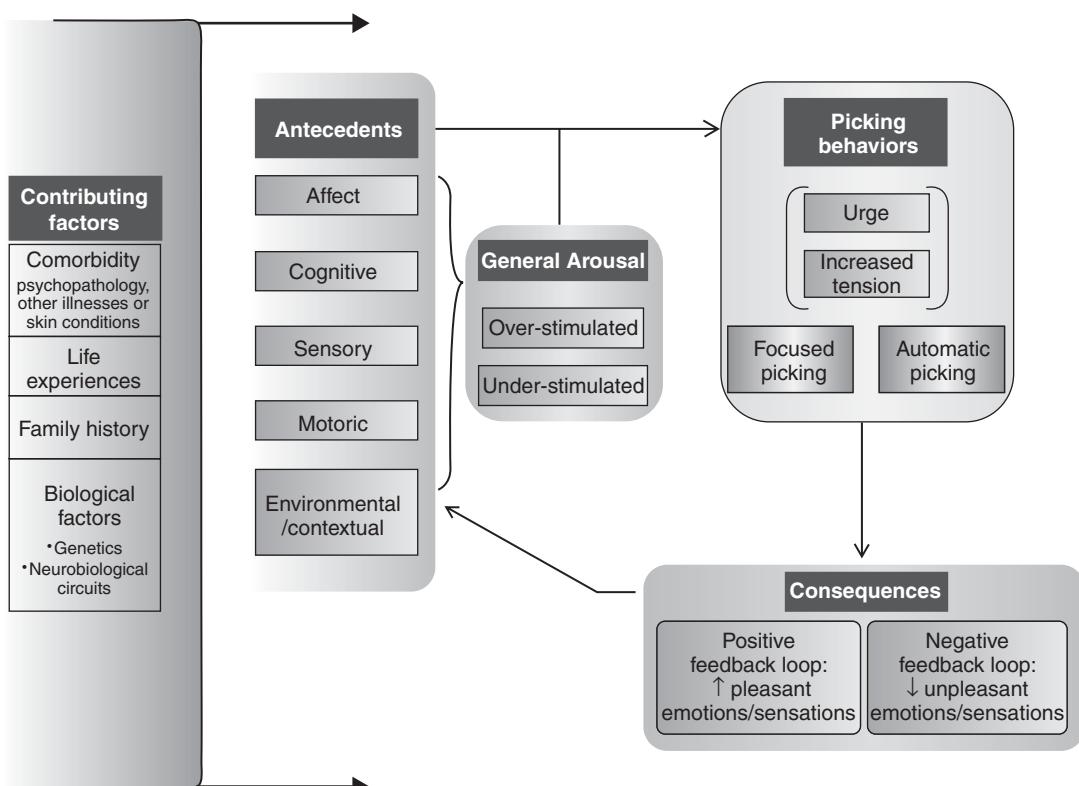


Fig. 27.1 Biopsychosocial model of PSP.

general level of risk for PSP. In particular, the presence of additional conditions or syndromes is important to consider since they may help explain initial occurrences of PSP (e.g., acne) or because the PSP may actually be a symptom of the other condition (e.g., picking as a symptom of body dysmorphic disorder [BDD] or Praeder-Willi syndrome).

The second building block of the model consists of a number of direct antecedents that can increase the chance of in-the-moment PSP. Several authors have discussed how antecedents can be classified as either internal or external in nature (Franklin & Tolin, 2007; Mansueto et al., 1997). Internal cues include affective (e.g., agitation, worry, boredom), cognitive (e.g., "I will feel satisfied if I just pick once" or "People will judge me if they see my scab"), and sensory (e.g., itchiness) phenomena. By contrast, external cues may include motoric triggers (e.g., touching or feeling for skin imperfections) and environmental or contextual cues (e.g., specific situations or visual signs). These factors are often established as triggers via classical conditioning (Azrin & Nunn, 1973; Franklin & Tolin, 2007), wherein the drive to perform the behavior is strengthened when the individual is exposed to something that has previously been linked with PSP. Antecedents may also trigger picking by producing a state of dysregulated arousal in which the individual becomes either hyper- or hypoaroused (Christenson et al., 1993; Franklin & Tolin, 2007). Research has found that feeling both over- and understimulated is linked to PSP (Bohne et al., 2002) and that the behavior may take on the role of a specific emotion regulation strategy (Begotka et al., 2003). Finally, it is possible, though not necessary, for the antecedents to be associated with a building tension in the individual, which results in a subsequent urge to pick.

Once picking begins, it can be classified as either automatic or focused picking. The former often occurs outside the person's awareness, whereas the latter is more likely to be associated with a specific goal (e.g., removing blemishes or regulating emotional distress). The consequences of picking are the final component of the model and represent the reinforcing agent. Within the negative feedback loop, picking serves to remove unpleasant emotions and/or sensations associated with the antecedents of the picking episode. Indeed, many individuals with PSP report that the behavior reduces negative emotions, such as sadness, anxiety, or boredom, along with removing or altering unpleasant sensations or

cognitions (Bohne et al., 2002; Diefenbach et al., 2008; Wilhelm et al., 1999). At the same time, it is also possible for a positive feedback loop to be active. In this pathway, picking behaviors are reinforced by increasing pleasant emotions and/or sensations (Bohne et al., 2002; Meunier et al., 2009). These immediate consequences, which act as explicit behavioral contingencies that maintain the behavior, are distinct from the more long-term consequences of PSP. The delayed outcome of these behaviors often includes a series of detrimental repercussions, such as negative self-evaluation, mood states, social consequences, and possibly even physical scarring (Keuthen et al., 2000; Wilhelm et al., 1999).

Given the complexity of the model, it is clear that very few individuals with PSP will look exactly alike. As such, specific treatment strategies (discussed below in greater detail) may be helpful for one individual and completely ineffectual for another. It is therefore imperative for the clinician to conduct a thorough functional analysis with the patient to identify the most appropriate treatment strategies. For example, an individual with a skin disease (e.g., acne, eczema) may benefit from treatment of the physical condition in conjunction with treatment aimed at the PSP behaviors. In instances where features of BDD are driving the PSP, an individual may need supplementary cognitive-behavioral therapy (CBT) focused on BDD and instruction in more classical response prevention for the PSP. Considering the specific antecedents a given individual may experience, different techniques (e.g., stimulus control or alternative behaviors) could be implemented. With regard to consequences, if the therapist and the client determine that PSP functions primarily to modulate negative affect, techniques focused on emotion regulation and increasing distress tolerance may be most effective. As these examples indicate, each component of the model represents an instance where the cognitive-behavioral techniques for PSP can be uniquely tailored to a given individual's needs so that the most effective treatment is delivered.

Assessment Tools

In what follows, we provide an overview of the most commonly used assessment tools for PSP. These include self-monitoring and functional analysis, photographs, clinician-rated measures, and self-report measures. In light of the diversity of PSP, we again stress the necessity of careful assessment before intervention.

Self-Monitoring and Functional Analysis

Assessment begins with self-monitoring of the frequency and duration of the picking behavior. Particular attention should be paid to the relevant antecedents and consequences of the picking. Although a number of self-monitoring forms exist in the literature, Mansueto and colleagues have developed a particularly useful and detailed monitoring form for BFRBs and especially PSP (Figure 27.2; see also Mansueto et al., 1999). On this form, the patient records the date, times that picking began and ended, site of picking (e.g., the face, arm), intensity of the urge to pick, and efforts to resist picking. The form also allows the patient to note where he or she is and what he or she is doing, feeling, and thinking at the time the picking begins. After the picking has ended, the patient is prompted to write any resultant thoughts, feelings, or physical consequences. Mansueto and colleagues (1999) suggest that assessment should include evaluation of the following five types of antecedents and consequents: cognitive (e.g., having the thought

"That hair is out of place"), affective (e.g., frustration, boredom), motoric (e.g., sitting in a certain position), sensory (e.g., an itch or tingling of the skin), and environmental (e.g., certain places or activities). Particular attention should also be paid to negative (e.g., relief from tension) and positive (e.g., pleasure, sensory stimulation) reinforcers of the behavior. As highlighted in the CBT model, these negative and positive feedback loops may play a crucial role in maintaining the picking behavior. Given the heterogeneous nature of PSP and the high levels of comorbidity, a careful functional analysis of the picking is essential to develop treatment interventions tailored to the patient's unique patterns of behavior.

Photographs

Photographs can provide an objective measure of the degree of picking and associated tissue damage. Twohig and colleagues (Twohig & Woods, 2001; Twohig et al., 2006) have used photographs as primary treatment outcome measures by having blind raters assess photographs of the affected areas for

Self-Monitoring Form for Body Focused Repetitive Behaviors

Day and Date: _____ **Time Began:** _____ **Time Ended:** _____

Location (where were you?)		
Activity (What activity were you engaged in? (E.g., watching TV, night time bathroom routine, etc.)		
Strength of Urges (0–10)	Degree of Awareness (0–10)	
Notable Feelings Prior to Picking		
Notable Thoughts Prior to Picking		
Site(s) of Picking		
Strength of Effort to Resist (0–10)		
Interventions/efforts used to delay, distract, or use substitutes and success of each (0–10)		
Why did you stop when you did?		
Consequences: -Thoughts and Feelings: -Degree of visible damage (0–10)		
What did you do after the picking episode ended?		
Comments and observations		

Note: This form is based on the Comprehensive Behavior (ComB) Model, which was developed by Dr. Charles Mansueto, Ph.D., and his colleagues at the Behavior Therapy Center of Greater Washington.

Fig. 27.2 Self-monitoring form for BFRBs, including pathological skin picking. This form is reproduced with the generous permission of Dr. Charles Mansueto, Ph.D.

degree of damage. Photographic evidence can also provide a concrete measurement of treatment progress for the patient. Of course, clinicians should remain sensitive to any feelings of shame and embarrassment that being photographed may induce.

Clinician-Rated Measures

YALE-BROWN OBSESSIVE COMPULSIVE SCALE MODIFIED FOR NEUROTIC EXCORIATION

The Yale-Brown Obsessive Compulsive Scale Modified for Neurotic Excoriation (NE-YBOCS) is based on the Yale-Brown Obsessive Compulsive Scale (Y-BOCS), a widely used clinician-rated measure of obsessive-compulsive disorder (OCD) symptom severity (Goodman et al., 1989). Like the original Y-BOCS, the NE-YBOCS includes 10 items. Items 1–5 have been modified to correspond to urges to pick or thoughts about picking, and items 6–10 correspond to the picking behavior (Arnold et al., 1999; Bloch et al., 2001; Grant et al., 2007). Each item is rated on a 0 to 4 scale yielding total scores that range from 0 to 40. Although no psychometric papers have been published on the NE-YBOCS, it has demonstrated good test-retest reliability ($\rho = .83$) and good construct validity when compared with other outcome measures in one treatment outcome study (Grant et al., 2007).

SKIN PICKING TREATMENT SCALE

Simeon et al. (1997) also modified the Y-BOCS to create a clinician-rated measure of PSP. The Skin Picking Treatment Scale (SPTS) comprises five items that assess the frequency and duration of picking, the severity of picking, the intensity of the urge to pick, the degree of perceived control over picking, and interference due to picking. As with the original Y-BOCS, each item is rated on a 0 to 4 scale. Total scores range from 0 to 20. Presently, no psychometric data have been reported on this measure, although it has been used in two treatment outcome trials (Bloch et al., 2001; Simeon et al., 1997).

Self-Report Measures

HABIT QUESTIONNAIRE

The Habit Questionnaire is a self-report measure developed by Teng, Woods, Twohig, and Marcks (2002) to assess for the presence of a range of BFRBs including skin picking, mouth chewing, nail biting, skin biting, and skin scratching. For each endorsed behavior, the respondent is asked to rate the frequency and duration of the behavior as well as any associated impairment, injury, medical attention, or

intervention. Respondents also report whether the behavior only occurs under the influence of alcohol or another substance and whether they have ever been diagnosed with any of the following comorbid conditions: OCD, Tourette's syndrome, autism, Asperger's syndrome, or developmental disability. The authors defined the presence of a BFRB as occurring more than five times a day for 4 weeks or longer and resulting in impairment in functioning, injury, medical attention, or intervention. In a sample of 105 undergraduate students, the Habit Questionnaire demonstrated moderate test-retest reliability ($\phi = .69$).

MILWAUKEE INVENTORY FOR THE DIMENSIONS OF ADULT SKIN PICKING

The most recently developed measure of skin picking is the Milwaukee Inventory for the Dimensions of Adult Skin Picking (MIDAS; Walther et al., 2009). Building on previous work in PSP and trichotillomania (Arnold et al., 2001; Christenson & Mackenzie, 1994; Flessner et al., 2008b), Walther et al. sought to develop a measure that would distinguish between automatic and focused picking. The self-report measure includes six items assessing automatic picking (e.g., “I am usually not aware of picking my skin during the picking episode”) and six items assessing focused picking (e.g., “I pick my skin when I am experiencing a negative emotion such as stress, anger, frustration, or sadness”). Each item is rated on a 1 (*not true of any of my skin picking*) to 5 (*true for all of my skin picking*) scale. Thus, scores on each subscale range from 6 to 30. Initial testing of the MIDAS has demonstrated adequate internal consistency (automatic subscale: $\alpha = .77$; focused subscale: $\alpha = .81$) and good construct validity.

SKIN PICKING IMPACT SCALE

The Skin Picking Impact Scale (SPIS; Keuthen et al., 2001a) is a 10-item self-report measure of the emotional (e.g., “I feel embarrassed because of my skin picking”), social (e.g., “I think my social life would be better if I didn’t pick my skin”), and behavioral (e.g., “It takes me longer to go out because of my skin picking”) consequences of skin picking. Respondents rate how much each item applies to them in the previous week on a 0 (*none*) to 5 (*severe*) scale. Thus, total scores range from 0 to 50. The SPIS has shown high internal consistency ($\alpha = .93$) and good construct validity in a sample of self-injurious skin挑者. A cutoff score of 7 or above was shown to reliably distinguish self-injurious skin挑者 from non-self-injurious skin挑者.

SKIN PICKING SCALE

The Skin Picking Scale (SPS; Keuthen et al., 2001b) is a six-item self-report measure modeled after the Y-BOCS (Goodman et al., 1989), assessing the frequency and intensity of the urge to pick and the time spent picking, as well as interference, distress, and avoidance due to skin picking. Each item is rated on a 0 (*none*) to 4 (*extreme*) scale. Thus, total scores range from 0 to 24. Initial testing revealed moderate internal consistency ($\alpha = .80$) and good construct validity. Similar to the SPIS, a cutoff score of 7 or above was shown to reliably distinguish self-injurious skin挑者 from non-self-injurious skin挑者.

SKIN PICKING SYMPTOM ASSESSMENT SCALE

The Skin Picking Symptom Assessment Scale (SP-SAS; Grant et al., 2007) is a 12-item self-report measure of skin picking modeled after two earlier measures of kleptomania (Grant & Kim, 2002) and pathological gambling (Kim et al., 2001). Respondents are asked to rate each symptom over the past week on a 0 to 4 scale. Total scores range from 0 to 48. As with the NE-YBOCS, no psychometric papers have been published on the SP-SAS. It has, however, demonstrated adequate test-retest reliability ($\rho = .74$) and satisfactory construct validity when compared with other outcome measures in one treatment outcome study (Grant et al., 2007).

Review of the Treatment Outcome Literature

Clinical trials designed specifically to treat PSP are few in number, and methodological ambiguities complicate interpretation. The literature is dominated by case reports, along with a few open trials and even fewer controlled and/or double-blind trials, all with small samples. Although definitive conclusions are difficult to draw, the studies suggest the utility of pharmacological and psychosocial treatments, including serotonergic and glutamatergic medications and CBT in treating PSP. In the following sections, we review the treatment outcome literature for PSP.

Pharmacotherapy and Biological Treatment

Simeon and colleagues (1997) conducted a double-blind pharmacotherapy trial in which they randomized 21 individuals with PSP to a 10-week course of fluoxetine (20–80 mg/day; $M = 53–55$ mg/day) or placebo. In completer analyses, fluoxetine led to greater improvement on two of three outcome measures: the Clinical Global Impression-Improvement

Scale (CGI-I) and a self-report visual analog scale rating change in picking, but not the clinician-rated SPTS. Intent-to-treat analyses revealed significantly greater improvement only on the self-report measure. The magnitudes of some nonsignificant effects (SPTS in the completer analyses and CGI-I in the intent-to-treat analyses) were moderate or large, however, suggesting that the differences are meaningful, especially in light of the small sample size and resultant low statistical power. Furthermore, all 6 completers who received fluoxetine (and 8 of 10 who were randomized to receive fluoxetine) achieved clinical responder status compared with 3 of 11 on placebo. Of note, four participants dropped out of the fluoxetine condition: two had improved considerably (CGI-I = 2) and two had worsened considerably (CGI-I = 6), at least one of whose deterioration was clearly caused by the medication.

Four open-label trials of selective serotonin reuptake inhibitors (SSRIs) have been conducted for the treatment of PSP. Bloch and colleagues (2001) investigated fluoxetine in a sample of 15 subjects, all of whom received the study medication (20–60 mg/day) for 6 weeks. Responders ($n = 8$) were then randomized, double-blind, to receive continued fluoxetine at the dosage reached by week 6 or placebo for 6 more weeks. Overall, responders who received continued fluoxetine were considerably improved from baseline to week 12 on measures of skin picking (NE-YBOCS and a 7-item, then-unpublished version of the SPS), whereas those who were randomized to placebo returned to their baseline functioning by week 12. These results are promising, but conclusions regarding the efficacy of fluoxetine should be tempered by the finding that nearly half of the original 15 subjects did not respond to fluoxetine in the first arm of the study. Arnold and colleagues (1999) administered fluvoxamine (25–300 mg/day; $M = 112.5$ mg/day) to 14 subjects over 12 weeks and found improvement in picking behavior and global symptoms (but not in symptoms of depression) on both observer (NE-YBOCS) and self-report (visual analog scales) measures. However, half of the subjects dropped out of the study, four (29%) because of medication-related side effects. Keuthen and colleagues (2007) treated 27 individuals with escitalopram (5–30 mg/day; $M = 25$ mg/day) for 18 weeks. Following treatment, the sample was generally improved on measures of skin picking (SPTS, SPIS, and SPS, as well as the CGI), mood, and quality of life. Of the 27 participants, 20 were classified as full or partial responders and 4 discontinued treatment due to

side effects. Finally, Kalivas and colleagues (1996) report a case series of 31 patients with various self-injurious skin-directed disorders (26 of whom were described as having “neurotic excoriations”) who were treated with sertraline (25–200 mg/day; $M = 95$ mg/day in responders and 72 mg/day in nonresponders) for an unspecified length of time. Three patients were not evaluated due to “intolerable adverse effects” (p. 589). Among the 28 participants who were evaluated, 19 experienced at least 50% reduction in open skin lesions.

Recently, Grant and colleagues (2007) conducted an open-label trial of lamotrigine, an anticonvulsant thought to affect glutamate. Twenty-four subjects were enrolled to receive lamotrigine (12.5–300 mg/day) for 12 weeks. As a group, subjects improved notably on measures of time spent picking as well as on secondary measures. Of the 20 subjects who completed the course of treatment, 16 were deemed responders. Only one of the four dropouts discontinued treatment because of a medication side effect. This study is notably the first to examine a non-SSRI in the treatment of PSP.

In addition to these few clinical trials, numerous case reports document the apparent utility of SSRIs, including fluoxetine (e.g., Gupta & Gupta, 1993), fluvoxamine (e.g., O’Sullivan et al., 1999), escitalopram (e.g., Pukadan et al., 2008), and paroxetine (e.g., Biondi et al., 2000) in treating at least some patients suffering from PSP (for reviews, see Arnold et al., 2001; Grant & Odlaug, 2009). Other authors, however, have published case reports suggesting that SSRIs may actually exacerbate skin picking in some patients (Denys et al., 2003). In addition to SSRIs, other pharmacological agents have also reportedly been effective in treating individual patients, including antidepressants such as clomipramine (e.g., Gupta et al., 1986) and doxepin (Harris et al., 1987); atypical or typical antipsychotics such as olanzapine (e.g., Blanch et al., 2004) and pimozide (Duke, 1983); the putative glutamate modulator, *N*-acetylcysteine (Odlaug & Grant, 2007); and naltrexone (Lienemann & Walker, 1989). Researchers have also reported success in augmenting previously ineffectual courses of some medications (e.g., citalopram, fluoxetine, venlafaxine) by adding other treatments, including atypical antipsychotics such as olanzapine (e.g., Christensen, 2004) and aripiprazole (Carter & Shillcutt, 2006; Curtis & Richards, 2007; Ginsberg, 2006); the antiglutamatergic agent riluzole (Sasso et al., 2006); inositol (Seedat et al., 2001); and CBT (Sahin et al., 2004).

A recent case report suggests that neurosurgery may be effective in reducing dangerous skin picking

in patients who do not respond to other forms of treatment. Kondziolka and Hudak (2008) documented the effectiveness of bilateral anterior capsulotomies using gamma knife radiosurgery in a patient with treatment-refractory skin picking in the context of severe OCD. The patient had not responded to numerous medication trials and courses of behavior therapy, and had received multiple skin grafts, many of which he picked through. By 7 months postsurgery, his skin picking and OCD symptoms had decreased somewhat, and he was accepted for 2 months of inpatient therapy. By 17 months postsurgery, the skin picking was sufficiently reduced to allow the wound to begin to heal, and the OCD symptoms were greatly improved.

Psychosocial Treatment

PSYCHODYNAMIC AND ECLECTIC THERAPIES

In 1953, Seitz published a case series of brief psychodynamic psychotherapy in 25 patients with “psychocutaneous excoriation syndromes” who did not respond to dermatological treatments. Based on earlier case reports and psychological studies of skin disorders, Seitz suggested that scratching is a conversion reaction stemming from “unexpressed rage and guilt, as well as the unsatisfied wishes for love,” and that excoriation releases “tension associated with repressed rage; it atones for guilt by means of mutilating self-punishment; and it provides regressive gratification of the need for love through cutaneous-erotic masturbatory pleasure” (p. 201). Treatment itself consisted of 12 weekly sessions designed to elicit the verbal expression of rage and inferiority and “dilution” (p. 201) of the associated guilt and shame. Seitz reported that all 25 patients experienced temporary exacerbations of the cutaneous disorder, usually at the eighth session, and that 12 of the patients dropped out of treatment. For 12 of the 13 completers, however, the excoriation had resolved by acute posttreatment (i.e., “symptomatic cure,” defined as “clearing of the lesions and absence of itching” [p. 201]), and all 12 maintained their gains at 3-month follow-up. Of those patients available for longer-term follow-up, six of seven were still in remission after 6 months and four of five after 1 year (different patients had relapsed at 6 months and 1 year). Participants who completed treatment differed at baseline from those who discontinued. They had significantly more mild skin lesions and were rated as more motivated. Although uncontrolled, this case series suggests that brief psychodynamic psychotherapy may be efficacious for individuals with mild skin picking who

are motivated. Other reports indicate lower rates of treatment success with psychodynamic or eclectic therapy (e.g., Fruensgaard, 1991a, 1991b).

COGNITIVE-BEHAVIOR THERAPY

Six studies and a number of case reports document the utility of various cognitive-behavioral techniques in treating PSP. These techniques are often implemented in combination with each other and are described in more detail below (see "Cognitive-Behavioral Techniques: A Clinician's Toolbox").

Only one between-groups controlled trial of CBT for PSP has been conducted. Teng, Woods, and Twohig (2006) compared brief habit reversal training (HRT; Azrin & Nunn, 1973; Azrin & Peterson, 1988) to no treatment (waiting list control; WL) in a sample of 19 chronic skin pickers. In this study, HRT consisted of three weekly meetings: a single 1-hour session of awareness and competing response training, followed by two half-hour booster sessions. Competing response training involved "participants [learning] to clench their fists for 1 min . . . contingent on the skin picking and its antecedents" (p. 416). Teng and colleagues found that HRT outperformed WL on self-monitored tallies of skin-picking frequency (77% versus 16% reduction in self-reported skin picking in the HRT and WL groups, respectively) and photograph ratings, and noted that the gains from HRT were maintained for at least 3 months. None of the participants had entirely ceased picking at posttreatment, however. Nevertheless, these results suggest that HRT can be an effective and fast-acting intervention for individuals with chronic PSP.

In addition to the study by Teng and colleagues (2006), two other studies have examined HRT for PSP using experimental designs. Using a multiple baseline across participants design, Twohig and Woods (2001) treated two brothers who engaged in damaging picking of their fingers with brief HRT similar in form and length to the treatment used by Teng et al. The frequency of picking and skin damage decreased for both participants after implementation of treatment; however, only one of the participants fully maintained his gains at follow-up. Lane and colleagues (2006) reported on the use of "competing activities" for a 9-year-old boy with PSP and comorbid attention deficit hyperactivity disorder (ADHD), low IQ, and learning and speech-language difficulties. At the beginning of each day, the boy was given a choice of three plastic balls "to keep his hands occupied so as to avoid skin picking" (p. 460). Using an ABCBAB design, Lane and

colleagues measured the percentage of time spent picking over the course of 29 days during which the child received medication only (Adderall for ADHD), medication with the intervention, or the intervention alone. The use of competing activities decreased the time spent picking (measured via direct observation) and appeared most effective when combined with medication for ADHD. No follow-up data or measures of long-term improvement in picking or the urge to pick were reported.

Two studies have examined acceptance-based forms of CBT using multiple-baseline designs. Twohig and colleagues (2006) examined the treatment of five chronic skin pickers with eight weekly 1-hour sessions of acceptance and commitment therapy (ACT; Hayes et al., 1999). Four of the five participants evidenced considerable treatment gains (i.e., low levels of self-monitored skin picking frequency and improvement in visible skin damage) at acute posttreatment; however, only one maintained the gains at 3-month follow-up. The results of this study suggest that ACT alone can be helpful, at least in the short term, but that "additional treatment development is needed particularly in the maintenance of gains" (p. 1520). It is noteworthy that the use of HRT techniques was explicitly proscribed in the aforementioned study. To evaluate the utility of combining techniques of ACT and HRT, Flessner and colleagues (2008a) conducted a pilot study of acceptance-enhanced behavior therapy in five individuals with chronic PSP or trichotillomania. The two participants with PSP improved (50% reduction in skin-picking severity), although somewhat less than did those with trichotillomania (65% reduction). Both participants with PSP initially received HRT but did not evince improvement until the addition of ACT techniques. It is impossible to know whether the addition of ACT techniques per se was responsible for the improvement (e.g., as opposed to continued time and effort in treatment).

There is evidence, as well, for the effectiveness of an Internet-based self-help program that follows the principles of HRT for PSP. In the only large-scale study of CBT for PSP ($N = 372$), Flessner and colleagues (2007) conducted an uncontrolled program evaluation of the computer-based intervention at www.StopPicking.com. The intervention lasted an average of 12 weeks and comprised an assessment phase, during which individuals monitored picking behavior and its associated antecedents and consequences; an intervention phase, which included various cognitive-behavioral coping strategies

(e.g., stimulus control, use of competing responses, relaxation, cognitive restructuring); and a maintenance phase. Postintervention, 63% of participants were deemed responders on the basis of achieving a 25% reduction in scores on the SPS (Keuthen et al., 2001b).

A few other case reports have added to the literature suggesting that PSP can be treated successfully and relatively quickly with HRT. In four patients with neurodermatitis, Rosenbaum and Ayllon (1981) found that a single treatment session induced a marked decrease in skin picking for at least 6 months. In another report, a woman with acne excoriée was able to reduce the time she spent picking from 14 hours to less than 10 minutes per week after 1 month of HRT, and the gains were still evident after 4 months (Kent & Drummond, 1989). Noting the difficulty of making inferences from treatment outcomes in skin picking associated with a dermatological condition to cases of picking without any such condition, Deckersbach and colleagues (2002) reported three cases of people with chronic skin picking in the absence of a dermatological condition and in patients with psychiatric comorbidity. All were treated with CBT that included HRT but was supplemented with other techniques, such as those to increase emotion regulation skills. In two cases, relatively brief courses of treatment (four and seven sessions) were associated with considerable behavioral gains, and in the third, the patient ceased picking after 2 years of therapy, six sessions of which were focused on skin picking. Deckersbach et al. (2003) treated a woman with severe PSP (automatic and focused) related to BDD with eight sessions of CBT that included HRT, cognitive restructuring, and emotion regulation training. Over the course of treatment, the patient decreased the frequency of skin-picking episodes from 15–20 per day to fewer than 3 per day, and her self-report symptom scores decreased by approximately 50%. These gains were maintained at 3-month follow-up. Of note, she evinced improvements in automatic skin picking more rapidly than in picking that served an emotion regulation function. Finally, Welkowitz et al. (1989) described the successful treatment of a man with excoriations at multiple sites with an intervention that included various behavioral strategies similar to elements of HRT.

Summary of Outcome Literature

Although large-scale studies are lacking, one double-blind study and five open-label trials suggest that SSRIs or glutamatergic agents may be efficacious in

treating PSP for at least a subgroup of individuals. Enthusiasm is reduced by high or equivocal rates of nonresponse in some studies as well as notable dropout rates. Furthermore, the lack of clear diagnostic criteria and the use of variable outcome measures complicate interpretation (limitations common to studies of psychosocial treatments as well). Moreover, it is not known whether the efficacy of continued pharmacological treatment wanes over time. Case reports document the possibility that various other pharmacological agents may improve treatment outcome when used instead of, or to augment, serotonergic medications. These await further investigation.

One between-groups study and several small experimental paradigms suggest that CBT has utility in treating PSP and has the potential to induce gains quickly. Cognitive-behavior therapy treatments for PSP include HRT, acceptance and mindfulness, and various other techniques that are now described in more detail.

Cognitive-Behavioral Techniques: A Clinician's Toolbox

As is evident from the treatment outcome literature, a number of CBT techniques are helpful for patients with PSP, often in combination with each other. They include HRT, stimulus control, acceptance and mindfulness, cognitive strategies, relaxation training, emotion regulation, and various other tools. The population of individuals who engage in PSP is quite heterogeneous with respect to the underlying pathology, and the formulation of a treatment plan as well as the selection of intervention techniques must be tailored to the individual (see “Cognitive-Behavioral Model of Skin Picking”). In the following section, we describe various CBT techniques with utility in treating individuals with PSP.

Habit Reversal Training

Habit reversal training is an approach to treating habitual behaviors of many types (Azrin & Nunn, 1973; Azrin & Peterson, 1988) that incorporates a number of specific techniques at least some of which are present in many variations of CBT for PSP. The techniques include awareness training, the use of competing responses, and relaxation (described separately below).

Chronic skin pickers are frequently not aware of their picking, especially automatic pickers. Even those who engage in focused picking may not be cognizant of the psychological and environmental antecedents or consequences of picking, or the

specific sequence and nature of the picking behavior itself. Hence, prerequisite to choosing an appropriate intervention strategy and, of course, implementing it in real time, is that the patients identify and recognize when and precisely how they pick. Awareness training is typically conducted both in and out of session and involves monitoring the occurrence of picking episodes (e.g., by tallying the number of such episodes and/or urges during set periods of time), as well as describing, frame by frame, what transpires before, during, and following picking. Ultimately, automaticity in detecting warning signs of picking is a critical antidote to the automaticity of habitual picking.

Competing responses are behavioral alternatives that are used to contravene episodes of picking, and involve engaging in behavior opposite to, and incompatible with, the patient's idiosyncratic picking behavior. For example, a patient who picks with her fingernails might make a fist or hold an object whenever she experiences the urge to pick. The patient is taught to select a competing response that can be maintained long enough for the urge to pass (minimum 1 minute) and that does not disrupt necessary activities during potential picking episodes (e.g., socially inconspicuous for someone who picks at work). Targeted practice and reinforcement are used to facilitate the application and generalization of competing responses.

Stimulus Control

To the extent reasonable, environmental antecedents and triggers are manipulated to decrease the likelihood of picking or the intensity of the urge to do so. Patients who engage in automatic picking can avoid behaviors and situations during which they mindlessly pick. For example, someone who watches television or reads with his chin resting in his palm, which leads to mindless skin touching and ultimately picking, may intentionally keep his hands away from his face (e.g., in his pockets or otherwise occupied) during those activities. Similarly, he may engage in prophylactic use of competing responses upon entering situations of risk. Patients who engage in focused picking can also find ways to disrupt the cycle of picking. For example, a patient who picks in the mirror may remove the mirror, alter the lighting in the room, or smear petroleum jelly on the mirror.

Acceptance and Commitment Therapy

Acceptance and commitment therapy (ACT; Hayes et al., 1999) is a form of CBT that emphasizes the

roles of experiential avoidance and behavioral rigidity in maintaining dysfunctional behavior. Acceptance and commitment therapy is highly compatible with HRT in treating PSP, and some have suggested that ACT may be particularly well suited for individuals who engage in focused picking to regulate emotion, as opposed to automatic picking (Flessner et al., 2008a). The full ACT treatment protocol is available on the Internet (http://www.contextualpsychology.org/treatment_protocols); it is written for OCD, but can be applied to skin picking by changing the term *obsession to urge to pick* (M. Twohig, personal communication, August 24, 2009).

As implemented by Twohig et al. (2006), ACT for PSP involves five steps that are learned and practiced over the course of 8 weeks:

1. Efforts to eliminate urges to pick, as well as other internal events (e.g., affective or motivational), are not effective. Hence, whereas many individuals who engage in pathological habitual behavior struggle to resist the negative affective experience of the urge itself, efforts to do so are highly unlikely to succeed.

2. Moreover, attempts to control urges themselves create or maintain the problem because in many cases they paradoxically exacerbate or magnify the internal experience or perceived lack of efficacy in managing it.

3. There is a difference between uncontrollable urges and controllable behaviors. Whereas efforts to resist the experience of an urge are likely unhelpful, those to resist the behavioral response to the urge (e.g., picking) are feasible, provided that one is willing to experience the discomfort of the urge. The mechanism of treatment is therefore not to decrease the urge, but rather to eliminate the picking.

4. Withstanding the urge to pick is difficult, especially if the urge itself is perceived as harmful or threatening in some way. Various exercises are introduced and practiced to alter the perception of verbal events (e.g., thoughts, urges) as threatening or overly meaningful.

5. Ultimately, rather than succumb to urges by choosing unwanted behavior designed to avoid or neutralize internal experiences such as urges or affective states, the individual learns to pursue valued action whatever the immediate internal cost. That is, the patient learns to act rather than react. In the case of PSP, the patient chooses not to pick because he or she does not wish to pick, without regard to whether that choice is likely to decrease the experience of negative internal states.

Cognitive Strategies

In contrast to the use of HRT and ACT strategies, which are fundamentally suited to most individuals with PSP, the choice to supplement CBT with cognitive techniques is particular to the patient's clinical manifestation. For example, Deckersbach et al. (2002) describe patients whose picking episodes were often triggered by negative emotions such as sadness, some of which were fueled by dysfunctional beliefs (e.g., "My face looks horrible" [p. 371]; "I will never find a job" [p. 371]; "I cannot handle this" [p. 372]; "I have to get rid of this scab" [p. 372]). In such cases, cognitive restructuring (e.g., Beck, 1995) can be helpful to address underlying beliefs and assumptions. In general, CBT approaches to skin picking typically incorporate cognitive techniques as a flexible and optional module when appropriate. Often these strategies are most relevant when the urge to pick is triggered or accompanied by dysfunctional beliefs about oneself or one's ability to manage distress and regulate emotions.

Relaxation Training

Picking behavior may exacerbate and be exacerbated by generalized stress and tension. Hence, HRT frequently incorporates relaxation training, which can be used regularly as a means of lowering baseline stress, as well as strategically as a complement to competing responses to resist acute urges to pick.

There are numerous variations of relaxation training that differ in terms of complexity and refinement. Progressive muscle relaxation (Bernstein & Borkovec, 1973) and applied relaxation (Öst, 1987) are formal protocols through which an individual learns to relax by systematically tensing and then relaxing various muscle groups. Ultimately, the individual learns to engage in cued (without tensing, and in response to a self-generated cue) and differential (while performing another activity) relaxation and to apply relaxation *in vivo*. As such, relaxation training is a bona fide treatment with demonstrated efficacy for a number of disorders (e.g., Manzoni et al., 2008; Öst, 1987). However, less comprehensive relaxation training can be used as an adjunct to other CBT techniques. This can include simplified versions of muscle relaxation (e.g., focusing on four muscle groups; cf. Wolpe & Lazarus, 1966) as well as diaphragmatic breathing. Implementation of relaxation should be tailored to the individual, who may find utility in using it to lower generalized tension, resist the urge to pick, and/or regulate emotion.

Emotion Regulation

Clinicians have noted that some individuals engage in focused PSP to modulate negative affect or emotion dysregulation. Moreover, anecdotal evidence and studies of trichotillomania suggest that self-injurious BFRBs with an emotion regulation quality may respond less well or more slowly to straightforward behavioral treatment (e.g., Deckersbach et al., 2002, 2003; Flessner et al., 2008a). Therefore, individuals for whom picking is triggered by affective distress or who pick to self-sooth (common, for example, in individuals with borderline personality disorder) may benefit from the development of emotion regulation and distress tolerance skills. Specific techniques include relaxation, exercise, participation in enjoyable activities, acting opposite to the distressing emotion, distraction, and other forms of self-soothing (for a comprehensive list of emotion regulation and distress tolerance strategies, see Linehan, 1993).

Other CBT Techniques

At times, PSP is manifest as an epiphenomenon in the context of another disorder. When it is symptomatic of another underlying disorder, intervention strategies that focus exclusively on the picking behavior are unlikely to be sufficient. In such cases, developing a treatment plan requires attention not only to the behavior itself, but to the underlying symptoms and motivation to pick as well. For example, more than one-third of patients with BDD engage in skin picking, often to smooth or remove perceived blemishes (Grant et al., 2006). For those individuals, the BDD treatment protocol would likely incorporate cognitive restructuring, as well as exposure and response prevention, HRT, and stimulus control techniques. Habit reversal training would be useful to facilitate response prevention (i.e., resisting the urge to pick during exposures) but would not adequately address the BDD by itself.

Future Directions

Research on PSP has grown in recent years; however, treatment studies are notably lacking in number and size. Considering the widely discrepant manifestations of PSP behavior, this significantly limits the ability to make clinical inferences on the basis of the empirical literature. Certainly, larger comprehensive investigations are warranted. In addition, future research would benefit from attention to a number of issues that follow.

We have emphasized throughout this chapter the heterogeneity of PSP. Indeed, examination of the treatment studies and case reports reveals samples drawn from a range of populations that differ on factors one might presume to predict treatment outcome, including type of picking (e.g., automatic versus focused) and diagnosis (e.g., the presence or absence of a dermatological condition, psychiatric disorder, and comorbidity). The severity of picking itself is not consistent, ranging from minor finger picking to dangerous self-mutilation. The extent to which these factors affect treatment is virtually unknown. Considering a condition with such variability, Paul's fundamental question of psychotherapy research is particularly salient: "What treatment, by whom, is most effective for this individual with that specific problem, and under which set of circumstances?" (1967, p. 111; emphasis in the original).

Larger studies would not only engender greater confidence in the efficacy of treatments for PSP (or lack thereof), but would permit examination of the clinical observation that subtypes of PSP respond differentially to various treatments (Grant & Odlaug, 2009). For example, a number of researchers have suggested that automatic picking is more responsive to behavioral interventions than is focused picking designed to regulate emotion. Therefore, mindfulness and emotion regulation skills may be particularly helpful for individuals who engage in the latter (Deckersbach et al., 2002, 2003; Flessner et al., 2008a). Similarly, a subset of patients with PSP seem not to respond to SSRIs (Keuthen et al., 2007) or even to deteriorate (Denys et al., 2003). Keuthen and colleagues (2007) offer the possibility that such differential outcome depends on whether the PSP behavior is impulsive or compulsive (see also Arnold et al., 2001). Without larger, systematic studies, however, such accounts are speculative.

Even to the extent that studies that directly examine moderation are not feasible, efforts to articulate and standardize diagnostic criteria would markedly facilitate the conduct and comparative interpretation of treatment outcome studies, as well as the quality of empirically grounded clinical care (Bohne et al., 2002). Likewise, the field would benefit from further evaluation (e.g., psychometric properties) of measures that permit careful assessment of PSP and, ultimately, treatments better tailored to the individual. Existing studies are difficult to compare in part because they utilize a variety of outcome measures, many with unknown psychometric

properties. This complicates even the evaluation of severity.

Finally, efforts to improve treatment outcome are generally warranted. Wilhelm et al. (1999) report retrospective data that suggest rather limited benefit from both behavioral and pharmacological treatments for most individuals with PSP. In addition, rates of nonresponse remain high in some pharmacological studies, and little is known about psychosocial treatment response rates. Future research should examine whether, as suggested by numerous case reports, combining medications and/or psychotherapy techniques can increase treatment success (Grant & Odlaug, 2009).

Author Note

The authors wish to thank Nancy Keuthen, Ph.D., for suggestions and guidance.

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Eli Coleman

Abstract

There is a growing recognition among clinicians that any type of sexual behavior can become pathologically impulsive or compulsive. There is quite a bit of debate about terminology for this condition, the diagnostic criteria, assessment methods and treatment approaches. In the absence of clear consensus, clinicians are struggling with how to help the many men and women who suffer and seek help from this type of problem. This chapter will review the author's assessment and treatment approach. Clinicians will need to keep abreast of the literature as new research evolves and follow the continued debate around this controversial area.

Keywords: impulse control, sexual behavior, assessment, treatment

As with many other pleasurable and self- and other-enhancing sexual behaviors, there is growing recognition among clinicians that sexual behavior can become pathologically impulsive or compulsive (Coleman, 1991; Coleman et al., 2003). Normal, healthy sexual behavior is often impulsive, driven, and thought about continuously, and the satisfaction of sexual behavior is often accompanied by a sense of abandonment and loss of control. So, when does the sexual behavior become pathological and in need of treatment? How is this assessed, and what are the treatment approaches? These are vexing questions that are to some degree unanswered and are under intense debate. While the paraphilic disorders have been recognized as clinical disorders, there have been serious challenges to the evidence presented to support their continued classification in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) and the World Health Organization's *International Classification of Diseases* (ICD; e.g., Moser, 2009). Currently, there is no diagnostic category syndrome in the DSM-IV-TR (APA, 2000) for normophilic behavior

that is taken to its impulsive/compulsive extreme. Yet, many men and women suffer from this problem and seek help for it, and clinicians are in need of guidance from the best information available at this time.

A Debate Over Terminology: What Do We Call It?

There has been great debate about the terminology and etiology of this clinical syndrome (Coleman, 2003). It is not newly described in the literature, but it has been described by many different terms: *hypersexuality*, *hyperphilia*, *erotomania*, *satyriasis*, *promiscuity*, *Don Juanism*, *Don Juanitaism*, and, more recently, *sexual addiction*, *compulsive sexual behavior*, and *paraphilia-related disorder* (e.g., Carnes, 1983; Coleman, 1991; Goodman, 1993; Kafka & Prentky, 1994; Levine, 1982; Money, 1986). The terminology has often implied different values, attitudes, and theoretical orientations, and the debate continues regarding classification, causes, and treatment (Coleman, 1986, 1990, 1991, 1992). In the ICD (Version 10), there is a category of Excessive

Sexual Drive. Examples of this are *nymphomania* and *satyriasis*. Most recently, the committee assigned to recommend revisions to the current DSM has suggested the term and a new category of sexual disorders: *hypersexual disorder* (APA, 2010). There has never been such a category in DSM. However, since the publication of the DSM-III in 1980, the category Sexual Disorder Not Otherwise Specified has included an example of Don Juanism ("distress about a pattern of repeated sexual relationships involving a succession of lovers who are experienced by the individual only as things to be used"; APA, 2000, p. 582)

The rationale for and empirical evidence in support of the proposal of the DSM task force is elaborated in the review by Kafka (2009). He argues that there are three extant pathophysiological models to describe this syndrome: sexual arousal/desire dysregulation, sexual addiction, and sexual compulsivity.

Kafka argues that mental health consumers and providers have a strong clinical need to recognize and diagnose a distinct group of men and women who have been seeking help for this problem in various ways (psychotherapy, 12-step support groups, and pharmacotherapy), yet there is no diagnostic category at present. In the absence of a diagnostic category, these individuals have been diagnosed with Sexual Disorder Not Otherwise Specified (APA, 2010).

Besides the clinical need, Kafka and the DSM workgroup argue that significant research is needed to consolidate an operational definition. "A DSM-V-based empirically derived definition should significantly enhance research efforts to explore some of the additional diagnostic validators for which there are no current data" (APA, 2010).

Kafka and the DSM workgroup argue that the paraphilic disorders are the nearest diagnostic category and that there may be comorbidity with some of these other paraphilic behaviors. However, they state that the category Hypersexual Disorder involves normophilic behavior rather than deviant or social anomalous behavior and is distinct in and of itself.

The DSM workgroup acknowledges that much research is needed to understand this disorder but that there is sufficient evidence to move this proposed disorder into field trials and possible inclusion in DSM-V. Clinicians will need to follow this debate, and it is uncertain how this clinical syndrome will be classified in the revisions of the DSM or ICD. There is also the possibility that this syndrome could be classified under the proposed new

category of Behavioral Addictions (APA, 2010; Holden, 2010). For the first time, this general category has been suggested. Some say this is a change that is long overdue; others, however, are more cautious (Holden, 2010). So far, only one behavior has been proposed for inclusion: Disordered Gambling. Pathological Gambling was included as a diagnostic category in previous versions of DSM but under Impulse Control Disorders Not Elsewhere Classified. Some argue that problematic sexual behavior should be included as well. This category is proposed to be subsumed under a newly proposed umbrella category of Addiction and Related Disorders (including Alcohol and other drug use disorders). It is unclear what the outcome of this debate will be.

The proposed category of Hypersexual Disorder at least recognizes the various pathological pathways and understands this as a complex psychosexual disorder; however, the category still connotes that the behavior may be "more than" or conveys the notion that the behavior is driven by hypersexual drive dysregulation. The problem with the category of behavioral addiction is that it assumes that it has commonalities in clinical expression, etiology, comorbidity, physiology, and treatment with Substance Use Disorders. As Kafka has pointed out, this is probably only one pathophysiological mechanism or pathway to the disordered behavior (Kafka, 2009). Treating impulse control sexual behavior as an addiction has been criticized for many reasons (Coleman, 2003). The issue of nosology will probably not be resolved until at least the revision of DSM to be released in 2013, and I suspect will remain a controversial issue.

In my work and throughout this chapter, I use the term *impulsive/compulsive sexual behavior* (ICSB) to describe this syndrome. I prefer this term at this point because of its descriptive nature, and it leaves open the possibility of multiple pathological pathways and treatments. In previous work, I have used the term *compulsive sexual behavior*; however, I have seen this term as limiting, and it failed to recognize that some individuals have more problems with impulse control rather than with obsessive-compulsive mechanisms (Raymond et al, 2003). Since they could be of either type, I have chosen to add *impulsive* to describe the phenomenon.

Impulsive/compulsive sexual behavior is a putative clinical syndrome characterized by the experience of sexual urges, sexually arousing fantasies, and sexual behaviors that are recurrent, intense, and cause distressful interference in one's daily life

(Coleman, 1987, 1991, 1992; Coleman et al., 2003). Individuals with ICSB often perceive their sexual behavior to be excessive, but they are unable to control it. They act out impulsively (act on impulses and lack impulse control) and/or compulsively (being plagued by intrusive obsessive thoughts and driven behaviors; Carnes, 1991; Coleman, 1991; Coleman et al., 2003; Kafka, 1991). There are several important aspects to this behavior: it can involve fantasies and urges as well as behavior. In addition, the behavior must reach a level of clinically significant distress and interference in one's daily life. More specific criteria will be discussed in the section on assessment.

There are two different types of ICSB: paraphilic and nonparaphilic. In many ways, I share Kafka's view that nonparaphilic ICSB is more similar to the paraphilias. The only main difference is that one involves normative sexual behavior and the other involves socially anomalous behavior or that which is considered deviant.

Paraphilic ICSB

Money (1986) defined paraphilias as "a condition occurring in men and women of being compulsively responsive to and obligatively dependent upon an unusual and personally or socially unacceptable stimulus, perceived or in the imagery of fantasy, for optimal initiation and maintenance of erotosexual arousal and the facilitation or attainment of orgasm" (p. 267). He continued, "In legal terminology, a paraphilia is a perversion or deviancy; and in the vernacular it is kinky or bizarre sex" (p. 267). Thus, paraphilic ICSB involves sexual behaviors that are not only impulsive or compulsive and repetitive, but also unconventional or socially deviant (APA, 2000; Money, 1986, 1988).

The paraphilias are categorized and have distinct diagnostic criteria in the *Diagnostic and Statistical Manual of the American Psychiatric Association* (2000). In the DSM-IV-TR, paraphilias are defined as "recurrent, intense sexually arousing fantasies, sexual urges, or behaviors generally involving (1) nonhuman objects, (2) the suffering or humiliation of oneself or one's partner, or (3) children or other non-consenting persons" (p. 566). The definition goes on to explain that "The behavior, sexual urges, or fantasies cause clinically significant distress in social, occupational, or other important areas of functioning" (p. 566). Paraphilias may involve illegal behavior, such as an adult having sex with a minor. However, not all sexual offenses are committed by people who meet

the diagnostic criteria for one who has a paraphilia. That said, most paraphilias do cause interference in intrapsychic and/or interpersonal functioning.

The eight paraphilias listed in the DSM-IV TR are pedophilia, exhibitionism, voyeurism, sexual sadism, sexual masochism, fetishism, transvestic fetishism, and frotteurism (see Table 28.1).

The DSM workgroup on paraphilic disorders has recommended a number of changes to be included in the next revision of the DSM (DSM-V). Most notably, the workgroup has suggested that a distinction needs to be made between a paraphilia and a paraphilic disorder. "A paraphilia by itself would not automatically justify or require psychiatric intervention. A *paraphilic disorder* is a paraphilia that causes distress or impairment to the individual or harm to others. One would *ascertain* a paraphilia (according to the nature of the urges, fantasies, or behaviors) but *diagnose* a paraphilic disorder (on the basis of distress and impairment). In this conception, having a paraphilia would be a necessary but not a sufficient condition for having a paraphilic disorder" (APA, 2010).

Table 28.1 Eight Paraphilias Defined in DSM-IV-TR

Pedophilia	Sexual arousal to an age range restricted to prepubertal or peripubertal children
Exhibitionism	Sexual excitement by evoking emotional response from a stranger by illicitly exhibiting an erotic part of the body
Voyeurism	Sexual arousal through the risk of being discovered while watching a stranger
Sadism	Sexual arousal to being the authority who demands discipline and imposes humiliation, abuse, torture, and punishment
Masochism	Sexual arousal by being the recipient of abuse, torture, punishment, discipline, humiliation, obedience, and servitude
Transvestism	Sexual arousal by wearing clothing of the other sex
Fetishism	Sexual arousal evoked by a particular object, substance, or part of a partner's body
Frotteurism	Sexual arousal through rubbing the genital area against the body of a stranger in a crowd

The DSM workgroup has also suggested a change in the classification of *pedophilia* and has suggested the term *pedohebophilic disorder*. The workgroup feels that a new term would better capture the range of ages to which pedophiles are attracted. One set of specifiers for pedohebophilic disorder would allow the clinician to record whether the patient is most attracted to prepubescent (Tanner 1) children, most attracted to pubescent (Tanner 2–3) children, or equally attracted to pubescent and prepubescent children. The workgroup has also suggested an additional category of Paraphilic Coercive Disorder. There has never been a category for people who are aroused by rape fantasies, which is common among individuals who commit rape.

The DSM workgroup has left the Paraphilia Not Otherwise Specified category for more unusual and less common paraphilic behaviors. John Money created a taxonomy of almost 50 distinct types of paraphilic behaviors including zoophilia (bestiality), asphyxiophilia (cutting off oxygen to increase or enhance sexual arousal or orgasm), and necrophilia (sex with dead people), to name a few (Money, 1986).

The DSM workgroup is obviously responding to the criticism that the current criteria may be over-pathologizing normative sexual variations. The workgroup has attempted to develop clearer diagnostic criteria with more behavioral indicators rather than the more subjective criteria used in DSM-IV-TR. However, in the process, they have removed the subjective term *recurrent*, which could lead to overdiagnosis since the threshold is low—only a few occasions or a small number of victims. These proposed improvements will certainly be met with criticism and will ultimately be tested in field trials. Clinicians will have to follow these developments, as these represent significant potential revisions of classification and criteria.

Nonparaphilic ICSB

In contrast to paraphilic ICSB, nonparaphilic ICSB involves normative and conventional sexual behavior that is engaged in recurrently and intensely, yet with similar negative consequences and distress (Coleman, 1991, 1992; Coleman et al., 2003). It involves normophilic behavior. *Normophilia* is “a condition of being erotosexually in conformity with the standard as dictated by customary, religious, or legal authority” (Money, 1986, p. 266). This type of ICSB can cause considerable interference in pair-bonding relationships (e.g., Coleman, 1991, 1995). There are also high risks associated with sexually transmitted infections (STIs), including HIV infection (Coleman,

et al., 2009; Kalichman & Cain, 2004; Kalichman et al., 2005). Besides STIs, unintended pregnancies can result (Henshaw, 1998; McBride et al., 2008). In addition, with the explosion of the Internet, viewing of pornography at home and at the workplace has become a serious problem (Cooper et al., 2002). This is one of the most common types of ICSB that we are seeing clinically today. Also, there are far more cybersex users who use the Internet to seek partners and who are at greater risk for STIs (Coleman et al., 2009; McFarlane et al., 2000).

There is no consensus on the types of non-paraphilic ICSB. There are at least seven subtypes: compulsive cruising and multiple partners, compulsive fixation on an unattainable partner, compulsive autoeroticism (masturbation), compulsive use of erotica, compulsive use of the Internet for sexual purposes, compulsive multiple love relationships, and compulsive sexuality in a relationship (Coleman et al., 2003; see Table 28.2).

Besides advocating for a new category of Hypersexuality Disorder, the DSM workgroup revision committee recommended a set of diagnostic criteria and specifiers for the type of “hypersexual” behavior. In their conceptualization, they have recommended the following specifiers:

- Masturbation
- Pornography
- Sexual Behavior with Consenting Adults
- Cybersex
- Telephone Sex
- Strip Clubs
- Other

While this list is similar to the seven types mentioned, the proposed specifiers are behavioral rather than dynamic.

In the past, we have used a slight alteration of the paraphilia diagnostic criteria and this represents an amalgam of criteria we have used (Coleman et al., 2003; (Raymond et al., 2003):

- A. Over a period of at least six months, recurrent intense sexually arousing behaviors, sexual urges, or fantasies that involving one or more of the following:
 - (1) Compulsive cruising and multiple sexual partners
 - (2) Compulsive fixation on an unattainable partner
 - (3) Compulsive autoeroticism
 - (4) Compulsive use of the Internet for sexual purposes

Table 28.2 Types of Nonparaphilic ICSB

Compulsive Cruising and Multiple Partners

• Constantly searching or “scanning” the environment for a potential partner. Relentless search to find, conquer, and satisfy the demand for a sexual outlet. Insatiable demand for multiple partners as part of a strategy for management of anxiety and maintenance of self-esteem

- Cruising as ritualistic and trance-inducing
- Partners as “things to be used”

Compulsive Fixation on an Unattainable Partner

- Compulsive fixation on an unattainable partner despite lack of a reciprocal response
- Elaboration on fantasies without the intrusion of reality
- Fueling the fantasy by the potential and fantasized reciprocation of love
- Idealizing and fictionalizing the love object

Compulsive Autoeroticism

- Obsessive and compulsive drive toward sexual self-stimulation of the genitalia
- Cessation of masturbation caused by exhaustion, injury, or extreme social pressure rather than sexual satisfaction
- Loneliness felt keenly after an orgasm
- Common practice of masturbating 5–15 times a day
- Common experience of physical injury
- Interference with occupational, social, interpersonal, and intimacy functioning

Compulsive Use of Erotica

- Obsessive and compulsive drive to seek sexual stimulation through erotica
- Hiding, hoarding, and/or compulsive collecting of erotic materials
- Spending excessive amounts of money seeking/buying erotica

Compulsive Use of the Internet

- Obsessive and compulsive use of the Internet in seeking sexual gratification
- Compulsive chatting, seeking of fantasized sexual partners
- Spending excessive amounts of time online, causing interference with occupational, social, interpersonal, and intimacy functioning

Compulsive Multiple Love Relationships

- Obsession and compulsion in finding the intense feeling of a new relationship
- Lack of capacity to freely choose multiple love relationships
- Fantasy and role playing essential in relationships; reality is intrusive
- Highly skilled romance artist

Compulsive Sexuality in a Relationship

- Compulsive expressions of sexuality in a relationship
- Demanding sexual expression through manipulation, coercion, or violence
- Absence of expression of sexuality that results in anxiety, depression, and anger
- Unending need for sex, expressions of love, attention, and signs of affection that temporarily relieve anxiety
- Relationships characterized by intense possessiveness, jealousy, and anger

- (5) Compulsive use of erotica
(6) Compulsive multiple love relationships
(7) Compulsive sexuality in a relationship

B. The fantasies, sexual urges or behaviors cause clinically significant distress or impairment in social, occupational or other important areas of functioning.

C. Not due to another medical condition, substance use disorder, or attributable to another Axis I or II disorder such as mania or a developmental disorder. Must take into account norms of gender, sexual orientation and sociocultural groups.

D. Duration of at least 6 months.

The APA proposed diagnostic criteria for DSM-V are (APA, 2010):

A. Over a period of at least six months, recurrent and intense sexual fantasies, sexual urges, and sexual behavior in association with four or more of the following five criteria:

- (1) Excessive time is consumed by sexual fantasies and urges, and by planning for and engaging in sexual behavior.
- (2) Repetitively engaging in these sexual fantasies, urges, and behavior in response to dysphoric mood states (e.g., anxiety, depression, boredom, irritability).

(3) Repetitively engaging in sexual fantasies, urges, and behavior in response to stressful life events.

(4) Repetitive but unsuccessful efforts to control or significantly reduce these sexual fantasies, urges, and behavior.

(5) Repetitively engaging in sexual behavior while disregarding the risk for physical or emotional harm to self or others.

B. There is clinically significant personal distress or impairment in social, occupational or other important areas of functioning associated with the frequency and intensity of these sexual fantasies, urges, and behavior.

C. These sexual fantasies, urges, and behavior are not due to direct physiological effects of exogenous substances (e.g., drugs of abuse or medications) or to Manic Episodes.

D. The person is at least 18 years of age.

As you can see, the DSM workgroup recommends more specific criteria for “intense and recurrent.” These more behavioral descriptions are probably much more useful as diagnostic criteria. They must still be considered and field tested. The most important thing that a clinician needs is a clear set of diagnostic criteria or an operational definition of the clinical syndrome. Additional issues about diagnosis are discussed below.

Assessment and Diagnosis

Impulsive/compulsive sexual behavior is unlikely to be a presenting complaint unless the clinician is attached to a specific sexual disorder clinic. Clinicians are generally uncomfortable talking about sex with their patients due to lack of training. It is more likely that the presenting complaint is an STI, an unwanted pregnancy, or marital or relationship problems. Other patients may present with anxiety, depression, alcohol or other drug abuse/dependency, or somatic complaints (Coleman, 1992; Coleman et al., 2003). There is a high comorbidity of ICSB with psychiatric disorders, particularly anxiety disorders, depression, and substance abuse disorders (Black et al., 1997; Kafka & Prentky, 1996; Raymond et al., 2003). Therefore, when patients present with these types of problems, the clinician should suspect that sexual behavior might be associated with them—as a coping mechanism, as a distressing outcome, or simply as a comorbid state. It is important to rule out such things as bipolar disorder or to differentiate between classic obsessive-compulsive disorder and this syndrome.

Men are more likely to suffer from ICSB than women. While there are no good epidemiological data on this disorder, we estimate that 5% of the population may be affected. When women are involved, they are more likely to suffer from non-paraphilic ICSB (Coleman et al., 2003).

When ICSB is suspected, an initial set of screening questions can be helpful. The following screening questions are recommended:

1. Do you, or others who know you, find that you are overly preoccupied or obsessed with sexual activity?

2. Do you find yourself compelled to engage in sexual activity in response to stress, anxiety, or depression?

3. Have serious problems developed as a result of your sexual behavior (e.g., loss of a job or relationship, sexually transmitted infections, injuries or illnesses, or sexual offenses)?

4. Do you feel guilty or shameful about some of your sexual behaviors?

5. Do you fantasize or engage in any unusual or what some would consider “deviant” sexual behavior?

6. Do you find yourself constantly searching or “scanning” the environment for a potential sexual partner?

7. Do you ever find yourself sexually obsessed with someone who is not interested in you or who does not even know you?

8. Do you think your pattern of masturbation is excessive, driven, or dangerous?

9. Do you find yourself compulsively searching for erotica for sexual stimulation?

10. Do you find yourself spending excessive amounts of time on the Internet engaging in various sexual pursuits?

11. Have you had numerous love relationships that are short-lived, intense, and unfulfilling?

12. Do you feel a constant need for sex or expressions of love in your sexual relationship?

Determining the Diagnosis

The screening questions can be helpful in determining if a problem exists. While many people have developed problematic sexual behaviors, this does not mean that they have met the threshold for ICSB.

Standardized Instruments

There are no clinically validated instruments to measure ICSB. Using standardized instruments to evaluate

for comorbid psychiatric conditions is certainly advisable. It is important to look for both Axis I and Axis II disorders, as well as to rule out any contributions from medical conditions. Some scales are under development, such as the Sexual Addiction Screening Test (Carnes, 1991; Nelson & Oehlert, 2008), the Compulsive Sexual Behavior Inventory (Coleman et al., 2001; Miner et al., 2007), the Hypersexual Scale (Reid & Garos, 2007), and the Sexual Compulsive Scale (Kalichman & Rompa, 1995; 2001; the Sexual Inhibition (SIS) and Sexual Excitation (SES) Scales (Janssen et al., 2002a, 2002b). The Compulsive Sexual Behavior Inventory has been translated into Spanish and has been found to be reliable (Coleman & Swinburne Romine, 2010a). In the absence of a fully developed and validated scale, these instruments should be used cautiously. It is helpful to have a number of nonstandardized instruments in asking screening questions, as some patients are sometimes more likely to report things in writing and others in a clinical interview (especially concerning sensitive and taboo subjects). A combined approach leads to a better assessment.

The Danger of Overpathologizing This Disorder

The overpathologizing of sexual behavior (including ICSB) can occur by failing to recognize the wide range of normal human sexual expression—not only in frequency but also in variety (Coleman, 1992, 2010). It can also occur among clinicians who have overly conservative attitudes and values regarding human sexual expression. It is important for professionals to be comfortable with a wide range of normal sexual behavior—both in type and in frequency. Another problem can be caused by lack of knowledge and training. Many clinicians lack appropriate training in human sexuality. As in evaluating any condition outside of one's area of expertise, it is good practice to seek consultation from a specialist in treating sexual disorders.

Sometimes individuals, with their own restrictive values, will diagnose themselves with ICSB, thus creating their own distress. Therefore, it is very important to distinguish between an individual whose values conflict with his or her sexual behavior and one who engages in sexual behaviors that are driven by impulsive, obsessive, and/or compulsive mechanisms.

A Conflict Over Values

There is an inherent danger in diagnosing ICSB simply because the individual's behavior does not fit

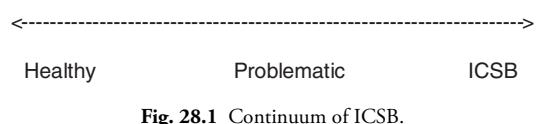
the values of the individual, group, or society. There has been a long tradition of pathologizing behavior that is not normative within a culture. For example, masturbation, oral sex, homosexual behavior, sadomasochistic behavior, or an extrarelational affair could be viewed as compulsive behavior because someone might disapprove of this behavior. There is no scientific merit in viewing these behaviors as disordered, compulsive, or deviant. When individuals are distressed about these behaviors, they are most likely in conflict with their own or someone else's value system rather than with compulsive sexual behavior (Coleman, 1992, 2010b).

Problematic versus Impulsive-Compulsive Sexual Behavior

Behaviors that are in conflict with someone's value system may be problematic but not impulsive, obsessive, or compulsive. Having sexual problems is common. Problems are often caused by a number of nonpathological factors. People may make mistakes; they may be ignorant. They may, at times, act impulsively. Their behavior may cause problems in a relationship. Some people use sex as a coping mechanism similar to the use of alcohol, drugs, or eating. This pattern of sexual behavior may become problematic. Problematic sexual behavior is often remedied, however, by time, experience, education, or brief counseling. Impulsive, obsessive, and compulsive behavior, by its nature, is much more resistant to change (Coleman, 1992, 2010b)

I have found that it is very helpful to view sexual behaviors on a continuum. At one end of the continuum is healthy sexuality; at the other end is the clinical syndrome of ICSB (see Figure 28.1)

It is very important for the clinician to distinguish between problematic sexual behavior and that which may be a function of ICSB. However, this is not easily done, as there are no clear, universally accepted criteria for knowing when a patient has crossed the line between problematic behavior and the clinical syndrome. Only a trained clinician specializing in assessing and treating these individuals would be able to determine the difference. And, in many cases, it is difficult to make that assessment initially. Often in the process of addressing the behavior, it becomes clear that more serious underlying pathological mechanisms are at work (Coleman, 1992, 2010b)



Developmental Process versus Compulsive Sexual Behavior

Some sexual behaviors might be viewed as impulsive, obsessive, or compulsive if they are not considered within their developmental context. Adolescents, for example, can become obsessed with sex for long periods of time. They can act impulsively. In adulthood, it is common for individuals to go through periods in which sexual behavior may take on impulsive, obsessive, and compulsive characteristics. In the early stages of romance, there is a natural developmental period in which individuals might be obsessed with their partners and compelled to seek out their company and to express affection. These are normal, healthy developmental processes of sexual development and must be distinguished from ICSB (Coleman, 1992, 2010b)

Summary

It is important to distinguish between the clinical syndrome of ICSB and behaviors that might be better explained by conflicts in intrapersonal or interpersonal values or as a developmental process. For example, a young single man who thinks masturbation is a sin might consider his behavior obsessive or compulsive. A wife who finds that her husband has viewed some erotica might see this as an addiction.

When ICSB is suspected, the health care professional is advised to refer to a specialist in this area for further assessment and possible treatment. One source of referral is the American Association of Sex Educators, Counselors and Therapists (<http://www.aasect.org>) or the Society for Sex Therapy and Research (<http://www.ssarnet.org/>). There are professional organizations in many countries with standards for training and experience that determine acceptance for membership or certification.

Treatment

Treatment of ICSB usually involves a combination of psycho- and pharmacotherapies (Coleman, 1995; Coleman et al., 2003).

Brief Treatment and Psychoeducation

Many behaviors can be described as problematic or subclinical. Often these behaviors can be readily addressed through brief psychoeducation or solution-focused therapy. Sometimes people simply need to understand the patterns of their behavior, and its negative consequences, and be more motivated to engage in sexually healthier behaviors. In cases where individuals feel that normative sexual

behavior is somehow pathological, the clinician can educate them about the normative range of sexual expression. Violating one's value system is normative, and individuals' mistakes can be used to guide them back to their valued path in life. Errors in judgment can be recognized as such and reaffirm good decision making. Disagreements in value systems between couples are common. These disagreements can be acknowledged and the couple can work to find compromise solutions for accommodating their disparate value systems, as is done with value conflicts in raising children, dealing with family members, or managing finances.

Psychological Treatment

In serious cases, it is more likely that a course of psychotherapy will be needed, often with adjunct pharmacotherapy (Coleman, 1995; Coleman et al., 2003). In ICSB, the patient is suffering from a psychosexual disorder. This is something that is deep-rooted and stems from a failure of healthy psychosexual development. Many of these patients have grown up in dysfunctional family environments and have serious underlying identity and intimacy problems. Many of their needs for nurturance, love, acceptance, and positive role models have not been met. As a result, there has often been a disruption in their psychosexual development that interferes with their ability to feel good about themselves as sexual beings and/or their ability to be intimate with another human being (Coleman, 1987). It is very helpful to conceptualize their problem as an identity disorder, an intimacy disorder, or an attachment disorder.

Therapeutic Modalities

A preferred modality is group therapy combined with adjunct individual and family therapy. Weekly group therapy becomes a laboratory for examining underlying mechanisms, learning from others' coping strategies, and learning new intimacy skills. It is a vehicle to explore the underlying dynamics and see them played out in the interactions with other group members. Group therapy reduces the intense feeling of shame that these individuals experience due to their aberrant sexual histories and their generalized feeling of shame about their basic identity. It also motivates patients to complete the rigorous treatment. In an ongoing group, new members are able to learn from other patients who have completed therapeutic tasks and are better able to understand the whole course of therapy. Group therapy is an opportunity to provide peer support

as patients start to build outside support systems for better coping. They are all able to celebrate when one member has completed the course and is ready to "graduate."

Individual therapy allows the therapist to address more individualized concerns, strengthen the therapeutic alliance, examine issues in more depth, and guide the patient on how to use the other modes of treatment more effectively (e.g., group or family therapy). It allows for ongoing treatment planning and determining therapeutic tasks at different developmental stages of the therapeutic process.

Family therapy is designed to address family-of-origin issues and conflicts in current interpersonal functioning. This could involve a partner or spouse, parents, siblings, or other significant persons in the patient's life.

Gaining Control Over Sexual Behavior

The first step in the therapy process is to work toward gaining some control over the problematic sexual behaviors. By the nature of the problem, it is difficult to gain control over all problematic behaviors at once. It is naturally helpful to gain control over the most problematic behaviors (e.g., engaging in sexual behavior with a high likelihood of developing STIs, including HIV, viewing erotica on work computers where work policies might result in loss of a job, engaging in dangerous sexual activities like autoerotic asphyxiation, or engaging in sexual behaviors that might lead to arrest and incarceration). It is helpful for therapists to understand that developing healthy sexual expression is often a long process. Setting unrealistic goals at the beginning of therapy can set the patient up for failure and discouragement. Therefore, it is helpful to define the boundaries with the patient and to ensure that the boundaries are not overly restrictive. Patients tend to want to restrict their sexual behavior to a narrowly defined script, since they are driven by an intense and immediate desire to be rid of all unhealthy patterns. Some of this desire is driven by a common etiological factors—overly restrictive sexual attitudes about sexual expression.

It is best to have the sexual boundaries set by the patient. Therefore, it is helpful for the clinician to ask the patient to suggest these boundaries. The clinician may have to challenge overly restrictive (or sometimes overly liberal) boundaries set by the patient. In the process of negotiation, the sexual boundaries are clarified in behavioral terms. The patient can then set goals for staying within those boundaries, and can report sexual behaviors that

have been both within the boundaries and outside of them. Violations are then monitored; the goal is to help the patient stay within the boundaries. This serves a number of functions:

1. It protects the patient from experiencing continuing serious harm as a result of the ICSB.
2. The patient develops a sense of competency by meeting his or her own goals.
3. Underlying mechanisms are more clearly identified. By ending the problematic behaviors, the patient is left without one of the main coping mechanisms for underlying etiological dynamics. Anxiety, depression, self-esteem, loneliness, and intimacy problems will become more evident. The risk is that these problems will exacerbate to a level that fuels the ICSB further. For this reason, the boundaries should not be too restrictive at this point and may need to be revised.
4. As staying within these boundaries is difficult, the patient becomes more motivated to address underlying mechanisms rather than simply thinking that he or she can gain control over the behavior by sheer will and determination.
5. Unless he or she stays within the boundaries, the patient simply remains within the cycle of ICSB and masks the underlying mechanisms that must be uncovered and addressed in therapy in order to work on long-term change and the development of healthy sexuality.

Pharmacotherapy as an Adjunct to Psychotherapy

Pharmacological agents can be extremely helpful in initially interrupting the vicious cycle of ICSB. They can be useful in assisting in the initial step of the therapeutic process: gaining control over the most serious ICSBs. Pharmacological therapy alone can be helpful to some patients when the problematic behavior is simply a function of neurotransmitter dysregulation that, when treated, allows the patient to establish healthy sexual behaviors (Coleman, 2005).

Many patients resist the idea of using medication. It is helpful to explain the nature of neurotransmitter dysregulation and its relationship to ICSB, and to familiarize the patient with the literature that has shown this therapy to be effective. One must be cautious and explain that there have been very few randomized clinical trials and that clinicians depend mostly on the case report literature. None of the medications now in use are approved by the Food and Drug Administration for ICSB;

this is “off-label” use. In addition, these medications may not be very helpful to patients with underlying personality disorders that are contributing to their ICSB. Obviously, the patient must consent to treatment.

Besides assisting in the initial stage of gaining control over some ICSBs, pharmacotherapy can assist in the entire process of therapy:

1. Because the mechanisms of ICSB often involve neurotransmitter dysregulation related to impulse control, anxiety, depression, and/or pleasure seeking, pharmacotherapy can speed up the process of therapy or improve its effectiveness.

2. Pharmacotherapy can assist in helping the patient through the rigors of the psychotherapeutic process. In psychotherapy, clinicians often ask patients with ICSB to recall their childhood abusive experiences (whether emotional, physical, or sexual). This reexperiencing of the trauma initially evokes a surprisingly calm or detached response. This is often followed by increased anxiety, which does not seem to be triggered by a particular event. Alternatively, an emotional response to a particular event (e.g., rejection) is felt far more acutely than the situation seemingly warrants. Medications can help modulate the emotional response sufficient for the patient to experience appropriate emotions and assist in resolving these issues (Coleman, 1995, 2005).

3. Learning new mechanisms for managing stress and improving coping skills is an essential part of the psychotherapeutic process. However, the lack of such mechanisms may not completely involve learned behaviors; some mechanisms are underlying maladaptive stress responses.

Medications can help mediate these maladaptive stress responses while the patient is learning new behavioral strategies for managing stress and improving coping skills.

Resolving Family-of-Origin Conflicts

As ICSB is often a function of an underlying disturbance in identity, intimacy, and attachment functioning, it is helpful for the patient to understand the source of the identity and intimacy problems and work to resolve them. As a beginning step in the therapeutic process, patients are asked to begin writing their sexual autobiography (including their early experiences not only with sexual activity but also with intimate relationships). Through this process, patients become more aware of the disruptions in their psychosexual development. These conflicts

are often played out in their dysfunctional relationships and sexual behavior; however, patients often do not recognize these patterns. For some patients, resolution of conflicts may be as simple as acknowledging and grieving over these losses. For others, it is helpful to review these conflicts with their family members to gain further understanding and a shared feeling of loss. The best resolution of family-of-origin conflicts is obtained when family relationships are improved in terms of intimacy functioning (Coleman, 1995). This is accomplished through family therapy. The next step is for patients to clearly see how these dynamics are played out in their cycle of ICSB, and the nature and dynamics of their intimate relationships (or lack thereof). Different patterns are learned, experimented with, and consolidated in the therapeutic process.

Understanding Triggers and Cycles of ICSB

After reviewing the patient’s sexual autobiography, learned patterns of sexual behavior and, in particular, ICSB are identified. The patients gain an understanding of the events, situations, and/or emotions that often trigger their ICSB. They are encouraged to identify their *cycle* of behavior, which describes the chain or sequence of events, the dynamics that lead to their ICSB, and those that perpetuate it. Each patient has a unique cycle. Once they understand their cycle, patients are better prepared to employ tactics to interrupt it and learn healthy intimacy and sexual functioning (Coleman, 1995, 2010b). This prepares them to develop their maintenance plan. Relapse prevention strategies are employed.

Learning New Coping Mechanisms and Breaking the Cycle

Once the cycle is understood, the patient is able to learn the most appropriate mechanisms for breaking it rather than letting the cycle perpetuate itself. This can involve strategies such as stress management, anger management, communication skills, relaxation, self-nurturing, developing and utilizing a support system, and/or changing cognitions. Therapy offers patients psychological tools to better manage their stress, anxiety, and depression, which can trigger their ICSB (Coleman, 1995, 2010b).

Learning New Intimacy Skills and Healthy Sexual Functioning

Once ICSB is under control, the patient is ready to learn new intimacy skills and develop healthy sexual functioning. This may involve major shifts in the ways in which patients interact with other people,

are intimate with their partner, or approach sexual activity with themselves or with a partner. These are learned skills. Patients need guidance from the therapist on ways of improving their relationship skills and approaching sexual activity. The basic principles of sex therapy can be very useful in this stage of the therapeutic process.

Intimate relationships are often destroyed or severely damaged as a result of a patient's ICSB. Repairing a relationship is quite a task. Partners often have major issues of trust and betrayal. They need time to grieve, to be angry, and to be able to forgive before reengaging in an intimate relationship. Both the patient and the partner need to develop new intimacy skills through honest and respectful communication, empathy, and commitment to work on the relationship. Partners often have their own issues that need to be worked through independently. Repairing a broken relationship comes at a later stage of the therapeutic process when the ICSB is under control, new patterns of relating can be learned, and trust restored (Coleman, 1995, 2010b).

Aftercare

We have found that many of our patients benefit from ongoing psychotherapeutic support through a much less intensive but ongoing therapy. The goal of this aftercare is to consolidate the gains obtained in therapy, to immediately address issues of potential relapse, and to foster the idea of ongoing sexual development. Patients who have completed an intensive course of treatment and have achieved the aforementioned goals enter an aftercare group composed of other patients who have also achieved the goals of therapy. This group offers ongoing support and helpful advice, since they understand the nature of the problem and have been successful in overcoming it. For patients who are unable to attend an aftercare group on an ongoing basis, the exact form of aftercare can be tailored to the individual situation. Offering individual psychotherapy periodically can be sufficient for many patients. All patients are encouraged to develop their own support system outside of therapy, and for some patients, this is sufficient aftercare support. The bottom line is that most patients need some type of ongoing support to maintain their progress and to grow further as sexual beings.

Summary

Psychological treatment for ICSB involves a complex set of therapeutic modalities and treatment

regimens. Group therapy is a preferred main modality of treatment combined with adjunct individual and family psychotherapy. Because ICSB is a deep-seated psychosexual disorder, a long process is needed not only to gain control over it but also to consolidate long-term gains and prevent relapse. The ultimate goal is not just to gain control over dysfunctional sexual behaviors but also to assist individuals to find healthy and pleasurable means of sexual expression and intimacy functioning.

Pharmacological Treatment

A number of pharmacological agents have been shown to provide helpful adjunctive therapy for ICSB. The most frequently utilized agents are the selective serotonin reuptake inhibitors (SSRIs; Bradford, 2000; Coleman, 2005, 2010b). These medications have been shown to be quite effective in treating depression and anxiety and in helping to control ICSB. They can be used to address underlying mechanisms of anxiety and depression and also to promote impulse control. They also have the side effect of decreasing the libido. The value of the SSRIs is that they seem to address the three main pathophysiological mechanisms outlined by Kafka (2009). A number of open-label studies have indicated that the SSRIs may be efficacious in the treatment of ICSB (Bradford, 2000; Coleman et al., 1992; Federoff, 1993; Kafka, 1991, 1994; Kafka & Prentky, 1992; Stein et al., 1992). These medications appear to improve mood, reduce anxiety, interrupt obsessive thinking, and help patients control urges to engage in ICSB. The older tricyclic medications and the newer atypical antidepressants can still be used; for certain patients, these may be more effective than the SSRIs (e.g., Coleman et al., 2000).

In more resistant cases, antiandrogens can be used (Bradford, 2000; Raymond et al., 2002b). These medications often do not address comorbid psychiatric disorders (especially anxiety and depressive disorders (Raymond et al., 1999), but they control libido and help moderate sexual urges. These drugs have much more severe side effects to be considered, but they remain potentially useful pharmaceutical agents (Bradford, 2000).

Mood stabilizers can also be used. These medications have been used in the past and remain a potential effective treatment modality (Cesnik & Coleman, 1989; Coleman et al., 2002). In patients for whom the SSRIs do not provide sufficient control for their ICSB, clinicians have augmented SSRI therapy with different types of mood stabilizers such

as lithium carbonate, carbamazepine, and valproic acid (Coleman, 2005, 2010b).

Finally, we have been encouraged by the results produced by naltrexone used with patients with ICSB. Naltrexone is an opioid antagonist that acts on opioid receptors. It effectively blocks these receptors, preventing the body from making use of opiates and endorphins. Opioid antagonists were first used to treat opium addicts; however, more recently, they have been shown to be effective in treating alcoholics, compulsive gamblers, kleptomaniacs, and smokers. Naltrexone not only seems to block the sensation and pleasure-seeking mechanisms but is also thought to have an effect on the impulse control centers of the brain as well. We reported on the first successful treatment of two individuals with ICSB with naltrexone. Both of them had severe symptoms of ICSB and had shown only mild improvement with SSRIs and other antidepressants. When naltrexone was added to the SSRI treatment, the patients lost the urge to engage in their prior problematic behavior and were able to stop their ICSB (Raymond et al., 2002a). We recently completed a case review with similar positive results (Raymond et al., 2010) and now are embarking on a double-blind, placebo-controlled study. Naltrexone can be used to augment SSRI therapy, and sometimes it can be useful on its own. These preliminary results are encouraging and, in our clinical experience, have added one more tool to our armamentarium of pharmacological treatment choices.

Summary of Pharmacological Treatment

Pharmacological treatment has been shown to be an effective adjunct to the psychological treatment of ICSB. Sometimes a single SSRI can be helpful; alternatively, other medications or the use of medications to augment the SSRI are efficacious. It takes a trained clinician who is familiar with these medications and the literature to effectively utilize these medications in treating patients with ICSB. We need controlled clinical trials in order to develop a more evidence-based clinical approach to the pharmacological treatment of ICSB. However, there is at least some evidence that a number of helpful treatment options are available (Bradford, 2000; Coleman, 2005, 2010b). Clinicians should keep abreast of the literature for future developments, as this is still an emerging field of study.

Conclusion

Impulsive/compulsive sexual behavior is a serious clinical disorder that deserves attention from health

care professionals. It can be easily overlooked and yet can lead to serious distress and negative consequences. The difficulty of identifying this problem is compounded by the fact that we do not have a consensus on what to call this syndrome, the diagnostic criteria, or the methods to treat it. We rely on a case report literature for guidance in the absence of clinical trials of psychological or pharmacological treatment. Careful assessment and combined multimodal and multidisciplinary treatment can offer assistance to many individuals suffering from ICSB. Specialists are needed in assessment and treatment.

There is much to be learned about this syndrome. Clinicians will need to keep up with the emerging literature in order to provide the best evidence-based care. In the meantime, it is encouraging that we have found effective treatments that can offer hope for improved sexual and intimate lives for patients who suffer from ICSB.

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Assessment and Treatment of Problem Internet Use

Kimberly Young

Abstract

Research over the last decade has identified Internet addiction as a new and often unrecognized clinical disorder that impacts a user's ability to control online use to the extent that it can cause relational, occupational, and social problems. Symptoms of problem Internet use are compared to the criteria used to diagnose other addictions. In particular, pathological gambling is compared to problematic Internet use because of overlapping criteria. As new cases of problem Internet use are being documented, this chapter describes the diagnostic and treatment implications of the disorder. As computers are used with great frequency, detection and diagnosis of Internet addiction are often difficult. Symptoms of a possible problem may be masked by legitimate use of the Internet, and clinicians may not ask questions about computer use because problem Internet use is still a new and often unrecognized condition. This chapter outlines diagnostic conceptualizations of problem Internet use including the DSM-V proposed criteria for Pathological Computer Use, assessment techniques, and treatment considerations.

Keywords: Internet addiction, problem Internet use, pathological computer use, assessment, diagnosis, treatment

Introduction

The Internet is a new technology that has impacted the world and provided many benefits to its users. At the same time, the Internet has had negative ramifications. Some people are becoming preoccupied with the Internet, are unable to control their use, and are jeopardizing their employment and relationships. The concept of *Internet addiction* has been proposed as an explanation for uncontrollable, damaging use of this technology. Symptoms are compared to the criteria used to diagnose other addictions, and the literature has characterized Internet addiction as an impulse control disorder comparable to pathological gambling because of overlapping criteria.

Much of this research lacks the empirical robustness of experimental design, relying more upon survey data and self-reported data from self-selected

populations. The research also lacks proper use of control groups and, in some cases, utilizes a small number of anecdotal case studies and questionnaires to draw conclusions. The research has further been complicated as studies have utilized various terms to describe Internet addiction, including *Internet dependency*, *problematic Internet use*, *excessive use*, *heavy use*, *pathological Internet use*, and *compulsive Internet use*.

Studies on Internet addiction originated in the United States. More recently, studies have documented Internet addiction in a growing number of countries such as Italy (Ferraro et al., 2007), Pakistan (Suhail & Bargees, 2006), and the Czech Republic (Simkova & Cincera, 2004). Reports also indicate that Internet addiction has become a serious public health concern in the People's Republic of China (BBC, 2005), Korea (Hur, 2006), and Taiwan (Lee, 2007). According to

Lee (2007), about 10% of China's more than 30 million Internet gamers are said to be addicted. To battle what has been called an epidemic by some reports, Chinese authorities regularly shut down Internet cafes, many illegally operated, in crackdowns that also include huge fines for their operators. The Chinese government has also instituted laws to regulate the number of hours adolescents can play online games and has opened the first inpatient treatment center for Internet addiction in Beijing.

It is difficult to estimate how widespread the problem is. A nationwide study conducted by a team from Stanford University's School of Medicine had estimated that nearly one in eight Americans exhibit at least one possible sign of problematic Internet use (Aboujaoude et al., 2006).

Therefore, this chapter reviews conceptualizations of Internet addiction, including the new proposed criteria for the diagnosis of Pathological Computer Use to be used in the revision of the DSM-V. In addition, this chapter reviews various forms that Internet addiction can take, including online sexual preoccupations, Internet gambling, and interactive applications from chatting to games.

This chapter also examines the treatment implications for working with Internet-addicted clients. Unlike other addictions, healthy overuse of the Internet can be very productive: people can surf for information, make vacation plans, book airline seats, do research, chat with friends, and bank online. Not all Internet use is unproductive or nonlegitimate. Treatment of Internet addiction involves the attempt to moderate and control compulsive use while retaining a healthy balance between computer use and other aspects of a client's life. In addition to discussing the larger issue of conceptualizing and diagnosing the problem, once it is brought into the treatment arena, the issue becomes how to help clients find that balance when they rely on computers in their daily lives.

Diagnostic Conceptualization

According to Dr. Maressa Hecht Orzack, director of the Computer Addiction Services at McLean Hospital, a Harvard Medical School affiliate and another pioneer in the study of Internet addiction, Internet addicts demonstrate loss of impulse control in which life becomes unmanageable for them but, despite the resulting problems, they cannot moderate their Internet use. The computer becomes the primary relationship in the addict's life (Orzack, 1999).

While time is not a direct factor in diagnosing Internet addiction, early studies suggested that persons classified as dependent or addicted Internet

users generally had an excessive habit, spending anywhere from 40 to 80 hours per week online, with sessions lasting up to 20 hours (Greenfield 1999; Young, 1998). Sleep patterns were disrupted due to late night log-ins and addicts generally stayed up surfing until late in the morning, with the reality of having to wake up early for work or school. In extreme cases, caffeine pills were used to facilitate longer Internet sessions. Such sleep deprivation caused excessive fatigue impairing academic or occupational performance, which also increased the risks of poor diet and insufficient exercise.

Given the popularity of the Internet, detecting and diagnosing Internet addiction is often difficult, as its legitimate business and personal use often mask addictive behavior. The best method for clinical detection of compulsive Internet use is to compare it against criteria for other established addictions. Researchers have likened Internet addiction to addictive syndromes similar to impulse control disorders on the Axis I Scale in the DSM and have utilized various forms of DSM-IV-based criteria to define Internet addiction. Of all the conditions referenced in the DSM, pathological gambling was viewed as most akin to this phenomenon. The Internet Addiction Diagnostic Questionnaire (IADQ) was developed as an initial screening instrument for diagnosis (Young, 1998). The following questions from this questionnaire conceptualize patterns associated with the disorder:

1. Do you feel preoccupied with the Internet (think about previous online activity or anticipate the next online session)?
2. Do you feel the need to use the Internet for increasing amounts of time in order to achieve satisfaction?
3. Have you repeatedly made unsuccessful efforts to control, cut back, or stop Internet use?
4. Do you feel restless, moody, depressed, or irritable when attempting to cut down or stop Internet use?
5. Do you stay online longer than originally intended?
6. Have you jeopardized or risked the loss of a significant relationship, job, educational or career opportunity because of the Internet?
7. Have you lied to family members, therapists, or others to conceal the extent of involvement with the Internet?
8. Do you use the Internet as a way of escaping from problems or of relieving a dysphoric mood (e.g., feelings of helplessness, guilt, anxiety, depression)?

Answers to these questions were used to evaluate nonessential computer/Internet use, such as non-business or nonacademically related use. Subjects were considered dependent if they endorsed when answering five or more of the questions over a 6-month period. Associated features included ordinarily excessive Internet use, neglect of routine duties or life responsibilities, social isolation, and being secretive about online activities or suddenly demanding privacy when online. While the IADQ provides a means to conceptualize pathological or addictive use of the Internet, these warning signs can often be masked by cultural norms that encourage and reinforce online use. Even if a client meets all the criteria, signs of abuse can be rationalized by statements such as "I need this for my job" or "It's just a machine" when in reality the Internet is causing significant problems in a user's life.

Beard and Wolf (2001) further modified Young's diagnostic criteria, recommending that all of the first five criteria be required for diagnosis of Internet addiction, since these criteria could be met without any impairment in the person's daily functioning. They also recommended that at least one of the last three criteria (i.e., criteria 6, 7, or 8) be required in diagnosing Internet addiction. The last three criteria were separated from the others because they impact the pathological Internet user's ability to cope and function (e.g., depressed, anxious, escaping problems) and also affect the user's interaction with others (e.g., significant other, colleagues at work). Shapiro et al. (2003) put forth a more comprehensive approach to diagnosing Internet addiction under the general heading of impulse control disorders per the DSM-IV-TR that further broadened the diagnostic criteria for problematic Internet use:

A. Maladaptive preoccupation with Internet use, as indicated by at least one of the following:
1. Preoccupations with use of the Internet that are experienced as irresistible. 2. Excessive use of the Internet for periods of time longer than planned.

B. The use of the Internet or the preoccupation with its use causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

C. The excessive Internet use does not occur exclusively during periods of hypomania or mania and is not better accounted for by other Axis I disorders.

Most recently, the American Psychiatric Association has considered including the diagnosis of Pathological Computer Use in the upcoming

revision of the DSM-V (Block, 2007; Block, 2008). Conceptually, the diagnosis is a compulsive-impulsive spectrum disorder that involves online and/or offline computer use (Dell'Osso et al., 2006; Hollander & Stein, 2006) and consists of at least three subtypes: excessive gaming, sexual preoccupations, and e-mail/text messaging (Block, 2007). All of the variants share the following four characteristics: (1) *excessive use*, often associated with loss of the sense of time or neglect of basic drives; (2) *withdrawal*, including feelings of anger, tension, and/or depression when the computer is inaccessible; (3) *tolerance*, including the need for better computer equipment, more software, or more hours of use; and (4) *negative repercussions*, including arguments, lying, poor achievement, social isolation, and fatigue (Beard & Wolf, 2001; Block, 2008). This last criterion seems to pull together all the previous forms of classification of Internet addiction, defining it in a comprehensive manner that includes the major components associated with the compulsive behavior.

Motivation for Treatment

Once Internet addiction is diagnosed, the next step is to assess the client's motivation for treatment. In the early stages of recovery, clients will typically deny or minimize their habitual use of the Internet and the consequences of their behavior for their lives. Often, a friend, a spouse, or a parent has pushed the individual to seek help, especially in cases of addiction. The client may feel resentful and deny that use of the Internet is a problem. To break this pattern, after diagnosis, the therapist should use motivational interviewing techniques that encourage the client to commit to treatment as an integral aspect of recovery (Greenfield 1999; Orzack, 1999).

The concepts and approaches of motivational interviewing evolved from experience in the treatment of problem drinkers and were first described by Miller (1983). They were later elaborated by Miller and Rollnick (1991), who provided a more detailed description of the clinical procedures. Motivational interviewing is a goal-directed style of counseling for eliciting behavior change by helping clients to explore and resolve ambivalence. It involves asking open-ended questions, giving affirmations, and using reflective listening.

Motivational interviewing is intended to confront the client in a constructive manner to evoke change or, using external contingencies such as the potential loss of a job or relationship, to mobilize a client's values and goals to stimulate behavior change. Clients dealing with addiction or substance

abuse problems often feel ambivalent about quitting, even after they admit that they have a problem. They fear the loss of the Internet; they fear what life might be like if they were unable to chat with online friends, engage in online activities, and use the Internet as a form of psychological escape. Motivational interviewing helps clients confront their ambivalence.

Questions can be asked such as:

- When did you first begin to use the Internet?
- How many hours per week do you currently spend online (for nonessential use)?
- What applications do you use on the Internet (specific sites/groups/games)?
- How many hours per week do you spend using each application?
- How would you rank order each application from most to least important (1 = first, 2 = second, 3 = third, etc.)?
- What do you like best about each application? What do you like least?
 - How has the Internet changed your life?
 - How do you feel when you log off?
 - What problems or consequences have stemmed from your Internet use? (If this is difficult for the client to describe, have the client keep a log near the computer in order to document such behaviors for the next week's session.)
 - Have others complained about how much time you spend online?
 - Have you sought treatment for this condition before? If so, when? How much success did you have?

The answers to these questions create a clearer clinical profile of the client. The therapist can determine the types of applications that are most problematic for the client (e.g., chat rooms, online gaming, online pornography). The length of Internet use, the consequences of the behavior, a history of prior treatment attempts, and the outcomes of any such attempts are also assessed. This helps clients begin the process of examining how the Internet impacts their lives.

It is helpful for clients to gain a sense of responsibility for their behavior. Allowing clients to resolve their ambivalence in a manner that gently pushes them makes clients more inclined to acknowledge the consequences of their excessive online use and engage in treatment. Generally, the therapeutic style is quiet and eliciting rather than aggressive, confrontational, or argumentative. For therapists accustomed to confronting and giving advice, motivational

interviewing may appear to be a hopelessly slow and passive process. However, the proof is in the outcome. More aggressive strategies, sometimes guided by a desire to "confront client denial," easily push clients into making changes for which they are not ready.

In cases of impulse control disorders, an individual's compulsive behavior is often associated with increasingly painful states of tension and agitation that are relieved by the completion of the act. For example, an alcoholic is driven to drink or an overeater is driven to binge on food during moments of tension and stress. The compulsive behavior serves to reduce the underlying emotional tension and serves as a reward for future behavior. Similarly, interactive online features such as chat rooms, instant messaging, or texting can be less about using the Internet as a communication tool and more about finding a psychological escape (Young, 2004).

Researchers have suggested that, like the person who craves a cigarette or a drink, Internet addicts feel a difference between online and offline emotions (Young, 1998). They may feel frustrated, worried, angry, anxious, and depressed when offline. When online, they feel excited, thrilled, uninhibited, attractive, supported, and more desirable. These strong positive emotions reinforce the compulsive behavior (Orzack, 1999). The behavior acts as a way to temporarily avoid negative or unpleasant feelings. Under the influence, the alcoholic feels as if all the other problems in life have disappeared. When eating, the overeater experiences a sense of peace and relaxation, lessening the overwhelming stress and frustration he or she feels. In the same manner, compulsive Internet users use the virtual world to escape life's problems temporarily, involving themselves deeply in virtual relationships carried on by chat, text, or e-mail. Over time, this coping mechanism can prove unproductive and potentially harmful as the issues hidden by the compulsive behavior culminate in larger problems.

Helping the client explore how he or she feels just before going online will help pinpoint the types of emotions being covered by the behavior (or how the client is using the Internet to cope or escape from problems). Answers may include issues such as a fight with a spouse, depressed mood, stress at a job, or a poor grade in school. Motivational interviewing should explore how these feelings diminish when online, looking for how the client rationalizes or justifies using the Internet (e.g., "Chatting makes me forget about the fight with my husband," "Looking at online porn makes me feel

less depressed,” “Gambling online makes me feel less stressed at work,” “Killing other players in an online game makes me feel better about my poor grade at school”). Motivational interviewing is also meant to help the client recognize the consequences stemming from excessive or compulsive use. Problems may consist of issues like these: “My spouse becomes angrier,” “My depressed feelings return when I turn off the computer,” “My job still stinks,” and “I will lose my scholarship if I don’t get my grades up.” The therapeutic relationship is more like a partnership or companionship than one of expert/recipient roles to examine and resolve ambivalence. The operational assumption in motivational interviewing is that ambivalence is the principal obstacle to be overcome in triggering change. Overall, the specific strategies are designed to elicit, clarify, and resolve ambivalence in a client-centered and respectful therapeutic manner.

Classic and New Internet Abusers

Based upon clinical interviews with over 2000 Internet addicts in the past decade, there appear to be two types of addicts or abusers. Some clients suffer from a prior addiction, such as to sex or gambling, and they use the Internet as a new outlet to act on this addiction. These clients are referred to as *Classic Addicts*. Other clients have no history of prior addiction; their addiction to the Internet is an entirely new problem behavior. These clients are referred to as *New Addicts*. Classic Addicts are generally relapsing on the Internet into those activities that they can perform in real life, such as pathological gambling or sexually compulsive behavior.

For examples, for those Classic Addicts in recovery from sex addiction, the Internet becomes another way to engage in sexually compulsive behavior. Recovering addicts who feel overwhelmed, or who experience personal problems or life-changing events such as divorce, relocation, or the death of a loved one, can become absorbed in a virtual world full of fantasy and intrigue; the Internet serves as a new way to engage in sexual behavior (Young, 2008). Sexual compulsives discover a new source for sexual gratification through online pornography and anonymous sex chat. The Internet allows them to continue their sexual behavior without the physical need to visit strip clubs or prostitutes and provides a new and socially acceptable way to cope.

Classic Addicts may also suffer from a history of alcohol or drug dependency, only to find their compulsive use of the Internet a physically safe alternative to their addictive tendency. They believe that

being addicted to the Internet is medically safer than being addicted to drugs or alcohol; at the same time, the compulsive behavior avoids the need to confront the unpleasant situation underlying the addiction.

Clients who suffer from multiple addictions (to the Internet as well as to alcohol, cigarettes, drugs, food, sex, etc.) are at the greatest risk of relapsing. This is especially true when it comes to the Internet. Often, addicts need to use the computer for work or school, so the temptation to return to the problematic behavior feels constant because the computer is always available. Multiple addictions also suggest that the client has an addictive personality and compulsive tendencies, making relapse more likely.

New Addicts meet two distinct criteria. First, they become addicted to new forms of Internet use created solely online, such as chat rooms, social networking, instant messaging, online role-playing games, or eBay shopping. For instance, someone who becomes addicted to chat rooms must use the Internet to chat. Someone who becomes addicted to eBay must use the Internet to access it. Granted, these activities have now become portable through mobile devices such as personal digital assistants (PDAs) or cell phones. The key element is that they are all considered Internet-specific activities.

Secondly, New Addicts are individuals with no previous significant addictive or psychiatric history. They develop an addiction to the anonymous, accessible, and interactive nature of online use. New Addicts may include a 50-year-old lawyer using sex chat rooms during work hours and without his wife’s knowledge, a 30-year-old business executive compulsively checking his Blackberry, a 20-year-old college student constantly on Facebook, or a 16-year-old boy constantly playing online role-playing games. For New Addicts, compulsive use of the Internet is a new clinical phenomenon.

New Addicts are attracted to the Internet, which is often first seen as a problem by others, usually a spouse, parent, or close friend. Their behavior progressively revolves around Internet use. It involves more and more of a focus on technology that may have initially been required for work use, such as a Blackberry, or may have been an activity pursued for recreation, such as a chat room meeting or game. As the behavior escalates, online use becomes more chronic and ingrained and develops into a compulsive obsession. In this stage, life becomes unmanageable for the addict, as relationships or careers are jeopardized because of the compulsive behavior.

In one such case, I worked with a 34-year-old minister arrested for possession of child pornography obtained from the Internet. He explained his fascination: "I soon discovered the vast array of pornography, including child pornography, available on the Internet. My attraction to online porn was born of sheer amazement at the volume of available material, and this amazement turned to fascination and ultimately to obsession. I knew it was wrong to look at this material. My life became a lonely, isolated mess. I realized that I could lose my job, my marriage, and the respect of everyone I love if I was caught. I have two daughters and would never think about doing anything inappropriate with them, but I could not bring myself to stop despite knowing all the consequences of my actions. What started as curiosity has put my faith and everything important to me in jeopardy."

The New Addict becomes preoccupied with the computer and constantly feels a longing to be online. The person attempts to conceal the nature of his or her online activities and fears that others will discover this secret life. Realizing the impact of this destructive behavior, the person rationalizes it and continues to engage in the activity despite its known potential risks, including possible job loss, divorce, or arrest. The online experience turns into a relief from pain and anxiety, the reward for success, and a way to avoid addressing other emotional issues in the person's life. The addiction is truly an altered state of consciousness in which normal behavior pales by comparison in terms of excitement and relief from troubles that is associated with use of the Internet. In this way, the online world becomes a private refuge. The addict displays a progressive retreat into the computer world as a means of avoiding life's complications and responsibilities.

Underlying Social Problems

Excessive or problematic Internet use often stems from interpersonal difficulties such as introversion or social problems (Ferris, 2001). Many Internet addicts fail to communicate well in face-to-face situations (Leung, 2007). This is part of the reason they use the Internet in the first place. Communicating online seems safer and easier for them. Poor communication skills can also cause low self-esteem and a feeling of isolation and create additional problems in life, such as trouble working in groups, making presentations, or going to social engagements. Assessment and treatment needs to address how Internet addicts communicate offline. Encouraging affect, communication analysis, modeling, and role

playing are helpful interventions to apply (Hall & Parsons, 2001).

Researchers have suggested that individuals who suffer from low self-esteem, and who feel lonely, restless, or withdrawn, can use online connections to feel better about themselves and their circumstances (Morahan-Martin & Schumacher, 2003). Socially awkward or emotionally troubled individuals may find it easier to engage in Internet relationships than risk the face-to-face rejection of a real person (Ferris, 2001). The anonymity associated with electronic communication can also increase the online user's feeling of comfort since there is no need to look for, and thus detect, signs of insincerity, disapproval, or judgment in facial expressions, as would be typical in face-to-face interactions (Morahan-Martin & Schumacher, 2003).

Among married couples, this can lead to online affairs (Whitty, 2005). An online affair is a romantic or sexual relationship initiated via online contact and maintained predominantly through electronic conversations and online communication (Atwood & Schwartz, 2002). Electronic communication allows individuals to feel less inhibited, which accelerates perceived intimacy. Online, people are more likely to be open, honest, and forthright, revealing personal truths and the intimacy that might take months or years to establish in an offline relationship may only takes days or weeks online (Cooper & Sportolari, 1997). Online, people can seem more glamorous than they are in day-to-day life (Whitty, 2005). Married Internet users can utilize e-mail and texting to seek out support, comfort, and acceptance from a romantic online partner that gives them a sense of belonging in a nonthreatening manner. Unfortunately, online affairs do more than create a troubling new clinical problem for couples dealing with Internet infidelity (Whitty, 2005). According to the American Academy of Matrimonial Lawyers, 63% of attorneys found that online affairs accounted for a growing number of divorce cases (Dedmon, 2003), creating new legal problems.

As demand has increased for mobile devices, PDAs, and cell phones with Internet access, problem mobile phone use has grown, especially among younger persons (Bianchi & Phillips, 2005). As a result, e-mailing and texting overuse have become growing social and clinical issues. Time spent e-mailing and texting can impair important and purposeful relationships. The more time is spent checking e-mail or chatting online, the less time is spent with one's family and friends. Ultimately, these

new ways of communicating have created a new type of *technostress*.

Due to their Internet addiction, individuals also often damage or lose significant real-life relationships, such as those with a spouse, a parent, or a close friend (Young, 2004). Often, these were individuals who provided the addict with support, love, and acceptance before the Internet, and their absence makes the addict feel worthless and reinforces past notions that they are unlovable. The addict must mend and reestablish these broken relationships to achieve recovery and find the support necessary to fight the addiction. Rebuilding relationships and providing new ways to relate to others allows for amends to be made. Involving loved ones in recovery can be a rich source of nurturing and sponsorship to help a client maintain sobriety and abstinence. Couples or family therapy may be necessary to help educate loved ones about the addiction process and engage them more fully in helping the client maintain boundaries established with the computer.

When evaluating social problems, it is important to investigate how the client has been using the Internet. If it has involved interactive environments such as chat rooms, instant messaging, or social networking sites, then the therapist should evaluate aspects of online use by asking questions such as the following: Does the person make up a persona? What kind of screen name does the person use? Does Internet use disrupt current social relationships? If so, how? These are important issues to evaluate in order to understand the social dynamics underlying online usage and how relationships formed on the Internet may be substituting or replacing relationships in real life. Possible issues to consider are:

- Have you been honest about your Internet habit with your friends and family?
- Have you ever created an online identity or persona?
- List online activities that you kept secret or thought others would not approve of.
- Have online friends disrupted real-life relationships?
 - If so, who was impacted (husband, wife, parent, friend) and how?
 - Does Internet use disrupt your social or work relationships?
 - If yes, please describe how this happens.
 - Please describe other ways in which Internet use has impacted your life.

Questions and requests like these help structure the clinical interview to provide more detailed information on how the Internet has impacted relationships in the client's life. Often clients create online personas, and the answers to these questions provide specific information on the characteristics and nature of these personas. They help therapists understand the client's psychological motives, the ways online personas develop, and how they may be used to fulfill missing or unmet social needs. Once this type of critical examination takes place, the therapist can work with the client to develop new social relationships or reestablish former social connections that will sustain his or her motivation for continued treatment.

Treatment Considerations

Cognitive behavioral therapy (CBT) has been shown to be an effective treatment for compulsive disorders such as intermittent explosive disorder, pathological gambling, and trichotillomania (Hucker, 2004). It has also been effective in treating substance abuse, emotional disorders, and eating disorders as well (e.g., Beck, 1979; Beck et al., 1993).

Cognitive behavioral therapy is a familiar treatment based on the premise that thoughts determine feelings. Patients are taught to monitor their thoughts and identify those that trigger addictive feelings and actions while they learn new coping skills and ways to prevent a relapse. Cognitive behavioral therapy usually requires 3 months of treatment or approximately 12 weekly sessions. The early stage of therapy is behavioral, focusing on specific behaviors and situations in which the impulse control disorder causes the greatest difficulty. As therapy progresses, there is more of a focus on the cognitive assumptions and distortions that have developed and the effects of the compulsive behavior.

In cases of Internet addiction, researchers have suggested that moderated and controlled use of the Internet is the most appropriate treatment (Greenfield, 1999; Orzack, 1999). Behavior therapy is the initial focus of recovery, examining both computer and noncomputer behavior (Hall & Parsons, 2001). Computer behavior deals with actual online usage, with a primary goal of abstinence from problematic applications while retaining controlled use of the computer for legitimate purposes. For example, a lawyer addicted to Internet pornography would need to learn to abstain from visiting adult web sites while still accessing the Internet to conduct legal research and to e-mail clients. Noncomputer behavior focuses on helping clients develop positive lifestyle

changes for life without the Internet. Life activities that do not involve the computer such as offline hobbies, social gatherings, and family activities are encouraged. Similarly to food addiction, in which recovery can be objectively measured through reduced caloric intake and weight loss, online addicts can objectively measure success by maintaining abstinence from problematic online applications and increasing meaningful offline activities.

Young (2007) suggests using a Daily Internet Log to evaluate computer behavior and establish a baseline for clinical treatment. Once a baseline has been established, behavior therapy is used to relearn how to use the Internet to achieve specific outcomes, such as moderated online usage and, more specifically, abstinence from problematic online applications and controlled use for legitimate purposes. Behavior management for both computer usage and adaptive noncomputer behavior focuses on current online behavior. Early outcome studies show that assertion training, behavioral rehearsal, coaching, modeling, and relaxation training are effective methods.

From a cognitive perspective, addictive thinkers, for no logical reason, feel apprehensive when anticipating disaster (Twerski, 1990). While addicts are not the only people who worry and anticipate negative events, they tend to do this more often than other people. Young (1998) first suggested that this catastrophic thinking might contribute to compulsive Internet use by providing a psychological escape mechanism to avoid real or perceived problems. Subsequent studies hypothesized that other maladaptive cognitions such as overgeneralizing or catastrophizing, negative core beliefs, and cognitive distortions also contribute to compulsive use of the Internet (Caplan, 2002; Davis, 2001; LaRose et al., 2001). Those who suffer from negative core beliefs may be the ones drawn most strongly to the anonymous interactive capabilities of the Internet in order to overcome their perceived inadequacies. Early treatment outcome studies show that CBT can be used to address these negative core beliefs, cognitive distortions, and rationalizations such as "Just a few more minutes won't hurt" that sustain problematic or compulsive use of the Internet (Young, 2007).

Future Practice

Over the last decade, the acceptance of Internet addiction has grown in the mental health field, with a new emphasis on assessment and treatment of the disorder. It is difficult to predict the results of these early endeavors. However, it is feasible that with

years of collective effort, Internet addiction may be recognized as a legitimate impulse control disorder worthy of its own classification in future revisions of the *Diagnostic and Statistical Manual of Mental Disorders*.

With the growing popularity of the Internet, increased awareness in the mental health field will help clinicians provide knowledgeable care and intervention for the Internet-addicted client. Since this is a new and often seemingly comical addiction, individuals may be reluctant to seek treatment, fearing that clinicians may not take their complaints seriously. Drug and alcohol rehabilitation centers, community mental health clinics, and clinicians in private practice should be aware of the negative ramifications of compulsive Internet use and recognize the signs, which may easily be masked by other comorbid conditions or legitimate use of the Internet.

To pursue effective recovery programs, continued research is needed to better understand the underlying motivations of Internet addiction. Future research should focus on how psychiatric illnesses such as depression or obsessive-compulsive disorder plays a role in the development of compulsive Internet use. Longitudinal studies may reveal how personality traits, family dynamics, or interpersonal skills influence the way people utilize the Internet. Further outcome studies are needed to determine the efficacy of specialized therapeutic approaches to treat Internet addiction and compare their outcomes with those of traditional treatment modalities.

As psychiatric comorbidity is a factor in the development of compulsive Internet use, research should also be considered for medication approaches in treating Internet addiction. No research has currently been conducted in this area. In severe cases, residential inpatient care may be warranted. Inpatient treatment programs provide coordinated and integrated services in hospital settings. Specialized recovery centers are emerging in China, Korea, and Taiwan. In the United States, the new Restart Program in Redmond, Washington, has opened, providing 45-day intensive care for Internet addicts. Generally, the goal of inpatient treatment is to provide a protective environment that includes medical stabilization, support, treatment for psychiatric or addictive disorders, and supervision. Inpatient treatment may combine group and individual treatments with family education and therapy as needed, especially for child and adolescent patients.

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Assessment and Treatment of Compulsive Buying

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Abstract

Compulsive buying disorder (CBD) affects a significant percentage of those in the general population. However, CBD has not received as much attention with regard to research and training providers in its assessment and treatment as other psychiatric disorders. Formal diagnostic criteria have been put forth, and there are a variety of assessment instruments for evaluating buying behavior using both questionnaire and interview formats. Based on the literature to date, it appears that disorder-specific cognitive-behavioral therapy has been most successful in treating those with CBD. Treatment providers should also be aware that mood disorders, anxiety disorders, and compulsive hoarding often accompany CBD and complicate its treatment, and these disorders should be addressed within the treatment paradigm. Further research is needed to define barriers to motivation for change and treatment compliance among individuals with CBD, as well as factors associated with both positive and negative treatment outcomes.

Keywords: compulsive buying, Compulsive Buying Scale, impulse control disorder, compulsive hoarding, cognitive-behavioral therapy

Introduction

Compulsive buying disorder (CBD) is characterized by shopping and buying behavior that results in marked psychological distress as well as financial and interpersonal problems (Faber & O'Guinn, 1992; O'Guinn & Faber, 1989). Although CBD may seem to have emerged only recently, Kraepelin (1909) and Bleuler (1983) originally described *onionmania*, or the urge to buy, a century ago. More recently, research has examined the prevalence of CBD as well as ways to classify its symptoms (Black, 2007). Using the Compulsive Buying Scale (CBS; Faber & O'Guinn, 1992), Koran and colleagues (2006) estimated the prevalence of CBD to be 5.8% within a large general population sample in the United States. Compared with other participants, those with CBD were younger and reported lower incomes but did not differ as to gender.

Compulsive buying disorder is not specifically described in the *Diagnostic and Statistical Manual of*

Mental Disorders (DSM-IV; APA, 1994) or in the *International Classification of Mental and Behavioral Disorders* (ICD-10; World Health Organization, 1994) but is currently conceptualized as an “impulse control disorder not otherwise specified.” In treatment-seeking individuals, the problematic buying behavior is associated with high rates of psychiatric comorbidity, especially mood and anxiety disorders, personality disorders, impulse control disorders (Black et al., 2000; Mitchell et al., 2002; Ninan et al., 2000; Schlosser et al., 1994), and compulsive hoarding (Frost et al., 2009; Mueller et al., 2007). Although research on this disorder has increased over the past 20 years, CBD remains frequently overlooked in clinical practice and many therapists lack specific information on its assessment and treatment.

Assessment

Compulsive buying disorder can be assessed by precise exploration of buying behavior and the

thoughts and feelings associated with buying and shopping. According to McElroy and colleagues (1994), who proposed diagnostic criteria, the disorder is characterized by frequent buying episodes or impulses to buy that are experienced as irresistible or senseless. The spending behaviors and impulses lead to personal distress, social, marital, or occupational dysfunction, and financial or legal problems. The excessive buying behavior does not occur exclusively during episodes of mania or hypomania. Individuals should be asked about these symptoms during the course of a general clinical interview. A variety of other interviews and assessment techniques may then be implemented if indicated.

Several interviews and questionnaires have been developed to assess CBD specifically or within the context of other impulse control disorders.

Questionnaires

COMPULSIVE BUYING SCALE (CBS)

The most widely used screening instrument in empirical research on CBD is the CBS (Faber & O'Guinn, 1992). This screening instrument consists of seven items utilizing a 5-point Likert scale for responses. Two items explore emotional reactions to shopping and five items ask about financial consequences of buying. The unidimensional scale reflects such characteristics as lack of impulse control, distress at the thought of others' knowledge of the person's purchasing habits, irrational use of credit cards, tension when not shopping, and the use of spending to feel better. Lower scores on the scale indicate higher levels of compulsive buying. The authors recommended a cut-off score of two standard deviations below the general population mean (-1.34) to identify those with compulsive buying. In the original study, the CBS correctly classified 89.9% of a general population sample and 85.3% of a compulsive buying group (Faber & O'Guinn, 1992).

CANADIAN COMPULSIVE BUYING

MEASUREMENT SCALE

This scale was originally written in French because the study was done with French-speaking consumers (Valence et al., 1988). The items have since been translated from French to English. The Canadian Compulsive Buying Measurement Scale consists of 13 items distributed among three dimensions: (1) tendency to spend (6 items); (2) reactive aspect (4 items); and (3) postpurchase guilt (3 items). Participants are required to express the extent to which they agree with each item on a 5-point Likert scale. The measure demonstrated adequate internal

consistency in the original study with 38 respondents with CBD and an equal number of noncompulsive buying individuals, with a Cronbach's alpha of .88 (Valence et al., 1988).

EDWARDS COMPULSIVE BUYING SCALE

Viewing compulsive buying as lying on a continuum from healthy to unhealthy buying behavior, Edwards (1993) developed the Edwards Compulsive Buying Scale to determine how compulsive or addictive subjects are in their spending behavior. The scale includes 13 items that load on five different factors: (1) tendency to spend (5 items); (2) compulsion/drive to spend (2 items); (3) feelings about shopping and spending (2 items); (4) dysfunctional spending (2 items); and (5) postpurchase guilt (2 items). Cronbach's alpha in a sample of 105 individuals with CBD and 101 subjects of a convenience sample for the entire scale was .91 (Edwards, 1993).

EXECUTIVE PERSONAL FINANCE SCALE

Spinella and colleagues (2007) developed the Executive Personal Finance Scale as a specific self-rating measure of executive aspects of personal money management regarding impulse control over spending, conceptualization of finances, and financial planning. The 20-item scale has four factors: (1) impulse control; (2) organization; (3) planning; and (4) motivational drive. Cronbach's alpha for the total score was found to be .86 in a sample of 225 participants (Spinella et al., 2007).

RIDGWAY COMPULSIVE BUYING SCALE

Ridgway and colleagues (2008) developed a six-item scale that includes dimensions of both obsessive-compulsive and impulse control disorders while excluding negative financial consequences and income-related items. The instrument is based on a definition of compulsive buying in terms of proposed underlying consumer behavior tendencies, separating consequence effects of the spending behavior. Cronbach's alpha in a sample of 551 university staff members was .81 (Ridgway et al., 2008).

Interviews

MINNESOTA IMPULSIVE DISORDER

INTERVIEW (MIDI)

The MIDI was developed by Christenson and colleagues (1994) for a descriptive study of compulsive buying. This semistructured interview consists of six separate screening modules examining criteria for the DSM-III-R impulse control disorders not elsewhere classified: trichotillomania, pyromania,

intermittent explosive disorder, kleptomania, and pathological gambling, as well as compulsive buying. Grant et al. (2005) reported that the MIDI had sensitivity of 100% and specificity of 96.2% for compulsive buying when comparing the instrument to the proposed diagnostic criteria of McElroy and colleagues (1994).

YALE-BROWN OBSESSIVE-COMPULSIVE SCALE—SHOPPING VERSION (YBOCS-SV)

Monahan and colleagues (2006) modified the YBOCS (Goodman et al., 1989) to measure symptom severity and change in persons with compulsive buying. The Shopping Version (YBOCS-SV) includes 10 questions, 5 exploring cognitions and 5 rating behaviors associated with uncontrolled buying. Consumers are asked about time involved, interference due to the preoccupations or the shopping, resistance to the thoughts or behaviors, and degree of control over the shopping and buying cognitions/behaviors. The authors compared a group of individuals clinically identified as having compulsive buying and control subjects and reported good interrater reliability for those with compulsive buying ($r = .81$), for control subjects ($r = 0.96$), and for both groups combined ($r = 0.99$). Cronbach's alpha was moderately high for treatment-seeking individuals with compulsive buying (.65) and for comparison subjects (.70; Monahan et al., 2006). The YBOCS-SV was sensitive to clinical change and was able to detect improvement during a clinical trial. Thus, this instrument is best used as a severity measure rather than as a screening measure of compulsive buying.

SCID-I IMPULSE CONTROL DISORDERS

The impulse control disorders section of the Structured Clinical Interview (SCID; First et al., 2002) includes questions about DSM-IV impulse control disorders not otherwise specified including intermittent explosive disorder, pathological gambling, pyromania, trichotillomania, compulsive buying, and kleptomania. A more recent version that has not been formally field tested yet includes a number of questions for CBD, as well as for proposed impulse control disorders such as impulsive-compulsive nonparaphilic sexual behavior disorder, impulsive-compulsive Internet use disorder, and impulsive-compulsive skin picking disorder.

It is important to note that self-ratings and semi-structured interviews should not replace the clinical evaluation. Clinicians ought to inquire in detail about shopping and spending, particularly buying

attitudes, the extent of preoccupation with buying and shopping, buying behaviors, urges to buy, feelings and thoughts associated with buying, and interference with social, financial, and occupational functioning. Assessment further should include one of the self-ratings presented above. For example, Faber and O'Guinn's CBS (Faber & O'Guinn, 1992) is a useful instrument to screen for clinically significant compulsive buying. To measure the severity and interference caused by buying behaviors and cognitions, the YBOCS-SV (Monahan et al., 2006) should be administered; this instrument also allows for the measurement of clinical change within treatment.

Treatment

Pharmacological Treatment

Guidelines for the pharmacological treatment of CBD are lacking, and treatment research on this topic is limited. With regard to the high comorbidity with anxiety and affective disorders, individuals with CBD may benefit from medications that are successful in treating anxiety and depression. In 1991, McElroy and colleagues (McElroy et al., 1991) described the successful treatment of three individuals with CBD and comorbid mood disorder with antidepressants (bupropion, nortriptyline, and fluoxetine). Lejoyeux and colleagues (1995) reported two cases in which treatment of comorbid depression led to improvement of CBD. Until now, the best-studied class of drugs for CBD have been the selective serotonin reuptake inhibitors.

SELECTIVE SEROTONIN REUPTAKE INHIBITORS (SSRIS)

The results of open-label trials suggested that SSRIs could reduce compulsive buying (Black et al., 1997; Koran et al., 2002; McElroy et al., 1991). For example, Black and colleagues (1997) treated 10 nondepressed subjects with CBD with fluvoxamine in a dose of up to 300 mg daily for 9 weeks. Nine of the 10 patients improved and were less preoccupied with shopping. Koran and colleagues (2002) enrolled 24 subjects with CBD in an open-label citalopram treatment trial. Exclusion criteria were obsessive-compulsive disorder, bipolar disorder, substance abuse/dependence disorders, or psychotic disorders. Seventeen subjects showed a substantial response and significant improvement in compulsive buying behavior. However, small randomized, controlled trials failed to confirm the optimistic results of open-label trials. Table 30.1 summarizes the results of the controlled psychopharmacological trials.

Table 30.1 Results of Medication Studies

Medication	Sample Size	Dosage/Day	Results
Fluvoxamine (Black et al., 2000)	Medication $n = 12$, Placebo $n = 11$	220 mg	Drug = Placebo
Fluvoxamine (Ninan et al., 2000)	Medication $n = 20$, Placebo $n = 17$	215 mg	Drug = Placebo
Citalopram (Bullock & Koran, 2003)	Open-label trial ($n = 24$) followed by double-blind, placebo-controlled discontinuation trial Responder $n = 15$ ↓ Medication $n = 7$, Placebo $n = 8$	20–60 mg	Five of eight randomized to placebo relapsed compared with none of seven randomized to continue taking medication
Escitalopram (Koran et al., 2007)	Open-label trial ($n = 26$) followed by double-blind, placebo-controlled discontinuation trial Responder $n = 17$ ↓ Medication $n = 8$, Placebo $n = 9$	10–20 mg	Drug = Placebo

Two subsequent double-blind, placebo-controlled studies (Black et al., 2000; Ninan et al., 2000) did not find a significant difference in efficacy between fluvoxamine and placebo. In the study of Black and colleagues (2000), primary outcome measures included the YBOCS-SV, the National Institute of Mental Health Obsessive-Compulsive Scale (NIMHOCS), and three Clinical Global Impression (CGI) ratings. The improvement experienced by fluvoxamine recipients was similar to that of the placebo group, suggesting a high placebo response rate. Likewise, Ninan and colleagues (2000) failed to demonstrate significant differences between fluvoxamine-treated and placebo-treated group using the YBOCS-SV, the Hamilton Rating Scale for Depression (HAM-D), and the Global Assessment of Functioning (GAF) as outcome measures. Investigators in both studies assumed that shopping diaries, reviews of shopping behaviors at study visits, and nonspecific support during these visits may have contributed to the positive response.

Two subsequent open-label trials by Koran and colleagues were followed by a double-blind, placebo-controlled discontinuation phase (Koran et al., 2003, 2007). These studies yielded mixed results. In the first study to test the effectiveness of citalopram (Koran et al., 2003), primary outcome measures were the relapse rate in the double-blind portion of the study and the change in YBOCS-SV from randomization baseline to endpoint. Investigators decided to omit shopping diaries to avoid their potential therapeutic effect. Results of the double-blind

phase suggested a true drug effect. Subjects randomized to double-blind citalopram did not report relapses and showed significant lower YBOCS-SV scores at endpoint. In the second study, Koran and colleagues (2007) used an identical study design to test escitalopram for CBD. The similar relapse rates in the drug and placebo groups during the double-blind phase contradicted the findings of the previous study conducted with citalopram (Koran et al., 2007).

Overall, it seems likely that the improvement in compulsive buying during open-label SSRI treatment was not a true drug effect. Similar medication and placebo rates suggest that the promising results of open-label medication trials may have largely been a placebo response. However, it cannot be excluded that the medication trials failed to show a drug effect due to the lack of power.

OPIATE ANTAGONISTS

Dopaminergic reward pathways may be involved in CBD, and opioid antagonists appear to reduce buying urges (Bullock & Koran, 2003). There have been several treatment studies involving opiate antagonists for the treatment of other impulse control disorders, particularly pathological gambling (Grant et al., 2006, 2008) and kleptomania (Grant et al., 2009). With regard to CBD, only case reports rather than controlled trials have been published. For example, Kim (1998) described the treatment response to the opiate antagonist naltrexone (100 mg/day) in a female patient with CBD and three other cases. Grant (2003) reported improvement of

CBD in three patients treated with naltrexone using higher doses (100–200 mg/day).

In summary, pharmacological studies on compulsive buying are limited by small sample sizes, inadequate numbers of male subjects, and high placebo response rates. Larger controlled pharmacological studies are needed to determine whether or not medication may be effective in the treatment of CBD.

Psychotherapy Treatment

The earliest case reports illustrated psychodynamic considerations in therapeutic interventions for CBD (Krueger, 1988; Lawrence, 1990; Winestine, 1985). Winestine (1985), in his case report, described a woman in her late 30s who sought treatment because of uncontrollable shopping sprees. The psychoanalytical treatment focused on the role of her remembered childhood seduction and demonstrated the interdigititation of childhood abuse with the development of intrapsychic conflicts and adult compulsive buying symptoms. With regard to Krueger (1988), who reported four casuistics, CBD may occur as a reparative effort for a fragmented sense of self and the experience of emptiness. The author recommended as therapeutic interventions emphatic resonance, understanding, and development of a more cohesive sense of self. Lawrence (1990) suggested that castration anxiety could be the main motivation of female compulsive buying.

A letter to the editor by Bernik and colleagues (1996) reported on two women with CBD. Both women suffered from comorbid panic disorder that was successfully treated with clomipramine (150 mg/day), with no effect on compulsive buying. To treat CBD, behavioral therapy was conducted with exposure to external cues and response prevention techniques (i.e., walking around street markets where buying attacks used to occur, touching the objects but not buying anything, first accompanied, then alone). Bernik et al. reported that after 3 to 4 weeks of daily exercises, distress and compulsive buying disappeared.

In recent years, several group cognitive-behavioral therapy (CBT) manuals have been published in a variety of languages (Benson & Gengler, 2004; Damon, 1988; Mitchell, 2010; Müller et al., 2008). Although psychotherapy research on this topic remains limited, CBT has been shown to be helpful in two randomized, controlled studies comparing the efficacy of group CBT to that of a waiting list control (Mitchell et al., 2006; Mueller et al., 2008). Both studies have tested the efficacy of the CBT program described in detail by Mitchell (2010).

This group therapy consists of 12 weekly sessions and specifically aims to interrupt and control the compulsive buying habits, to identify and restructure maladaptive thoughts and feelings associated with compulsive buying, and to establish healthy purchasing patterns. Treatment interventions focus on delineating factors that maintain the uncontrolled buying episodes and strategies for controlling shopping and buying. In addition, more general sessions on self-esteem, problem solving, and stress management are included.

Mitchell and colleagues (2006) conducted a pilot trial with female adults with current compulsive buying problems; 28 individuals were assigned to receive active treatment and 11 to the waiting list condition. The findings at the end of treatment showed a substantial reduction in the number of compulsive buying episodes and time spent buying, as well as improved scores on the YBOCS-SV and the CBS in the CBT group. The significant improvement was maintained at 6-month follow-up.

The results of the German replication study (Mueller et al., 2008) supported Mitchell and colleagues' findings. In the German psychotherapy study, 51 women and 9 men with current CBD were enrolled. Thirty-one participants were subsequently randomly assigned to the CBT group and 29 to the waiting list condition. The analyses of the primary outcome variables on the CBS, the YBOCS-SV, and the German Compulsive Buying Measurement Scale found that CBT, compared with the waiting list condition, resulted in a reduction of compulsive buying behavior. Predictor analysis showed that poorer attendance at the group therapy sessions and higher pretreatment compulsive hoarding traits, as measured with the Saving Inventory-Revised (Frost et al., 2004), were significant predictors of a poor outcome (Mueller et al., 2008).

Although the principal disorder was CBD, in the U.S. as well as in the German study, a broad range of comorbid psychiatric disorders were found. The large number of concurrent Axis I disorders was consistent with previous findings (Black et al., 2000; Mitchell et al., 2002; Ninan et al., 2000; Schlosser et al., 1994). Since the typical treatment-seeking patient with CBD suffers from comorbid psychopathology, the inclusion of patients with concurrent psychopathology increased the external validity of these two psychotherapy studies (Rothwell, 2005). Further treatment research is needed to examine specific psychotherapeutic effects and to develop subtyping strategies to account for individual comorbid psychopathology (e.g. compulsive hoarding).

In addition, self-help groups, financial counseling, and couples' counseling may be helpful to individuals with CBD. Several self-help books for CBD are already available (Arenson, 1991; Benson, 2001, 2008; Catalano & Sonenberg, 1993; Wesson, 1991). In general, it should be noted that there is a need to evaluate the efficacy and effectiveness of bibliotherapy.

Cognitive-Behavioral Psychotherapy Strategies

Since there is evidence that CBT may be effective, the following sections describe the main cognitive-behavioral strategies that are helpful in the treatment of CBD.

SELF-MONITORING: PURCHASING RECORDS

Self-monitoring is considered essential in precisely describing the frequency of and situational circumstances involved in shopping episodes. Patients should be asked to complete daily purchasing records. The self-observation requires patients' active cooperation and may represent the first step in changing excessive buying behavior. Purchasing records can help develop a clearer picture of compulsive buying antecedents and consequences and allow the clinician to track progress in terms of severity and frequency of buying/shopping episodes, as well as intensity of urges to buy. Sometimes simply the systematic filling out of purchasing records leads

to a reduction of purchasing attacks, at least temporarily. Table 30.2 shows an example of a completed purchasing record.

It is important to note that for individuals with CBD, it can be difficult to differentiate between appropriate (normal) and inappropriate (compulsive/pathological) purchases. Most persons with CBD tend to minimize and justify their impulsive purchases. For example, one patient did not recognize that the monthly purchasing of completely new decorations for her apartment was inappropriate. Another patient justified the purchase of 10 gardening books with overlapping contents as necessary in that she might, in the future, want to begin an education in gardening.

MOTIVATION

Therapeutic progress requires that clients are ready to change. To clarify the motivation to change, individuals with CBD should examine the benefits of normalizing their buying behavior. At the same time, it is important to consider personal resources and weaknesses. The pros and cons of normalizing the buying behavior must be discussed at the beginning of treatment. Often CBD has extremely negative social and financial consequences for the affected persons and their relatives. Treatment-seeking patients with CBD are mostly externally motivated to change (e.g., because of severe conflicts with their spouses and huge debts). Furthermore,

Table 30.2 Purchasing Record

Date	Time	Items	Cost (\$)	Thoughts	Feelings
10/05	4:00 p.m.	Shoes for my son	80	Pricey, but he needs new shoes	Pleasure
10/06	6:00 .pm.	Underwear	100	Nice	First proud, then remorseful
10/08	3:00 p.m.	Shirts, jeans	100	I really need the jeans, I liked the T-shirts	Confused
10/09	6:00 p.m.	Makeup, perfume	40	Was on sale	Happy
10/10	4:00 p.m.	Shirts, jeans, gifts	250	Nice jeans, gifts for a potential future occasion	First felt happy, then was confused, felt sad, guilty
10/11	2:00 p.m.	6 pairs of slippers DVDs 2 sets of glasswear Wii game for my son	100 30 25,- 60	Cannot decide, slippers are on sale, want it, don't really need that, but like it so much Son will be happy	First happy, excited, then guilty, ashamed
10/12	10:00 p.m. (online)	Wii controller for my son Wii games for my son	200	Can't really afford them, but want it Want to please my son	Restless, irritable, after ordering very guilty and remorseful, anxious about the debts

compulsive buying can be used as a short-term coping strategy to avoid negative mood states and conflicts. Individuals with CBD should know what to expect to as they modify their buying habits and consider whether they want and are able to invest in this change. According to Miller and Rollnick (2000), the motivation to change can be influenced by the therapist's behavior (e.g., supportive and empathetic styles of intervention combined with an emphasis on the personal responsibility of the client for change, active listening, and open-ended questions enhancing the patient's perceived self-efficacy). Table 30.3 summarizes some reasons for and against changing unhealthy buying habits that were expressed by participants in a therapy group.

FUNCTIONAL ANALYSES

Using a model of short-term positive and negative reinforcement, compulsive buying episodes are used to escape from conflicts, to relieve negative mood states, or simply to reduce boredom. Compulsive buying disorder is maintained by its short-term positive consequences, whereas the negative consequences are often not considered. It is important that patients identify specific individualized cues that trigger their buying episodes and the short- and long-term consequences of their problem buying behavior. Patients should be encouraged to recognize their behavioral patterns, thoughts, and feelings associated with compulsive buying.

STIMULUS CONTROL

Patients can develop specific strategies to deal with environmental buying triggers identified in their functional analysis. Stimulus control strategies are designed to rearrange environmental cues for compulsive buying by encouraging the individual to avoid the buying cue entirely (e.g., discard catalogs/sale advertisements), restrict the stimulus field (e.g., shop only in specific stores), or strengthen cues for desired alternative behavior (e.g., spend more time with friends). It is also important that healthy buying behavior is increased and rewarded during treatment. Furthermore, techniques of internal stimulus control (e.g., internal monologues) are helpful. In addition to the physical avoidance of compulsive buying triggers, the responses to cues can be changed through the delay of response and the implementation of alternative behaviors.

ALTERNATIVE BEHAVIORS

One way to decrease compulsive buying is to develop alternative behaviors in which to engage instead of going shopping. Patients should schedule realistic positive activities to engage in during future high-risk situations identified in their functional analysis (long-term plans) or plan alternative behaviors for moments when they feel shopping urges (short-term plans). It is important to establish an individualized list of alternative behaviors for both types of situations.

Table 30.3 Reasons For and Against Changing Unhealthy Buying Habits

Pros	Cons
Excessive debt, no financial security	Ability to buy whatever and whenever I want
Conflict at home	Provides some security to me, self-esteem builder
No space at home, clutter	Escape from problems and bad moods
Remorse, loss of control	Get pleasure, entertainment if feeling lonely, void filler
Legal problems	Relief from tension, stress, anger, sadness, boredom
Anxious about losing my job	Self-present
Lies, hiding, no openness and honesty	Enjoy the salesperson's attention and compliments
Worries about daughter, who also started to buy compulsively	Gifts are a way to please to others
Depression, guilt, shame	Social reinforcement for appearing attractive, well off
Social withdrawal	Distraction from aversive tasks, release, time-out

COGNITIVE RESTRUCTURING

Cognitive techniques are described in detail in many textbooks (Beck et al., 1987). Strategies for eliciting and testing dysfunctional thoughts that become associated with shopping and buying are an essential element in the psychotherapeutic treatment of individuals with CBD. Patients are encouraged to become aware of the maladaptive styles of thinking in which they engage that may lead to problematic shopping responses and to further question, test, and modify these thoughts. For example, patients are asked to keep records of dysfunctional thoughts in which they record the emotions and automatic thoughts that occur in buying/shopping situations. Patients are further taught to develop more rational responses to their maladaptive thoughts that trigger compulsive buying episodes and to record them in the appropriate column (see Table 30.4).

BEHAVIORAL CHAINS

Most of the time, problem buying behavior consists of a series of components in which each aspect represents a link in a behavioral chain. Figure 30.1 presents an example of such a chain. It is important to obtain a description of the events co-occurring with the onset of the compulsive buying attack. Becoming aware of such events may be difficult. Specifying an initial prompting event and the series of cues, thoughts, and feelings that trigger the

problematic buying episode creates an opportunity to break the chain early in the cycle and to avoid compulsive buying behavior.

EXPOSURE

In learning to control urges to buy impulsively, it is important that individuals with CBD learn to expose themselves to high-risk buying situations and to avoid buying in those situations. This is important because they will periodically be in situations where urges and opportunities for compulsive buying arise. Exposure and ritual prevention techniques have been found to be quite effective in the treatment of other disorders and are described in many textbooks (Craske & Barlow, 2008; Foa & Kozak, 1996).

MONEY MANAGEMENT

Materialistic values, maladaptive patterns of beliefs about money, and deficits in money management are typical in individuals with CBD (Dittmar, 2005; Rose, 2007; Mueller et al., 2010). Due to the suggested importance of psychological and social factors, as well as consumerism and attitudes toward money, treatment should focus on the improvement of money management as appropriate. Noting that credit card use has a moderating effect on compulsive buying and can provoke excessive spending behavior (Raghbir, 2008; Roberts & Jones, 2001),

Table 30.4 Cognitive Restructuring

Cues	Responses	Revised Responses	Consequences	Revised Consequences
Invitation to a birthday party	<p><i>THOUGHTS</i></p> <p>There will be only academics there. At least I should look nice. I need new clothes.</p> <p><i>FEELINGS</i></p> <p>I feel self-conscious, a little anxious, ashamed.</p> <p><i>BEHAVIORS</i></p> <p>I buy a completely new outfit: new pants, blouse, shoes, jewelry.</p>	<p><i>THOUGHTS-REV</i></p> <p>I'm not less intelligent than the other guests.</p> <p>I have enough nice clothes in my closet.</p> <p><i>FEELINGS-REV</i></p> <p>Still a little irritable, but feel better, calm.</p> <p><i>BEHAVIORS-REV</i></p> <p>Wear something from my closet.</p>	<p><i>SHORT -TERM</i></p> <p>Excited about the chance to buy something new.</p> <p><i>LONG -TERM</i></p> <p>Again failed.</p> <p>More debts.</p> <p>Angry with myself and desperate.</p>	<p><i>SHORT-TERM-REV</i></p> <p>My thoughts and worries about invitation and potential shopping decrease.</p> <p><i>LONG-TERM-REV</i></p> <p>I can manage these situations. Proud.</p>

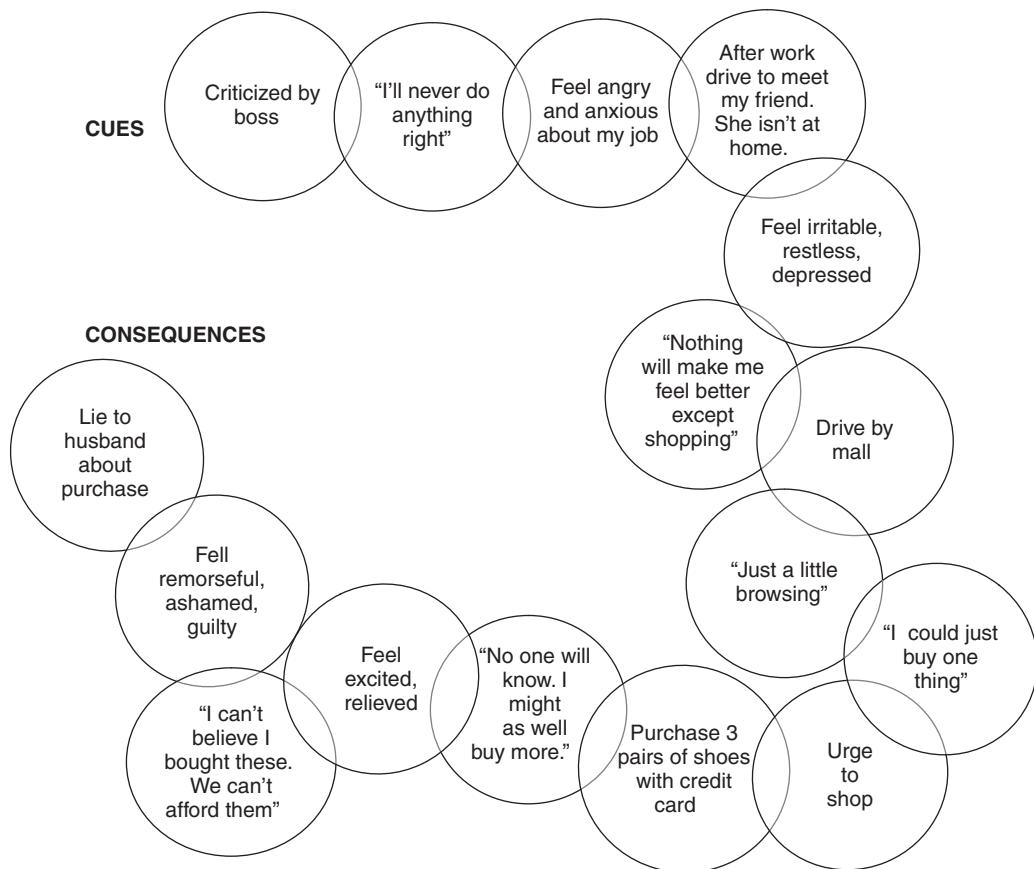


Fig. 30.1 Behavioral chain.

the closing of credit card accounts may be helpful to limit excessive purchases. Patients with CBD should be encouraged to give up their credit cards and to use cash or debit cards instead. In addition, recommendations for appropriate money management should be discussed, including the operationalization of appropriate consumer habits.

COMPULSIVE HOARDING

A strong association between compulsive hoarding and compulsive buying has been reported (Frost et al., 2002). The most visible symptom of compulsive hoarding is clutter. People with compulsive hoarding are unable to discard the purchased items. Acquisition has been defined as a pattern of compulsive hoarding including both the acquisition of free items and compulsive buying (Frost et al., 2009). Individuals with CBD often do not use the things they buy, and many of them do not even unwrap the items or remove the tags; instead, they store or hoard them. In a treatment-seeking sample of individuals with compulsive buying, the severity of hoarding symptoms was associated with

the severity of compulsive buying (Mueller et al., 2007). Furthermore, a psychotherapy treatment study with patients with CBD has reported that those with hoarding symptoms were significantly less likely to respond to CBT than nonhoarding patients (Mueller et al., 2008). Because hoarding patients with CBD might be embarrassed to admit their hoarding behavior, especially the amount of clutter in their homes, there is a need for screening of compulsive hoarding and specific treatment interventions to reduce hoarding behavior that focus not only on acquisition but also on clutter and difficulty discarding items.

GROUP VERSUS INDIVIDUAL THERAPY

Based on our clinical experience with patients with CBD, group treatment may be as effective as or more effective than individual therapy. Individuals with compulsive buying are mostly embarrassed about their buying behavior and are used to hiding their spending patterns from friends, family members, and therapists. In the CBD specific group, many of them first have the heart to open up about their

excessive shopping and buying, the extent of their preoccupation, and the consequences. Furthermore, many individuals with CBD are prone to rationalize their buying attacks. The understanding and feedback of other people with the same inappropriate behaviors and thoughts stimulate them to overcome this problem. The strict focus on normalization of spending patterns associated with group cohesion and support by other group members may motivate patients to modify their spending habits.

Summary

Compulsive buying disorder is defined as frequent buying of more than can be afforded and frequent buying of items that are not needed. The buying impulses and behaviors cause marked distress, significantly interfere with social or occupational functioning, and result in interpersonal difficulties and financial problems. Compulsive buying disorder is associated with significant psychiatric comorbidity, particularly mood and anxiety disorders, compulsive hoarding, and personality disorders. It is currently conceptualized as an “impulse control disorder not otherwise specified.” Compulsive buying disorder affects a significant percentage of the general population, but it has received much less attention with regard to research and training providers in its assessment and treatment compared to other psychiatric disorders with similar prevalence rates. Formal diagnostic criteria have been put forth, and there are a variety of assessment instruments for evaluating compulsive buying behavior with both questionnaire and interview formats.

Based on the literature to date, it appears that disorder-specific CBT has been most successful in treating CBD. Cognitive-behavioral treatment should include daily purchasing records, functional analysis, stimulus control strategies, increased use of alternative behaviors, identification of behavioral chains, cognitive restructuring, improvement of money management, and exposure practice. Treatment providers should also be aware that mood disorders, anxiety disorders, and compulsive hoarding often accompany CBD and complicate its treatment; these problems should be addressed within the treatment paradigm.

Future Directions

Further research is needed to test the efficacy of other methods of treatment, especially pharmaceutical approaches. With regard to psychotherapeutic treatment, psychiatric comorbidity should be

taken into account in tailoring treatment strategies. A subtyping approach might be useful in determining treatment needs.

Further research should also focus on barriers to motivation for change and treatment compliance among individuals with compulsive buying, as well as factors associated with both positive and negative treatment outcomes.

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PART 5

Impulse Control Disorders in Nonpsychiatric Clinical Settings

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Impulse Control Disorders in Medical Settings

Sean G. Sullivan

Abstract

Impulse control disorders (ICDs) and conditions with impulse control features provide a challenge in terms of identification, treatment, and follow-up when mental health specialists are in short supply. Medical settings, in particular the largest, primary health care, provide an opportunity to address many impulse-affected conditions currently poorly assessed and treated in health care settings. Barriers to intervention for ICDs in primary health care are time constraints; understanding of the etiology, symptoms, and appropriate interventions; the health and social costs; and prioritizing of training in and treatment of conditions perceived as more serious or appropriate to a primary health care service. These barriers may possibly be overcome in primary care settings, and in this chapter, a model to address problem gambling is described.

Keywords: impulsivity, health care, primary care, interventions, gambling

Impulse Control Conditions

Impulse control disorders (ICDs) comprise a small group of recognized mental health conditions; a newer and developing group that may also be considered in the forthcoming revision of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM); and, lastly, a much wider group of recognized mental health disorders that have impulse control features. Many of the newer conditions, such as Internet overuse, currently appear to have some acceptance within the broad category of addiction (Block, 2008). Aspects of compulsion have been identified within ICDs. Another view is that these conditions exist within an obsessive-compulsive spectrum, providing possibly a complex explanation of their etiology and treatment (Grant & Potenza, 2006; Hollander, 1993; Stein et al., 2006).

Recognized ICDs include pathological gambling, trichotillomania, kleptomania, pyromania, and intermittent explosive disorder. Other conditions that have similarities include compulsive

buying, compulsive sexual behavior, compulsive gaming, and compulsive Internet use. Compulsive Internet use can also be subcategorized into a range of expanding cross-linked behaviors, including Internet pornography addiction, chat room addiction, Internet gaming addiction, Internet gambling, and texting and e-mail addictions. Conditions with impulsive or compulsive features but categorized within another field are wide-ranging and include obsessive-compulsive disorder, substance use disorders, schizophrenia, personality disorders, conduct disorder, paraphilic, and mood disorders (APA, 2000; Grant & Potenza, 2006).

The heterogeneity of these disorders provides a rich source of future research; however, the widespread clinical criteria can also present a challenge to those working in nonpsychiatric clinical settings. Recent research has found that every third patient in primary care showed signs of psychiatric symptoms, such as depression or anxiety, or of alcohol problems, indicating substantial opportunity as well

as evidence of substantial need (Nordstrom & Bodlund, 2008).

Primary Care Settings

Medical settings are widespread, including medical centers, hospitals, specialist medical services, and, more recently, medical services integrated with other disciplines, such as psychological and clinical social services (WHO, 2008).

The most accessible medical settings are categorized as primary care. They are the first point of contact of the public with the medical system and are often generalist in perspective. Although the health professional who typically represents primary care settings is the family doctor or general practitioner (GP), many other health professionals may support the delivery of a primary care service. These include nurses (general practice nurses, community nurses, and nurse practitioners), social workers, psychotherapists, physiotherapists, opticians, and others, including pharmacists, midwives, and dentists. Although attempts are made to define primary care services, there is “in practice no absolute or consistent view about whether particular settings and services are part of primary health care or not” (COA, 2008, p. 10).

Accessibility and Opportunity to Treat

Primary services may allow a very high proportion of the public access to skilled services. Examples include the United Kingdom, where 86% of all health needs are managed, with 15% of the entire population seeing their family or general practice (GP) physician in a two-week period (RCGP, 2004), and Australia (COA, 2008) and New Zealand (MOH, 1999), where at least 80% of the population access their GP annually. There is now a blurring in primary care of traditional boundaries between curative care, preventive medicine, and health promotion (WHO, 2008).

In the past, mental health issues were not well addressed in primary services. This situation has changed in generalist services, with improvement in screening, greater awareness of the burden of illness that patients carry, and strategies to manage mental health disorders in general practice (Khin et al., 2003). The National Comorbidity Survey Replication (Kessler, et al., 2004) found that people with mental health or substance abuse disorders were more likely to be treated in a primary care setting; 22.8% of them were treated by a primary care physician, a nurse, or another generalist, compared with 16% who were treated by a social worker, psychologist,

or counselor and just 12% by a psychiatrist (Kaplan, 2005).

Continuity of Care

The opportunity to inquire about previous health issues during subsequent patient visits (referred to as *longitudinality*) has been found to be critical to better health outcomes (WHO, 2008; Starfield, 1998). In reaction to the complexity of patients' needs, including mental health and multicomorbidity, there has been an increased focus on more comprehensive and person-centered approaches and continuity of care. The organizing of primary care networks and centers enables accessibility for both sick and healthy patients, and may involve the primary care provider in coordinating with other levels of care (WHO, 2008).

Screening for Mental Health Disorders in Primary Care

Many GPs rely on the presence of physical symptoms to identify psychiatric disorders. Although there is a strong relationship between physical symptoms and disorders, GPs' ability to recognize psychiatric disorders using this method has been found to be low (Rasmussen et al., 2008). A range of mental health screens have been used in primary care settings, commonly to identify depression, anxiety, or clinical stress (Leon et al., 1996). These are relatively brief and often self-administered. They include the Kessler K10 (10 items), which targets distress that may be due to depression or anxiety; the Patient Health Questionnaire (PHQ-9; 9 items), which targets depression; and the General Anxiety Disorder Scale (GAD-7; 7 items), which targets anxiety. Recently, because of the need in primary care for very brief, easy-to-interpret tools, there has been a drive to develop even briefer screens, such as the four-item Brief Health Questionnaire to identify depression and anxiety (Lang et al., 2009).

An alternative approach is a regular health screener, especially for asymptomatic conditions. An example is the “lifestyle” screener, the Case-find and Help Assessment Tool (CHAT), which is completed by patients (24 items). It is a composite questionnaire that inquires about nine mental health and lifestyle behavior topics, combining 9 one- or two-question screens, followed by a help option question (BPAC, 2009; Goodyear-Smith et al., 2008). Although screening in primary care settings is common, evidence of its effectiveness is often not strong. In the U.S. Preventative Services Task Force screening recommendations for the Mental Health

Conditions and Substance Abuse category, only smoking cessation is strongly recommended, with depression and alcohol misuse falling at the lower recommended level. Depression intervention includes screening and follow-up, while the recommendations for alcohol misuse include screening and behavioral interventions. Notably, screening in primary care for both illicit drug use and suicide risk is not recommended because of insufficient evidence for or against the effectiveness of routine screening for these problems (USPSTF, 2008; USPSTF, 2004). The Preventative Services Task Force supports the screening of some mental health conditions with features of impulse control deficits, such as alcohol misuse and tobacco use. There is insufficient evidence at this stage for the screening of other conditions (illicit drug use and suicide risk) and no advice regarding problem gambling or other ICDs.

ICDs and Primary Care

Similarities in the symptoms of ICDs described in the DSM-IV include an increasing sense of tension or arousal prior to the behavior, followed by pleasure, gratification, or relief during the behavior and then by regret, self-reproach, or guilt. However, there is a rider that this description applies only to “most” of these ICDs, and that negative cognitions and emotions following these behaviors may or may not occur (APA, 2000; WHO, 1992).

The WHO *International Statistical Classification of Diseases and Related Health Problems* (10th Revision; ICD-10) categorizes specific ICDs under Habit and Impulse Disorders that are not classifiable under other categories, and that are characterized by repeated acts that have no clear rational motivation, cannot be controlled, and generally harm the patient’s own interests and those of other people (WHO, 1992). These disorders are further described as being poorly understood from a causation perspective and as being grouped together only because of broad similarities of description, not because they appear to share other important features. In ICD-10, pathological gambling, pyromania, kleptomania, and trichotillomania are specifically described, with a remaining catchall category for unspecified habit and impulse disorders.

Trichotillomania

Although disorders involving the hair are common in primary care practices (Kordon et al., 2003), cases of trichotillomania in this setting are unusual, although not unknown. Behavioral therapy and pharmacotherapy with selective serotonin reuptake

inhibitors (SSRIs) or clomipramine have been compared, with habit reversal therapy being found superior to pharmacotherapy (Bloch et al., 2007). A recent study of *N*-acetylcysteine found a statistically significant reduction in symptoms. The authors stated that behavioral therapy in conjunction with pharmacotherapy warrants further research for what appears to be a disease requiring long-term therapy (Grant et al., 2009b). Although continued care in primary care settings may appear appropriate for this condition, behavioral therapies, even though they are effective, may exceed the time available in primary care, while pharmacotherapy, with or without behavioral therapy, appears to require further evidence of effectiveness. In this climate of uncertainty concerning the treatment approach and possible time requirements, even though the symptoms of hair pulling are obvious if uncovered, it appears likely that primary care health providers will refer the patient to specialist mental health providers for treatment. The same conclusion might be reached for compulsive skin picking, although similar symptoms can occur with abuse of methamphetamine and other drugs (formication), which may require even further assessment.

Intermittent Explosive Disorder

Aggressiveness and loss of control disproportionate to the stimulating event is again unusual, and symptoms may overlap with withdrawal effects from abuse of methamphetamine and other drugs (Kessler et al., 2006). Kessler and colleagues (2006) identified a lifetime prevalence of intermittent explosive disorder of 7.3% and a 12-month prevalence of 3.9%, with 28.8% of individuals having received treatment for their anger. Comorbidity with attention deficit hyperactivity disorder (ADHD) was found to be high (19.6%–22.5%); therapy with long-acting stimulants has been suggested as appropriate. Once again, the effects are similar to those of withdrawal from drug abuse, which may or may not be a consideration; however, this possible overlap may complicate primary care intervention and influence the preference for referral.

Kleptomania and Pyromania

Both of these conditions are relatively rare; they are often found in forensic treatment services rather than in primary care. Levels of lifetime diagnoses of kleptomania are high. Treatment is not definitive; a combination of pharmacotherapy and psychotherapy may be appropriate (Dannon et al., 2004; Koran et al., 2007). Naltrexone has been found to

be effective in the treatment of kleptomania (Grant et al., 2009a). The difficulty of identifying kleptomania and pyromania outside of a forensic situation, and assessing these and coexisting conditions suggests that they will rarely be addressed in primary care settings; referral to a specialist is the likely consequence.

Pathological Gambling

Persistent and recurrent maladaptive gambling has been recognized as a possible ICD that could be addressed within a primary care setting and viewed as a chronic medical condition (Morasco et al., 2006; Potenza et al., 2002; Sullivan et al., 2006). With the increase in harm that occurs as the problem gambling progresses, there is a strong benefit in identifying the condition at an early stage (Wardle et al., 2007). Kessler and colleagues (2008) found that 96.3% of problem gamblers had at least one other lifetime mental health disorder, indicating a population with a strong health need. In addition, Zimmerman et al. (2006) found that among psychiatric outpatients, pathological gamblers were significantly more likely to be affected by another ICD; the prevalence rate for coexistence was 20%. Kessler et al. (2006) found that pathological gamblers had an even higher rate (42.3%) of disorders with impulse control deficits (oppositional-defiant, conduct, attention deficit hyperactivity, or intermittent explosive disorders).

Primary Health Care Challenges

General practitioners or family physicians are considered to be in a good position to identify ICDs such as problem gambling, especially given their ability to address the personal, family, and social problems that the gambling may cause (Kramer, 1997; Sullivan et al., 2006; Unwin et al., 2000). With the advent of newer types of impulse disorders such as Internet addiction, which have raised growing concerns in medical settings throughout the world, GPs may have a further role to play (Block, 2008; Petry, 2006). However, there are several barriers to the treatment of ICDs in primary care. These will now be discussed.

Time Limitations

One of the major impediments to addressing ICDs is the limited time that busy primary health providers have to identify, address or intervene, to discuss health issues and give feedback. General practitioners and other health professionals may often be

restricted to 15–20 minutes per patient, and in a busy practice, extended consultation results in delays for other patients, increased stress for other sick patients, and even overrunning deadlines for clinic consultation. The scarcity of GPs in many countries often requires them to prioritize time, since the next patient may have a more important and more curable illness; at the same time, there is a broad focus on patients, their families, and community health (Bowman, 2009). In a time allocation study in the United States, the average time for 390 consultations was 17.4 minutes, covering 6.5 topics per session. Of over 2500 topics, more than 70% were biomedical, 12% were psychosocial, 7% were personal habits, and less than 4% were mental health concerns (Tai-Seale & McGuire, 2006).

General practitioners have indicated that lack of time was the greatest barrier to their intervening in gambling problems in their patients or their family members, especially if coexisting depression was identified (Sullivan et al., 2006). A possible solution may be referral to other health professionals in the practice with sufficient time, provided that GPs are willing to allow greater nurse or medical assistant involvement in the patient's care (Aspy et al., 2008). Additional funding to address mental health issues would also help by reducing the GP's workload or adding trained health care professionals to the primary care center.

Screening

Early principles of screening for asymptomatic conditions stated that the condition should be a significant health problem and that the natural history of the condition should be understood (Whitby, 1974; Wilson & Jungner, 1968). There has been a growing demand in preventive services for evidence of the effectiveness of the screening outcome, especially in reducing morbidity or mortality, before assigning limited clinical resources (Harris et al., 2001). Other important considerations are the prevalence of the condition and the effectiveness of treatment for identified patients.

Impulse control disorders are diverse, their natural history is not well understood, and there is incomplete evidence that screening will reduce their associated morbidity or mortality. Therefore, as a group, ICDs face challenges in being prioritized for screening. However, GPs have indicated that they have a role in helping patients with gambling problems, provided that training is available, and patients

have acknowledged that this is an appropriate role for GPs (Sullivan et al., 2006). Screening of less obvious problems remains important, as it has been recognized that patients are more likely to present with problems other than psychological ones (Kordon et al., 2003). Screens, however, have to be user friendly for medical settings; that is, they must be quickly administered, scored without delay and without the need to resort to scoring matrices, be valid, and have utility. In addition, patients need to overcome the shame or guilt that may result in denying their symptoms. A unifying feature of the ICDs is that there is discomfort in disclosing the behaviors. By describing the behavior as a health issue, and by normalizing the inquiry to one relevant to the setting and, if possible, the presenting condition, the screen may become more sensitive.

Skills/Training Needs

For many primary health generalists, the ability to identify and intervene in core mental health conditions is a challenge. Those generalists who feel untrained or not competent to address the disorders may not be motivated to intervene. General practitioners are generally required to continue their training throughout the life of their practice, but they can decide which training to select. For many, qualifications for reregistration will follow training courses, with the professional college providing approval. Therefore, it is often necessary to make a case for specific training points and obtain approval from the college. This approval has been obtained for specific stand-alone training (Sullivan et al., 2006), but it is more likely to be integrated into the general practice if ongoing approval is obtained. Practice guidelines have been developed for GPs in some Australian states for screening and intervening in problem gambling, but currently this remains the exception to the rule (Jackson & Thomas, 2009).

Stigma

As with many substance addictions, impulse control disorders may be perceived as retaining some degree of control, raising a perception of self-indulgence. Furthermore, in the absence in impulse control disorders of the chemical present in substance addictions, no attribution can be directed toward control by the chemical over the behaviour to mitigate this negative perspective. In addition, patients may be embarrassed about disclosing such behavior, even to a health professional, and may also fear the labeling that such a diagnosis may bring.

Knowledge

Impulse control disorders comprise an expanding group of behaviors that have many differing symptoms, displayed in different settings but otherwise covert, and have different appropriate interventions. Few GPS have the knowledge to intervene effectively with ICD patients.

Coexisting Issues

It is axiomatic that if ICDs are relatively rare disorders, or perceived to have a relatively low burden of illness, they are unlikely to motivate screening. Therefore, ICDs may become identified only by serendipitous general inquiry or by direct disclosure by a patient.

Medication

An appropriate medical intervention that has recognized effectiveness may encourage screening and intervention by a primary health provider. Conversely, the absence of any such intervention may discourage such screening.

Funding

A barrier to intervention in mental health issues is funding to compensate for the extended time that may be required for optimal care. In addition, time spent screening for ICDs may result in assessment of fewer core disorders and/or less prevalent disorders (Holmwood, 1998). Access to and confidence in specialist referral services may also affect whether those in primary care screen for ICDs (Khin et al., 2003).

Primary Care Intervention in Alcohol Use Disorders

Alcohol use disorders have impulse control features but are commonly perceived as addictions (Derevensky, 2007). Interventions for these conditions may inform strategies to intervene in problem gambling, and perhaps in other ICDs, in primary care settings.

Screening for alcohol and other drug misuse in primary care settings is common (Mintzer et al., 2007; Yarborough, 2001). However, it remains underused, particularly for identification of hazardous alcohol use (Kypri et al., 2008).

Alcohol misuse has been assessed using questions embedded in general health questionnaires (Goodyear-Smith et al., 2004) or in brief screens such as the long-used four-item CAGE for alcohol dependence (Berks & McCormick, 2008; Ewing, 1984). The single question recommended by the

National Institute on Alcohol Abuse and Alcoholism and validated in primary care is the briefest one to date and has sought to address the underdiagnosis of alcohol problems in primary care:

"How many times in the past year have you had X or more drinks in a day?" (where X is five for men and four for women, and more than one time being a positive response; Smith et al., 2009; Taj et al., 1998)

A recent report concerning the challenges of screening for alcohol in primary care settings in Norway describes issues that could be generalized to other countries. The use of a brief intervention package was piloted; lack of confidence was found to be a major barrier. General practitioners believed that they were more competent to give advice on smoking than on alcohol abuse. This belief was combined with the lack of time available to give more intensive counseling on alcoholism. The GPs also preferred selective interventions for suspected high-risk consumption symptoms than more generalized brief interventions (e.g., general screening), which were also considered to be intrusive for their patients. Financial incentives for the additional counseling were also considered to be lacking; one example was a successful incentivized tobacco intervention strategy, although the authors noted that this was not always the case when a green prescription (diet and exercise) project was incentivized (Bringedal & Aasland, 2006). The authors concluded that, for Norwegian GPs, addressing a disorder such as alcohol misuse in an integrated treatment strategy was more appropriate and acceptable than brief intervention for the specific disorder.

Problem Gambling Interventions in Primary Care

Problem Gambling Screening

A variety of brief screens are available for the identification of problem gambling (SACES, 2005). Specialist GP problem gambling screens include items embedded in the 10-item composite CHAT screen, the MULTICAGE-CAD4, the 1-item PGRTC screen, and the 8-item EIGHT screen (Goodyear-Smith et al., 2008; Jackson & Thomas, 2009; Thomas et al., 2008; Rodríguez Monje et al., 2009; Sullivan, 2007).

THE MULTICAGE-CAD4

The MULTICAGE-CAD4 is a self-completed screen that measures eight forms of addiction behavior risk (alcohol, other drugs, gambling, Internet addiction, video game addiction, eating disorders, compulsive

shopping, and sex addiction) in a primary care setting. It was described as suitable for primary health care centers to identify often hidden addictions in patients presenting for other purposes (Rodríguez Monje et al., 2009).

THE EIGHT SCREEN

The Early Intervention Gambling Health Test (EIGHT) screen was developed for GPs in the 1990s and was designed to be completed in approximately 1 minute. Responses were restricted to two options (yes or no) in order to maintain brevity in its completion and its scoring. A score of 4 or more identified a problem; however, later research categorized scores into gambling or problem gambling (Sullivan, 2007). False positives were found to be low in a range of settings, including primary care, when the screen was measured either against other screens or against a clinical assessment by an experienced therapist. The EIGHT screen has been examined in a number of primary care settings, with positive responses from GPs, nurses, and patients (Penfold et al., 2006; Sullivan et al., 2000).

THE PGRTC SCREEN

A one-item screen developed by the Problem Gambling Research and Treatment Centre was adapted from the Canadian Problem Gambling Severity Index (CPGI). The single question "Have you ever had an issue with your gambling?" was correlated with the full CPGI. A sensitivity of 78.5% and a specificity of 96.4% were found. Twenty-two of 94 (23.4%) positives on the single screen were also on the CPGI and were true positives, while the balance (76.6%) were false positives. It was concluded that for a low-prevalence condition these were acceptable outcomes, with few cases of problem gambling missed by the single screen (Jackson & Thomas 2009).

CHAT

Composite health screens offer an important resource for the busy practitioner to assess a range of conditions in a brief, valid questionnaire. Such screens may be provided routinely or periodically to all patients or as needed. Composite screens are administered routinely and do not require indicators for use when applied in a generalist setting.

The Case-finding and Help Assessment Tool (CHAT) screens for nine lifestyle and mental health conditions, including pathological and subclinical problem gambling, depression, anxiety, alcohol misuse, other drug misuse, smoking, abuse and violence,

anger problems, and physical inactivity. These conditions are addressed in 24 items, allowing a wide range of inquiry in a relatively brief period, generally about 2 minutes. For each condition, one question asks if the patient wants help with that item (no/yes, but not today/yes; Arroll et al., 2005; see Table 31.1).

Alcoholism commonly coexists with problem gambling, as do anxiety and depression, smoking, and other drug abuse. For many of the issues screened for in the CHAT, embarrassment, shame, or guilt exist; by embedding these questions in a range of conditions, these barriers may be reduced. The screen is self-administered, and has been applied

Table 31.1 Case Finding and Help Assessment Tool (CHAT)

Please choose the answer that most correctly applies to you

CHAT Questions	Response Options	Positive
How many cigarettes do you smoke on an average day (<i>tick no if you do not smoke</i>)	None less than 1 a day 1–10 11–20 21–30 31-more	>10 cigarettes a day
Do you ever feel the need to cut down or stop your smoking?	No/Yes	Yes
Do you want help with your smoking?	No/Yes, but not today/Yes	Yes, but not today/Yes
Do you ever feel the need to cut down on your drinking alcohol? (tick no if you do not drink alcohol OR do not feel the need to cut down)	No/Yes	Yes
In the last year, have you ever drunk more alcohol than you meant to?	No/Yes	Yes
Do you want help with your drinking?	No/Yes, but not today/Yes	Yes, but not today/Yes
Do you ever feel the need to cut down on your non-prescription or recreational drug use? (tick no if you do not use other drugs OR do not feel the need to cut down)	No/Yes	Yes
In the last year, have you ever used non-prescription or recreational drugs more than you meant to?	No/Yes	Yes
Do you want help with your drug use?	No/Yes, but not today/Yes	Yes, but not today/Yes
Do you sometimes feel unhappy or worried after a session of gambling? (tick no if you do not gamble OR do not feel unhappy about gambling)	No/Yes	Yes
Does gambling sometimes cause you problems?	No/Yes	Yes
Do you want help with your gambling?	No/Yes, but not today/Yes	Yes
Over the last 2 weeks, how often have you been bothered by having little interest or pleasure in doing things?	Not at all Several days More than half the days Nearly every day	More than half the days/Nearly every day

(continued)

Table 31.1 Case Finding and Help Assessment Tool (CHAT) (continued)

Please choose the answer that most correctly applies to you

CHAT Questions	Response Options	Positive
Over the last 2 weeks, how often have you been bothered by feeling down, depressed, or hopeless?	Not at all Several days More than half the days Nearly every day	More than half the days/Nearly every day
Do you want help with this?	No/Yes, but not today/Yes	Yes, but not today/Yes
Over the last 2 weeks have you been worrying a lot about everyday problems?	No/Yes	Yes
Do you want help with your anxiety or worrying?	No/Yes, but not today/Yes	Yes, but not today/Yes
Is there anyone in your life of whom you are afraid or who hurts you in any way?	No/Yes	Yes
Is there anyone in your life who controls you and prevents you doing what you want?	No/Yes	Yes
Do you want help with any abuse or violence that you are experiencing?	No/Yes, but not today/Yes	Yes, but not today/Yes
Is controlling your anger sometimes a problem for you?	No/Yes	Yes
Do you want help with controlling your anger?	No/Yes, but not today/Yes	Yes, but not today/Yes
As a rule, do you do less than 30 minutes of moderate or vigorous exercise (such as walking or a sport) on 5 days of the week?	No/Yes	No
Do you want help with getting more exercise?	No/Yes, but not today/Yes	Yes, but not today/Yes

Responses

Any positive to a question warrants a further enquiry or assessment for that health issue.

For example:

- for a positive relating to alcohol we recommend use of the scored tool AUDIT (Alcohol Use Disorders Identification Test)
- for a positive relating to depression we recommend use of the scored tool PHQ-9 (Primary Health Questionnaire-depression)
- for a positive relating to anxiety we recommend use of the scored tool GAD-7 (General Anxiety Disorder-7)

Source: © Department of General Practice & Primary Health Care, The University of Auckland

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in a number of general practice settings and in a range of populations in New Zealand. There, the CHAT has been included as a recommended screening and case-finding tool in the Best Practice Guideline (BPAC, 2009). The CHAT has also been provided in the Australian government's Department of Health and Aging Risk Factor Resource Kit for use in general practice; in the development of the Canadian HealthCheckPlus, a real-time Internet-based health screening resource; and in the strategy to integrate alcohol screening into family medicine

in Missouri, in the United States (J. Walker; personal communication).

In most cases, the items were extracted from existing brief screens that had previously been independently validated in primary care settings. Overall, there was positive feedback from patients, with comments that the CHAT raised their awareness and was nonthreatening, brief, and clear. General practitioners also reacted positively, with over 90% saying that they would use it in their practice. Acceptability appeared high, with just

0.4% of patients objecting to the question on abuse, compared with 15% or more objecting to such questions found in other research. Of those who self-identified gambling problems, half wanted help from the practitioner the same day, while the remainder wanted help at another time.

An Australian Primary Health Strategy

The representative body for general practice in the state of South Australia, General Practice SA Inc., developed a resource kit for members to intervene in problem gambling issues with their patients (GSA, 2009). The kit allows the members, including GPs, practice nurses, and mental health clinicians, to screen for problem gambling. It then gives them the option of either treating the condition within the primary practice or referring the patient to a specialist who treats problem gambling. A poster is provided for the practice waiting room to both legitimize the topic for patients and to encourage patient inquiry. The members of General Practice SA have learned that statistically, problem gambling in Australia exceeds stroke and coronary heart disease combined and is comparable in prevalence to type 2 diabetes. A series of resources are provided to primary care health professionals, including a manual of information about problem gambling in a Frequently Asked Questions format (e.g., "Who is at risk?") and a range of brief documents describing the etiology of problem gambling, reasons to screen, assessment and treatment options, and information about legal options for gamblers and their families. A brief summary of the various manuals is provided for utility, a referral pathway or intervention algorithm, and a poster for the patients' waiting room with the statement "Talk to us. We can help you deal with gambling problems: if you, or someone you care about, have a gambling problem, take the first step and talk to your GP, Nurse or Health Worker."

There is another incentive for the physician in that, in addition to the consultation fee, a further payment is generated by first identifying the gambling problem and then completing a Mental Care Plan (Medicare Item No. 2710) before referring the patient to a mental health clinician. The clinician may be employed by the general practice; therefore, training these clinicians (who are also targeted by the resource kit) is incentivized. These clinicians may also establish their own cooperative arrangements with specialist problem gambling services. The members of these services, even if they are unlikely to be registered medicare providers, will receive the client referral.

WHO TO SCREEN?

Patients who should be screened include those who raise the topic following the poster's suggestion, who present with any of a range of at-risk conditions, or who belong to one of the following at-risk categories:

1. Patients presenting with symptoms of problem gambling described in the kit
2. Those with a severe mental health disorder, as identified by an appropriate score on the Kessler Psychological Distress Scale K10, a measure of stress level (Kessler et al., 2003)
3. Members of culturally and linguistically diverse communities
4. Indigenous people
5. Employees of gambling establishments
6. Women experiencing grief, trauma, or loneliness who also present with depression or anxiety
7. Young males who, in the absence of substance abuse issues, present with stress, anxiety, and financial problems
8. Middle-aged men with substance abuse issues

The primary care health professional is requested to ask the following questions (PGRTC questions): "Have you ever had an issue with gambling?" or "Has anyone in your family ever had an issue with gambling?"

ASSESSMENT

Patients who respond yes to the first question (their own gambling is causing problems) are either offered an assessment by the primary health care professional or referred to a specialist problem gambling service or mental health program provider selected from the regional list provided in the kit. The assessment can then be completed either by the patient, using the EIGHT screen, or by the health professional using the PGSI.

TREATMENT

Following assessment of the patient's gambling by the health professional or the external agency, the patient is offered a choice of treatment options:

- Treatment within the practice, using recognized psychotherapy such as motivational interviewing or cognitive behavioral therapy (CBT), together with strategies such as self-exclusion from gambling venues and, if appropriate, a Protection Orders Scheme for family members affected by a member's gambling.

- Sharing treatment with the gambling treatment service or mental health program; this may have particular relevance when the patient is also affected by coexisting health issues that may or may not abate as a result of ending the problem gambling behavior.
- External treatment only by referral to a problem gambling treatment provider or a mental health program.

The kit includes a referral note resource in which health professionals can provide details of their primary health care practice, together with other information that may impact the treatment. Usually, the preferred option is that the problem gambling treatment specialist provides the assessment and the therapy. If the practitioner is confident, then this may be provided within the practice.

To date, there has been no formal review of the use of the kit, with GPs indicating willingness to screen and refer rather than assess and treat. There has been less enthusiasm among GPs participating in problem gambling training, possibly due to the competing need to deal with other health issues. However, there is a possibility that mental health clinicians in each practice, or practice nurses (in 70% of the SA practices) or their practice managers, may be willing to consider this role. The project did not provide for this option, but it may be possible in the future (J. Walker; personal communication, July 21, 2009).

This strategy is also being developed in another Australian state, Victoria, where the Melbourne Division of General Practice is building on the SA problem gambling intervention kit (J. Walker; personal communication, July 20, 2009).

This approach will rely on the GP's recognition of the symptoms and his or her willingness to introduce the topic into the consultation. An alternative approach is that those with more time available, such as practice nurses, mental health clinicians in primary care settings, or other primary care practitioners, will be given the responsibility for assessment and treatment following appropriate training. Of particular interest in demonstrating the utility of this strategy will be the extent to which patients presenting with the identified at-risk symptoms or those belonging to the at-risk populations are invited to respond to the brief screen, as well as their responses and the outcomes that follow. An additional screen that may be offered to patients who agree to help with assessment could be the Concerned Others Gambling Screen (COGS; Sullivan et al., 2007),

which can identify effects of others' problem gambling on family members and offer various forms of help for the patient to choose from (Sullivan et al., 2007; Sullivan et al., 2006).

Interventions Following Identification of Problem Gambling

Few problem gamblers seek treatment (Slutske, 2006). In the absence of stronger evidence for drug intervention, the PGRTC recommends that those patients identified as problem gamblers be referred to specialist problem gambling services.

However, brief interventions for problem gambling for those who do not wish to participate in more intensive treatment, or for practitioners with limited time to offer, have been shown to be effective (Petry et al., 2008). A 10-minute advice session with a therapist in which gambling problems, risks, and ways to avoid risky situations are discussed was found to be as effective as longer sessions, and improvements persisted at the 9-month follow-up. This supports the effectiveness of brief interventions within the time constraints of medical settings.

Unwin and colleagues (2000) stated that family physicians should have heightened awareness of the impact of problem gambling, and of screening and treatment options. They recommended screening patients for problem gambling who presented with depressive or alcohol problems and addressing the risk of suicide. Family support was sought to help the patient follow the treatment recommendations, and the gambler and family were referred to Gamblers Anonymous and Gam-Anon. Specific therapy involved pharmacological treatment of coexisting disorders, such as depression, and therapies such as behavioral therapy, cognitive therapy, and CBT, or referral to specialist problem gambling treatment services. The stated low adherence to advice from referral physicians suggested that the family physician who identifies the gambling and coexisting health issues may often be required to address the gambling problems (Sullivan et al., 2007).

In another study, following brief training in screening and provided with a manual with information, referral resources, and screening and other brief interventions based on motivational interviewing strategies (Sullivan, 2003; Sullivan et al., 2006), GPs screened patients to identify those affected by their own or a family member's gambling. Patients were receptive to inquiries about their gambling, while GPs were surprised at the numbers of patients and their families affected by gambling problems. Although most GPs referred these patients to

specialist problem gambling treatment services, they considered their interventions to be effective. The greatest barrier identified was the time required to address both the gambling and the coexisting depression, which was also screened for.

MEDICATION AND PROBLEM GAMBLING

A number of pharmacological interventions have been studied as a treatment for pathological gambling. Serotonin reuptake inhibitors, such as clomipramine, citalopram, and fluvoxamine, which are effective in treating obsessive-compulsive disorder, have been found to have only modest effects on pathological gambling. A small number of studies of mood stabilizers (lithium and carbamazepine) in problem gambling have indicated moderately positive outcomes, while findings of recent studies of opioid antagonists (naltrexone) have been mixed (Grant et al., 2008a, 2008b; Toneatto et al., 2009). N-acetylcysteine has also been considered a possible treatment for reward-seeking behaviors such as problem gambling (Grant et al., 2007). A meta-analysis of studies up to 2006 found that pharmacological interventions for pathological gambling were more effective than placebos, although there were no differences in effectiveness among the three main classes of these drugs (antidepressants, opiate antagonists, and mood stabilizers; Pallesen et al., 2007). However, studies have shown that nonpharmacological treatments have larger overall effects than pharmacological treatments (Leung & Cottler, 2009).

With a high prevalence of mood disorders, anxiety disorders, and drug misuse in those affected by gambling, it is possible that these coexisting problems may be promoting the development, maintenance, and relapse of problem gambling.

In the absence of a recognized medication for problem gambling, the medical focus will be on the treatment of coexisting conditions. If these conditions are treated with established medical regimens, this may also have a positive effect on the gambling.

REFERRAL

The role of the GP in referral, particularly in mental health issues, has been to assess, educate the patient about the condition, provide effective referral, and monitor the patient's long-term progress (Blashki et al., 2003a). Referral to specialist services has often been the preferred response of GPs (Sullivan et al., 2006). It is the recommended response in the South Australian GP project unless a specialist is available for both assessment and therapy (GSA, 2009a).

The development of specialist treatment settings for problem gambling, with qualified clinicians, professional competency standards, evidence-based therapies, and ready accessibility, has helped address the reluctance that GPs may have had concerning the referral of their patients or shared-care arrangements being established for their patients. In many cases, specialist problem gambling treatment providers will establish on-site arrangements with health centers to facilitate the referral and shared-care arrangement.

Psychotherapy

Psychological treatment for patients in general practice can be available from GPs and other primary care health professionals who have both the time and training to deliver it. In this setting, approaches for mental health issues include supportive problem solving, when the problem is able to be solved, or CBT when the problem is due to a distorted perception or belief (Mynors-Wallis et al., 2000). Often a Socratic rather than an advice role may initially be a different expectation for both the primary care provider and the patient, so as to draw the solution out of the patient in a more psychological treatment approach (Blashki et al., 2003b). Limited and specific CBT, often termed *cognitive behavioral strategies*, that enable the primary care provider to address unrealistic perceptions and negative patterns of thinking can be provided without intensive training (Blashki et al., 2003c; Nathan & Gorman, 1998).

For many GPs, time constraints are a barrier to psychotherapy. In primary care practices the primary care team is generally led by the GP, with expertise in interpreting patients' health needs, planning their care, and referring them to other health providers. Such providers could be practice nurses within the health center, generalist mental health clinicians within the center, or specialist mental health clinicians externally based but also possibly integrated within the center (Gilmer et al., 2009). In the United Kingdom, nurses, although usually employed by GPs, are now forming nurse teams that are aligned with and located within the general practice, although not employed by it. They may have specific roles, including screening and chronic disease management (Hoare et al., 2008). Specialist training for nurses employed as behavioral psychotherapists has been available for over 30 years in the United Kingdom, and nurses who have received appropriate training have been perceived as suitable primary care practitioners to treat patients with gambling problems (Tolchard & Battersby, 2001).

Brief intervention by mid-level health professionals, usually nurses, has often been used in primary care for hazardous and harmful alcohol use (Babor et al., 2005; Grucza et al., 2008; Peltzer et al., 2006, 2008). Another possibility is that community pharmacists can provide brief interventions (in alcohol abuse) if appropriate training is available (Sheridan et al., 2008).

Researchers dealing with addictions in the United Kingdom suggest that persons with severe problem gambling should be referred to specialist problem gambling treatment services. Those with less severe problems could be treated within the primary care practice, perhaps by other health care professionals such as practice nurses (McCambridge & Cunningham, 2007).

Integration of Mental Health and Primary Care Services

Primary care settings are well suited to identify and intervene in mental health issues (MaGPIe-Research-Group, 2003). However, there are problems when people receive integrated care from both mental health specialists and general medical care providers. Often there is insufficient evidence of positive outcomes, particularly with alcohol abuse behavioral programs. In addition, the outcomes of therapy for depression and anxiety disorder involving integrated approaches generally appear to weaken over time (Butler et al., 2008). Examples of the integration of psychology and medicine, particularly in primary care, are relatively few (Kessler & Cubic, 2009). Primary care settings provide the ability to identify and address other health issues that commonly coexist with the target mental health issue and to monitor the patient's recovery over time. However, the low prevalence of many of the specified ICDs will continue to mitigate against their identification or their inclusion in regular screening, even if strategies to integrate their treatment with specialist services are established.

The British Medical Association has recommended that the dedicated, publicly funded national health services (NHS) be expanded throughout the United Kingdom. Researchers concerned with the secondary prevention of addictions are uncertain about the extent to which GPs should contribute to this effort, and they note that it takes GPs into new areas that may not be embraced by the majority of them (McCambridge & Cunningham, 2007). However, they have stated that gambling problems may contribute significantly to the issues dealt with by GPs and may, if evidence is found to support this

role, encourage the identification of problem gambling as good clinical practice (Goodyear-Smith et al., 2006; Pasternak & Fleming, 1999; Potenza et al., 2002).

A Model for Composite Problem Gambling Intervention in Primary Care

The South Australian model provides a strategy with many of the features necessary to address problem gambling in primary care settings. It addresses many of the barriers found in alcohol interventions, such as brevity of screening, funding for additional time, and tailor-made resources that can be utilized by both GPs and other health professionals in the primary care setting. The one factor that may continue to provide a challenge is the ability to identify symptoms. There is a broad range of symptoms and heterogeneous populations that are identified as at risk; further follow-up on this strategy and the proposed extension to the state of Victoria may provide evidence concerning its use. An alternative approach that could be integrated into this model is the use of a composite screener, such as the CHAT, for all clients periodically. The model could therefore provide:

1. Education on the use of the screen by all health professionals, including each of the items, their frequent interrelationship, appropriate interventions, and referral resources, with such training supported by professional organizations through credits toward continuing training requirements
2. Strategies for regular screening, provided either by paper or electronically, and a stepped process for addressing any positive responses, including those of the GP, nurse, mental health clinician, or specialist external provider sited within the primary care service who has time to provide the necessary interventions (e.g., positive responses may be provided by the GP and other center health professionals)
3. Development of brief intervention training, including resources such as homework manuals that can be given to the patient and supported by primary care health providers, to supplement any pharmacological intervention
4. Financial support for the additional time required to address problem gambling issues, which will include psychological interventions and may include more than one health professional at the center
5. Ongoing monitoring and review of the condition or conditions in subsequent consultations

Primary care physicians have been targeted as being in a good position to identify ICDs and raise awareness about the importance and effectiveness of treatment (Mak, 2004). For many, the next step will be referral to an external specialist mental health service, with the possibility of retaining a liaison role with that service, addressing coexisting general medical conditions, and monitoring progress during later consultations for other issues. Recently, there has been a movement to provide mental health specialists and other primary care providers with additional time and specialist training within health centers. The provision of options to treat ICDs such as problem gambling within such centers, the availability of kits and training to optimize such screening, assessment, and treatment of this condition may set an example for the treatment of other ICDs within a primary care setting. The present high public accessibility, motivation to address common coexisting conditions, ability to monitor and follow up, and increased research on ICDs focusing on evidence-based interventions may act to optimize intervention into ICDs by well-placed health providers in the future.

Future Directions

- Impulse control disorders usually coexist with many other, often more recognizable and better-understood mental health disorders such as depression. Whether this association is causative, unidirectional (and in which direction), or bidirectional is not well understood. Further knowledge may increase the motivation to screen for ICDs and to treat or refer patients.
- Describing some ICDs, such as pathological gambling, pyromania, trichotillomania, and kleptomania, as addictions, if similarities can be drawn, may provide the advantage of belonging to a recognized group, with common theories concerning etiology, intervention strategies, and biological, psychological, and social aspects.
- Identifying effective pharmacological interventions that could be used in conjunction with psychotherapies would assist GPs in screening and providing other brief interventions.
- Researching the effectiveness and efficiency of specific screens versus composite screens to identify and provide interventions for ICDs, such as the PGRTC and CHAT screens, as well as support resources, may optimize their use.
- Identifying other primary care health practitioners in health organizations who may have a role in ICD intervention and training opportunities will enable them to intervene.

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Impulse Control Disorders in Neurological Settings

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Abstract

This chapter will argue that impulse control disorders, including addiction, are the product of an imbalance between two separate but interacting neural systems: (1) an impulsive amygdala-striatum-dependent neural system that promotes automatic and habitual behaviors and (2) a reflective prefrontal cortex-dependent neural system for decision making, forecasting the future consequences of a behavior, and inhibitory control. The reflective system controls the impulsive system via several mechanisms. However, this control is not absolute; hyperactivity within the impulsive system can override the reflective system. While most prior research has focused on the impulsive system (especially the ventral striatum and its mesolimbic dopamine projection) in promoting the motivation and drive to seek drugs, or on the reflective system (prefrontal cortex) and its mechanisms for decision making and impulse control, more recent evidence suggests that a largely overlooked structure, namely the insula, plays a key role in maintaining poor impulse control, including addiction. This review highlights the potential functional role the insula plays in addiction. We propose that the insula translates bottom-up, interoceptive signals into what subjectively may be experienced as an urge or craving, which in turn potentiates the activity of the impulsive system and/or weakens or hijacks the goal-driven cognitive resources that are needed for the normal operation of the reflective system.

Keywords: impulse control, executive function, inhibitory control, decision making

Introduction

Impulsivity may be defined as “a predisposition toward rapid, unplanned reactions to internal or external stimuli with diminished regard to the negative consequences of these reactions to the impulsive individual or to others” (Chamberlain & Sahakian, 2007; Potenza, 2007, p. 5). In contrast, *compulsivity* represents a tendency to perform unpleasantly repetitive acts in a habitual or stereotyped fashion in order to prevent perceived negative consequences, leading to functional impairment (Chamberlain & Sahakian, 2007; Fineberg et al., 2010). These two constructs may be viewed as diametrically opposed, or alternatively, as similar, in that each implies a dysfunction of impulse control. Each potentially involves alteration within a wide range of neural processes includ-

ing attention, perception, and coordination of motor or cognitive responses (Fineberg et al., 2010). While many studies have addressed these issues in psychiatric populations, relatively little attention has been paid to these disorders in neurological settings. This chapter outlines the various forms of brain damage that are associated with poor decision making and impulse control disorders.

The initial foray into the neuroscience of decision making came from the systemic examinations of changes in the real-life behavior of patients with localized brain damage produced by accidents, strokes, or neurological diseases. Work with lesion patients has not only identified brain areas that are critical for adaptive decision making, but has also inspired the development of conceptual neural

models for explaining how humans make choices (Bechara & Damasio, 2005; Damasio, 1994; Shallice, 1993; Stuss & Anderson, 2004). Among the most influential work using the brain lesion method has been the work on the decision-making capabilities of patients who have suffered injury to the ventromedial prefrontal cortex region of their brain. After their brain injury, these patients tend to recover with normal intelligence, memory, speech, sensation, and movement. However, in striking contrast to this remarkable recovery, their emotion and affect, their social behavior and personality change completely. As a result, these patients begin to have difficulties planning their workday and choosing friends, partners, and activities. The actions they elect to pursue often lead to losses of diverse order, such as financial losses, losses in social standing, and losses of family and friends. The choices they make are no longer advantageous—the patients often decide against their best interests and fail to learn from previous mistakes—and are remarkably different from the kinds of choices they were known to make before their brain damage. These observations—that is, normal intellect, abnormalities in decision making, emotion, and feeling in these patients—led Antonio R. Damasio to propose what has become an influential neural theory of decision making, the Somatic Marker Hypothesis. The central feature of this theory is that emotion-related signals (somatic markers) assist cognitive processes in implementing decisions. A further aspect of this theory is that these somatic markers can be nonconscious: they can bias behavior even when a person is not really aware of them (Damasio, 1994).

At the time, the decision-making deficit seen in these neurological patients was puzzling because their poor judgment and failure to learn from repeated mistakes were so obvious in their real lives, but there was no laboratory probe to detect and measure this impairment. This challenge was overcome, however, after the development of what became known as the Iowa Gambling Task (IGT; Bechara et al., 1994, 2000). The development of this task enabled researchers, for the first time, to detect these patients' elusive impairment in the laboratory and investigate its possible causes. This line work drew attention to the potential value of studying the neural basis of decision making in the laboratory through the use of structured decision-making tasks, tasks that involve choices that mimic real-life situations in the way they factor uncertainty, reward, and punishment. (see Bechara & Damasio, 2005, for review). However, this line of work, along with so many other

lines of studies on motivation and behavioral control, now offers new insights into the neural mechanisms underlying so many conditions associated with poor decision making and impulse control.

Outline of a Neurological Model

Impulse control disorders may be explained in terms of abnormal activity in any one, or a combination, of three key neural systems shown to play important roles in (1) implicit associations and the promotion of impulsive, automatic, habitual behaviors (Everitt & Robbins, 2005; Everitt et al., 1999); (2) decision making and impulse control, as well as the ability to resist stimuli that are rewarding in the short term but lead to negative consequences in the long term (Bechara, 2005); and (3) translating homeostatic and interoceptive signals triggered by deprivation states, or by exposure to reward cues, into craving and what may become subjectively experienced as an intense “urge” (Naqvi & Bechara, 2009).

This neurological model resonates well with dual-process models of decision making, cognition, and associative memory, which have gained substantial momentum in behavioral research (Kahneman, 2003; Rhodes et al., 1997). In characterizing the distinctions between two cognitive systems (Kahneman, 2003), a Nobel Prize recipient, stated that “the operations of System 1 are typically fast, automatic, effortless, associative, implicit (not available to introspection), and often emotionally charged; they are also governed by habit and are therefore difficult to control or modify. Operations of System 2 are slower, serial, effortful, more likely to be consciously monitored and deliberately controlled; they are also relatively flexible and potentially rule governed” (p. 698). According to Kahneman, the implicit or automatic cognitive system (System 1) is the system governing the majority of human decision making, whereas System 2 monitors the operations of System 1. This perspective of relatively automatic processes being a sort of “default” mode guiding behavior, unless overridden by deliberate cognitive processes, has been expressed in social psychological theories (Devine, 2001; Fazio, 1990) and memory research (Nelson et al., 1998) for over a decade. The distinction between automatic and controlled processes (and similar distinctions) is reinforced further through several independent lines of research in neuroscience (Bechara et al., 2006; Chein & Schneider, 2005; Levy et al., 2004; Yin & Knowlton, 2006a)

Recent dual-process models have described addictive behaviors as a dynamic interplay between

two systems: (1) a relatively implicit or automatic appetitive system, which we called the *impulsive system*, and (2) an executive control/inhibitory system, which we called the *reflective system* (Bechara, 2005). A key neural region that supports the implicit (impulsive) system is the amygdala-striatal (dopamine-dependent) neural system, which is critical for the incentive motivational effects of a variety of nonnatural rewards (e.g., psychoactive drugs) and natural rewards (e.g., food; Balleine & Dickinson, 2000; Dagher, 2009; Dagher & Robbins, 2009; Di Chiara et al., 1999; Everitt et al., 1999; Koob & Le Moal, 2001; Robbins et al., 1989; Robinson & Berridge, 1993; Stewart et al., 1984; Wise & Rompre, 1989). A critical neural region in the reflective system is the ventromedial prefrontal cortex region (which we have considered as inclusive of the medial orbitofrontal cortex; Bechara et al., 2000). However, other neural components, including the dorsolateral prefrontal cortex (for working memory) and the cingulate cortex, are also part of this neural circuitry and are essential for the normal operation of the ventromedial prefrontal cortex (Bechara, 2004b).

However, more recent evidence suggests that there is a third neural system mediated through the insular cortex, which plays a key role in translating interoceptive signals into what one subjectively experiences as a feeling of desire, anticipation, or urge (Naqvi & Bechara, 2009; Naqvi et al., 2007). We have argued that activity of the insular cortex elicited by homeostatic imbalance and deprivation states, or by reward cues, serves to sensitize the motivational circuits that propel individuals toward reward (the habit or impulsive system) and to “hijack” the prefrontal system, preventing it from using the cognitive resources necessary to exert self-control and the willpower to resist reward. Thus, in this neural model, insular cortex activity elicited by craving and urge is thought to influence the dual-process systems by (1) intensifying activity of the habit (impulsive) system and (2) disabling the inhibitory control and decision-making functions of the prefrontal (reflective) system.

As a result of continued use of a given substance, automatic associative processes become increasingly strong, overriding or overwhelming control processes, with the addictive behavior coming more under stimulus control and less under voluntary control (Bechara et al., 2006; Stacy et al., 2004; Wiers et al., 2007a). The imbalance of these systems can lead to impaired decision making, which is highly relevant, not only to substance abuse, but to

natural reward as well. Research from multiple perspectives suggests that automatic processes linked to reward cues can be implicitly activated, but these activated associations may be most readily translated into behavior among those without sufficient protection in executive control functions. That is, individual differences in certain specific executive functions, like affective decision making and impulse control, may modulate the effects of activated implicit associations on behavior (Kane & Engle, 2002; Royall et al., 2002). Reductions in certain executive functions may essentially make automatically activated associations more powerful in their effects on behavior—increasing their “free reign” or their status as the “default” system (Kahneman, 2003). These reductions in executive functions, compounded by increased automatic associations, are further exacerbated by states of deprivation associated with craving and urge and mediated through the insular cortex. Given individual variations in decision-making ability or inhibition based on control processes supported by the prefrontal cortex that regulate behavior (Braver & Ruge, 2006), there is clear potential for some functions to modify the influence of associative processes on behavior. This type of interaction, underscoring the synergistic effects of various automatic and control processes, has been supported in behavioral levels of analysis in diverse areas including dietary behavior (Hofmann et al., 2007), problem solving (De Neys, 2006), and addiction (Finn & Hall, 2004; Grenard et al., 2008; Houben & Wiers, 2009; Thush et al., 2008).

Neural Systems That Energize, Motivate, and Drive Behavior

The amygdala-striatal (dopamine-dependent) neural system has been the subject of over 30 years of research in the literature on the neurobiology of addiction. This is the neural system, which is critical for the incentive motivational effects of a variety of nonnatural rewards (e.g., psychoactive drugs) and natural rewards (e.g., food; Balleine & Dickinson, 2000; Di Chiara et al., 1999; Everitt et al., 1999; Koob & Le Moal, 2001; Robbins et al., 1989; Robinson & Berridge, 1993; Stewart et al., 1984; Wise, 1989). This is also the neural system that has been argued to be critical for learning implicit associations and responsible for the transfer of reward seeking from controlled to automatic and habitual behaviors (Everitt et al., 1999; Everitt & Robbins, 2005). In our own research, we have referred to this neural system as the impulsive system, and we have shown

that it becomes hyperactive and begins to exaggerate the incentive value of rewards in individuals with substance abuse problems (Shiv et al., 2005).

Several lines of basic research on drug addiction led to the same conclusion that continued drug use results in the strengthening of motivation-relevant associative memories, which promote continued use, and an implicit or relatively spontaneous process begins to govern behavior (e.g., Smith & DeCoster, 2000; Stacy, 1997; Wiers & Stacy, 2006). For instance, Everitt, Robbins, and colleagues argued that dopaminergic activity in the nucleus accumbens, and in some anatomically connected structures, reinforces the repetition of behaviors and supports the encoding and processing of proximal stimuli associated with the rewarding experience (e.g., Cardinal & Everitt, 2004; Everitt & Robbins, 2005). Neutral stimuli associated with appetitive behaviors such as drug use come to represent and cue the behavior. As cue-behavior-outcome associations are strengthened, patterns of associations signal and drive behavior without the necessary involvement of reflective processes (e.g., White, 1996). Cues can then trigger an essentially automatic pattern of activation in memory that can be described in various neural network or connectionist models (e.g., Hopfield & Tank, 1986; Queller & Smith, 2002). A similar conclusion was obtained by the incentive sensitization theory (Robinson & Berridge, 1993, 2003; Wyvill & Berridge, 2001). This theory suggests that, through repetition of rewarding appetitive experiences, neural sensitization of the mesocorticolimbic circuitry occurs and mediates motivational processes by attributing incentive salience to reward-related stimuli (e.g., drug-related cues). Cues associated with reward are then able to elicit "wanting" for a specific reward (Robinson & Berridge, 1993, 2003; Wyvill & Berridge, 2001). Another line of research has linked implicit learning and the establishment of automatic stimulus-response connections to this same striatal neural system and its link to the extensively studied mesolimbic dopamine system (Knowlton et al., 1996; Kringelbach, 2005; Schoenbaum et al., 2006).

Overall, research on the neurobiology of drug addiction shows that increased mesolimbic dopamine activity reinforces the repetition of behaviors, influencing learning, attentional processes, and the strengthening of associations of reinforcing effects (see Di Chiara, 2002). Once a strong habit is formed, cues elicit the habit regardless of anticipated outcomes (Wood & Neal, 2007; Yin & Knowlton,

2006a, 2006b). Habits become automatic and difficult to change. Another pivotal feature of habit systems is that participants do not necessarily know what triggers their habits.

One of the most commonly used indirect tests of association in memory is the implicit association test (IAT). Hundreds of studies have been published using the IAT to evaluate a variety of attitude associations, for example, toward gender, age, and racial categories, among numerous others (e.g., Greenwald et al., 1998; Jelenec & Steffens, 2002; Milne & Grafman, 2001; Ziegert & Hanges, 2005). The IAT is a concept categorization task that evaluates the relative strength of associations of contrasted categories with contrasted attribute categories through rate of processing. The basic assumption is that past learning experiences can be represented by the facilitation of information processing of associated concepts as measured by the rate of processing of different combinations of stimuli. That is, individuals react faster when categorizing strongly associated concepts that share a response key and slower when categorizing concepts that are less likely to be associated and share a response key (see Greenwald et al., 1998).

The IAT has been found to effectively differentiate substance users from nonsubstance users in studies that have adapted the IAT to evaluate implicit associations in alcohol (e.g., De Houwer et al., 2004; Jajodia & Earleywine, 2003; Thush et al., 2007, 2008; Wiers et al., 2002, 2005), marijuana (Ames et al., 2007; Field et al., 2004), tobacco (Swanson et al., 2001), and cocaine use (Wiers et al., 2007b). Food-related indirect tests of association have also been found to differentiate obese from nonobese youth (Craeynest et al., 2005, 2006). Craeynest et al. (2006) reported that on a food-related IAT, nonobese individuals identified more strongly with nonfat foods, while obese individuals identified equally strongly with both fat and nonfat foods. On the Extrinsic Affective Simon Task, a variant of the IAT, Craeynest et al. (2005) also were able to differentiate obese adolescents from their lean peers, with obese youth having stronger positive implicit attitudes toward both unhealthy and healthy foods.

Neural Systems That Inhibit or Control Behavior

While the habit (or impulsive) system, which is key to generating at least the "wanting" component to seek reward (Robinson & Berridge, 2003), may explain one important aspect of the behaviors associated with approach behaviors, it is clear that

it does not explain how one controls his or her behavior. Therefore, the *executive control* system, which depends primarily on the functions of the prefrontal cortex, is necessary to control these more basic impulses and allow more flexible pursuit of long-term goals (Fellows, 2004; Wheeler & Fellows, 2008). This is the system engaged when you stop yourself from reaching for that second dessert today, because you want to look good in your swimsuit next summer. This is also the system that traditional public health messages are trying to influence. Using tasks that are thought to measure the strength of this executive control system, which basically enables one to exert willpower and self-control and delay gratification for better long-term goals, there is evidence that people with impulse control disorders do express a basic weakness in these executive control mechanisms (Fineberg et al., 2010).

In our own research, we have referred to this neural system as the *reflective* neural system (Bechara, 2005). A critical neural region in the reflective system is the ventromedial prefrontal cortex region (which we have considered as inclusive of the medial orbitofrontal cortex; Bechara et al., 2000). However, other neural components, including the dorsolateral prefrontal cortex (for working memory) and the cingulate cortex are also part of this neural circuitry, and are essential for the normal operation of the ventromedial prefrontal cortex (Bechara, 2004b). Adequate affective decision making reflects an integration of cognitive and affective systems, as well as the ability to more optimally weigh short-term gains against long-term losses or probable outcomes of an action. For example, drug use known to have short-term reinforcing effects (but long-term negative consequences) should be less likely or problematic for individuals scoring higher on tasks that assess this ability. The functional distinction of affective decision-making processes as assessed with the IGT comes from extensive clinical research with patient populations with damage in frontal lobe regions (see Bechara & Van Der Linden, 2005). Structural imaging studies link IGT performance to neural regions of the ventromedial prefrontal cortex (Anderson et al., 2000; Bechara et al., 1998). Affective decision making has been shown to be important in behavioral regulation across numerous studies and various populations (see Brand et al., 2006).

DISTINGUISHING DECISION MAKING FROM INHIBITORY CONTROL

While many researchers still group all mechanisms of decision making and inhibitory control under

one umbrella, the rubric of executive functions, we have argued that the two are separable neuropsychological mechanisms (Bechara & Van Der Linden, 2005). More specifically, we have argued that, within the reflective system, there is a distinction in functioning between (1) simple inhibitory and impulse control processes (some are mediated by the lateral orbitofrontal and inferior frontal gyrus regions, and some are mediated by the more posterior sectors of the medial prefrontal region, i.e., the anterior cingulate cortex, both dorsal and ventral) and (2) affective decision making (mediated by more anterior regions of the medial prefrontal cortex, including the frontal pole), which are highly relevant to behavioral control ability and to the decisions individuals make frequently on a daily basis (Bechara, 2005; Bechara & Van Der Linden, 2005). Both inhibitory/impulse control function and affective decision making are important specific aspects of higher order executive control functioning (Winstanley et al., 2006). Good inhibitory functioning reflects the ability to actively stop a prepotent behavioral response (e.g., drinking or eating in excess) after it has been triggered (Braver & Ruge, 2006; Logan et al., 1997)). Inhibitory processes are relevant primarily when there is a need for inhibition of a prepotent behavioral tendency or impulse, and such tendencies do not surface continuously but instead are activated primarily by antecedent cues (e.g., Wood & Neal, 2007). Inhibition, then, becomes most relevant in the face of these cues. Individuals with deficits or failures in these systems have a tendency to act more impulsively. Adequate affective decision making reflects an integration of cognitive and affective systems (hence, considered “hot”—emotionally-linked—cognition, and the ability to more optimally weigh short-term gains against long-term losses or probable outcomes of an action; Bechara, 2005; Bechara & Van Der Linden, 2005). The functional distinction between simple inhibitory/impulse control and affective decision-making processes, as assessed with the IGT, comes from extensive clinical research with patient populations with damage in frontal lobe regions (see Bechara & Van Der Linden, 2005) as well as imaging studies that delineate the likely neural basis of each of these functions (for review of inhibition, see Simmonds et al., 2008; for review of IGT regions, see Lawrence et al., 2009).

Besides this broad distinction between decision making and simple inhibition, we have proposed several different neural mechanisms of decision making and impulse control, which can be tapped

into by different sets of neuropsychological tests and linked to separate brain regions.

Decision Making

Impairments of emotion and social behavior are often observed after damage to the ventromedial (VM) region of the prefrontal cortex (Brodmann's areas [BA] 25, lower 24, 32, and the medial aspect of 11, 12, and 10). Previously well-adapted individuals become unable to observe social conventions and decide advantageously on personal matters. Their ability to express emotion and to experience feelings in appropriate social situations becomes compromised. Studies aimed at understanding the nature of these deficits revealed that impaired decision making is at the heart of the problem. Specifically, evidence suggests that the VM region serves as a link between (1) a certain category of event based on memory records in high order association cortices to (2) effector structures that produce an emotional response. During decision making, category events are brought to working memory, which includes several processes. However, maintaining an active representation of memory over a delay period involves the dorsolateral sector of the prefrontal cortex. Effector structures that mediate the emotional response are located in the brainstem. Thus, decision making is a complex process that depends on systems for memory and emotion. Damage to the systems that impact emotion and/or memory compromises the ability to make advantageous decisions. The VM region links these systems together; therefore, when these systems are damaged, there are many manifestations, including alterations of emotional experience and social functioning.

The paradigm most frequently used to assess decision making is the IGT (Dunn et al., 2006; Ernst et al., 2003; Fellows, 2004; Grant et al., 2000; Monterosso et al., 2001; Petry, 2001; Whitlow et al., 2004), which was initially developed to investigate the decision-making defects of neurological patients in real life. The IGT has been shown to tap into aspects of decision making that are influenced by affect and emotion (Bechara, 2003; Turnbull et al., 2005). Numerous studies across a wide range of populations demonstrating poor behavioral decisions (e.g., substance users, psychopathic offenders, pathological gamblers, and adolescents with externalizing behavior) have shown that the IGT detects decreased decision-making performance in comparison with that of nonproblematic control groups (for reviews, see Brand et al., 2006; Dunn et al., 2006). The generality of decision-making effects

detected with the IGT was demonstrated in two recent studies (Johnson et al., 2008; Xiao et al., 2008). Both studies suggest that IGT performance and affective decision-making ability are likely to predate problems with appetitive behaviors in some adolescents.

The prefrontal region is relatively large, and not all of its cortices mediate the same function. A deficit in decision making as measured by the IGT can arise from a dysfunction in the VM cortex, especially the more anterior/rostral sector of the VM region, that is, toward the frontal pole, or just anterior to Brodmann's area 25. When the damage extends to more posterior areas of the VM region (i.e., including the anterior cingulate) or to the lateral orbitofrontal area (inferior frontal gyrus), additional problems appear. These problems seem to reflect impairments in several mechanisms of impulse control or response inhibition.

This decision-making mechanism may be similar to the personality trait of *nonplanning impulsivity*, that is, living for the moment and disregard for the future (Patton et al., 1995) or the trait of *premeditation*, that is, the tendency to think and reflect on the consequences of an act before engaging in that act (Whiteside & Lynam, 2001). Preliminary evidence supports a link between premeditation and performance on the IGT (Zermatten et al., 2005). More studies are needed to explore other possible relationships between measures of decision making obtained by neuropsychological tasks and traits of impulsivity measured by personality tests.

Motor Impulse Control

Motor impulse control concerns the ability to deliberately suppress dominant, automatic, or prepotent responses (Friedman & Miyake, 2004). For instance, acting quickly without an intention to act reflects an instance of weakness in this mechanism. Poor performance on laboratory instruments such as the Stroop task, the go/no-go task, and the Hayling task (Burgess & Shallice, 1996) reflects deficits in this type of impulse control. A critical neural region for this mechanism of impulse control appears to be the more posterior area of the ventromedial prefrontal cortex region, which includes the anterior cingulate, and perhaps the basal forebrain, as patients with lesions in this area exhibit signs of disinhibition and poor impulse control (Bechara, 2004a). Indeed, lesion studies in humans suggest that impairments in reversal learning of previously rewarded responses are more closely associated with damage involving this region, which includes the

subgenual sector of the anterior cingulate (Owen et al., 1991; Rolls et al., 1994). In the laboratory, we found that these patients have difficulties with the delayed nonmatching to sample task. In this task, a sample stimulus appears (e.g., a red card) and disappears, and after a short delay, the sample card is presented again with another stimulus (e.g., a black card). The task is to remember the sample card and then inhibit responding to it, that is, select the non-matching (black) card (Bechara et al., 1998). In this task, patients with damage in this region quickly make the incorrect response, and a few milliseconds later realize that they made an error and declare: "Oh, I meant to pick the other card." It appears that these patients detect the error that they have made a few milliseconds too late. Other examples of tasks demonstrating signs of disinhibition in patients with this type of lesion include asking the patient to draw a circle. Once the patient draws the first one, he or she keeps on drawing more and more circles, as if unable to stop, until the examiner stops the patient (e.g., see Goldberg, 2001, for a nice description of these patients).

Disturbances in this mechanism may relate to the personality trait of *motor impulsivity*, that is, acting without thinking (Patton et al., 1995) or the trait of *urgency*, that is, the tendency to experience strong impulses, frequently under conditions of negative affect (Whiteside & Lynam, 2001). Although this relationship has intuitive appeal, empirical evidence for it has not yet been sought.

Attention/Perceptual Impulse Control

Attention/perceptual impulse control reflects the ability to resist the memory intrusion of information that was previously relevant but has since become irrelevant (Friedman & Miyake, 2004). In other words, it concerns the ability to inhibit irrelevant thoughts or memories. Difficulty inhibiting particular thoughts, shifting from one thought to another, or focusing on one particular thought reflect instances of weakness in this mechanism. This inhibition construct can be assessed by using tasks requiring task-set switching (such as the Wisconsin Card Sorting Test (WCST) or the Intra-dimensional/Extra-dimensional [ID-ED] Shift Paradigm). Critical neural regions for this mechanism appear to be the lateral orbitofrontal and dorsolateral (inferior frontal gyrus) regions of the prefrontal cortex; patients with damage in these areas make perseverative errors and have difficulty shifting attention (Bechara, 2004b).

Disturbances in this mechanism may relate to the construct of *cognitive impulsivity*, that is, making

up one's mind quickly or having a problem concentrating (Patton et al., 1995), or the personality trait of *perseverance*, that is, the ability to remain focused on a task that may be boring or difficult (Whiteside & Lynam, 2001). However, research is needed in order to provide empirical evidence for such a possible relationship.

It is very important to realize that although these different control mechanisms can be dissociated under experimental conditions, they are all interrelated and act together in a functioning brain. For example, when inhibiting a prepotent response, such as in the Stroop task, it is difficult to do this without engaging mechanisms of attention/perceptual control (or resistance to proactive interference). Therefore, it is very difficult to dissociate these mechanisms in normal individuals, but the combination of functional imaging and lesion studies suggests that these mechanisms are dissociable under appropriate conditions. Injuries or diseases that affect any of these mechanisms exert a devastating impact on the behavior of affected individuals. However, differentiating these various mechanisms of executive control functions is important because it helps explain, at times, contradictory results from studies that address the sensitivity of a variety of measures, from executive function tests to tests of frontal lobe dysfunction (e.g., see Demakis, 2004). We argue that this is because executive deficits are a multidimensional disability (Varney & Stewart, 2004); they are detected by different sets of neuropsychological measures, which tax relatively distinct functions that are subserved by relatively separate sectors of the prefrontal cortex.

Neural Systems That Intensify Motivation and Weaken Control of Behavior

Based on our research, we have argued that the process of decision making depends in many important ways on neural substrates that regulate homeostasis, motivation, emotion, and feeling (Bechara & Damasio, 2005). We have suggested that two broad conditions could lead to compromised decision making and poor ability to resist the temptation of a reward. One condition may involve a dysfunction in the prefrontal cortex system (the reflective system), which is critical for inhibitory and impulse control and affective decision making. Another condition may involve hyperactivity in the amygdala-striatal system, which is critical for promoting habitual and impulsive behaviors toward reward. However, based on more recent evidence (Naqvi & Bechara, 2009), a long-forgotten structure, the insular cortex, has

emerged as a neural structure that plays a key role in interoceptive representations generated from smoking cues, and we have speculated that this function extends to other biological urges, such as the hunger elicited by food cues. We have argued that activity of the insular cortex elicited by homeostatic imbalance and deprivation states, or by reward cues, serves to sensitize the motivational circuits that propel individuals toward reward (the habit or impulsive system) and that hijack the prefrontal system, preventing

it from using the cognitive resources necessary to exert self-control and the willpower to resist reward (Figure 32.1).

Indeed, it has been argued that the insular cortex (and probably the somatosensory cortex, especially SII) plays a key role in translating interoceptive signals into what one subjectively experiences as a feeling of desire, anticipation, or urge (Craig, 2009; Naqvi & Bechara, 2009). Recent evidence also shows that strokes that damage the insular

Neural structures involved in:

1. Appraisal of incentives or motivationally competent stimuli, e.g., drug cues.

2. Triggering (induction) of motivational state.

3. Execution of motivational state.

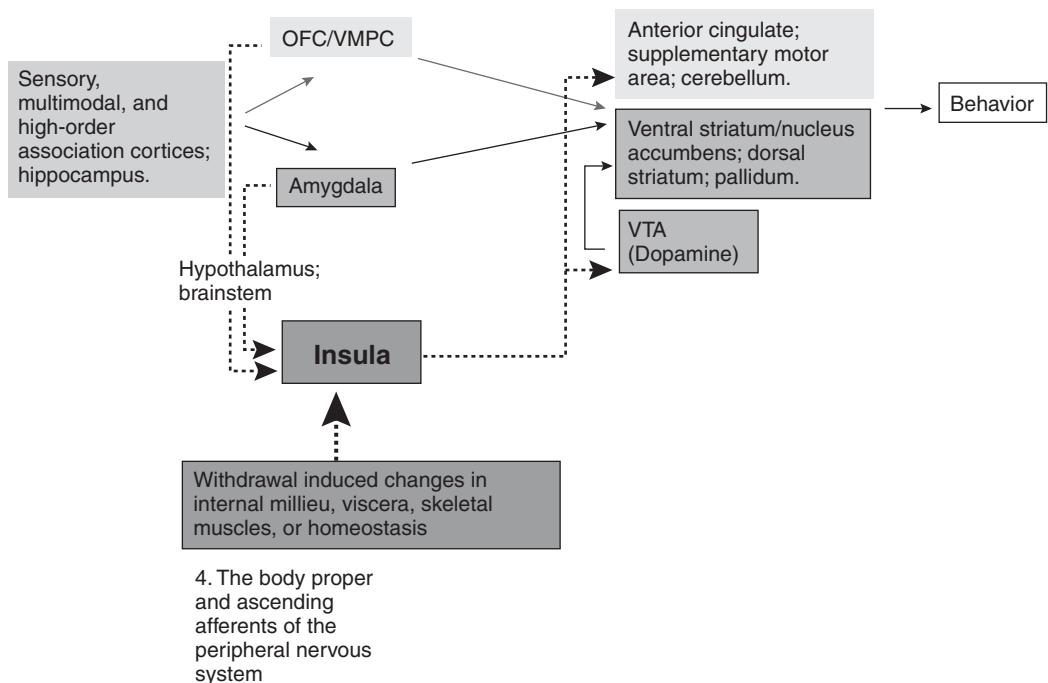


Fig. 32.1 A schematic neurological model illustrating a proposed functional role for three key neural systems in impulse control disorders, using addiction as one example: (1) The amygdala-striatal neural system, which we have termed the *impulsive system*, excites the traditional reward system involved in the execution of motivational states to seek drugs, such as the ventral striatum/nucleus accumbens and the mesolimbic dopamine system (highlighted in red). (2) The mesial orbitofrontal/ventromedial prefrontal cortex (OFC/VMPC) is a key structure in a neural system we have termed the *reflective system*, which forecasts the future consequences of a behavior such as seeking drugs. (3) The proposed functional role of the insula is highlighted in green. Incentive stimuli (e.g., drug cues) generate motivation in the animal (or human) and instigate approach responses in relation to themselves through the impulsive system. However, internal factors associated with deprivation states (such as withdrawal) are viewed as a “gate” that determines how effective the incentive input is in exciting the motivational circuits that “pull” and “steer” the animal (or human) toward the appropriate goal object. This process, we propose, is dependent on the insula. Feedback loops arising from the body, reflecting the status of the viscera and homeostasis, and mediated through the insula, will adjust the strength of the conflicting signals, thereby sensitizing the impulsive system and potentially overriding the inhibitory control of the reflective system. An additional possibility is that insula signals may subvert the decision-making processes of the reflective system into formulating plans for action to seek and procure drugs.

cortex tend to literally wipe out the urge to smoke in individuals previously addicted to cigarette smoking (Naqvi et al., 2007). These studies suggest that enhanced sensitivity in regions involved in the sensory and visceral processing of reward stimuli may make the stimulus more rewarding, and thus generate greater craving and the urge to approach the rewarding stimulus. In turn, this contributes to excessive use of a behavior, such as excessive food consumption. One proposed mechanism for the way this may take place is that activation of interoceptive representations through the insula can, on the one hand, sensitize the habit (impulsive) system. On the other hand, the insula activation may impact the functions of the prefrontal cortex, so that it can subvert attention, reasoning, planning, and decision-making processes to formulate plans for action to seek and procure food. Put differently, these interoceptive representations have the capacity to hijack the cognitive resources necessary for exerting inhibitory control to resist calorie-rich foods (Naqvi & Bechara, 2009).

Conclusion

We argue that impulse control disorders relate to an imbalance between two relatively separate but interacting neural systems: a hyperactive striatum-dependent neural system that promotes impulsive, automatic, and habitual behaviors and a hypoactive prefrontal cortex-dependent neural system that reinforces the ability to inhibit impulses and resist stimuli that are rewarding in the short term but lead to negative consequences in the long term. Within this system, we have proposed that there are several separate mechanisms of decision making and impulse control: (1) Decision making, as measured by the IGT, is a relatively more complex process involved in the control of several other cognitive, behavioral, and affective processes mediated by other regions of the brain. The more anterior/rostral sector of the ventromedial prefrontal cortex seems the most critical neural substrate subserving this mechanism. (2) Motor impulse control, as measured by a variety of tasks such as the Stroop, go/no-go, and learning reversal tasks, relates to mechanisms concerned with the control of behavioral or motor response processes. The anterior cingulate seems to be a critical neural substrate subserving this mechanism. (3) Attention/perceptual impulse control, as measured by the WCST, ID-ED shift, and other switching tasks, relates to mechanisms concerned with the control of cognitive processes (e.g., information held in working memory).

The lateral prefrontal region, especially the inferior frontal gyrus, seems to be the critical neural substrate underlying this mechanism. (4) Finally, a relatively newly discovered system that seems to play a key role in modulating these previously described systems is the insular cortex, which is involved in translating homeostatic and interoceptive signals triggered by deprivation, or by exposure to reward cues, into craving and what may become subjectively experienced as an intense urge for a reward. The consequences of overactivation of the insular cortex are intensification of the impulsive (striatum-dependent) system and weakening of the reflective (prefrontal cortex-dependent) system. Clinical evidence shows that damage to this neural system as a result of a stroke is sufficient to wipe out the urge and tendency to smoke in individuals previously addicted to smoking.

Future Directions

Our discovery of the important role of the insula specifically in smoking addiction does not undermine the seminal work performed to date on the roles of other components of the neural circuitry implicated in addiction, and impulse control disorders in general, especially the mesolimbic dopamine system and the prefrontal cortex. Addressing the role of the insula only complements this prior work and advances our efforts in finding novel therapeutic approaches for treating several impulse control disorders, including breaking the cycle of addiction. Therefore, stimulation of future research on the insula has a number of implications for clinical studies. The most obvious is that therapeutically modulating the function of the insula may make it easier to overcome one's addiction and solve other impulse control problems. This could be accomplished by designing new pharmacological therapies that target receptors within the insula. Invasive techniques such as deep brain stimulation are another option. However, noninvasive techniques such as repetitive transcranial magnetic stimulation are promising, once the techniques are modified so that they can reach deeper brain structures such as the insula.

Author Note:

The research for this study was supported by the following grants from the National Institute on Drug Abuse (NIDA): DA11779, DA12487, and DA16708; National Science Foundation (NSF) Grant No. IIS 04-42586; and NINDS Program Project Grant P01 NS19632.

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PART 6

Lifespan and Gender Issues

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Impulsivity in Childhood

Kelda H. Walsh and Christopher J. McDougle

Abstract

This chapter discusses impulse control disorders in children 12 years of age and younger. In this age group, the available research focuses on fire setting/pyromania, trichotillomania, and pathological gambling. Less well studied are kleptomania, intermittent explosive disorder, and the impulse control disorder not otherwise specified, pathological skin picking. Clinical presentation, diagnosis, epidemiology, age of onset, risk factors, sociocultural factors, and comorbidity will be reviewed. Psychotherapeutic interventions for the age group will be explored, with particular emphasis on behavioral therapy. The available literature on psychopharmacological treatments, particularly selective serotonin reuptake inhibitors and opioid antagonists, will also be explored.

Keywords: children, fire setting, pyromania, trichotillomania, pathological gambling, kleptomania, intermittent explosive disorder, impulse control disorder, pathological skin picking, selective serotonin reuptake inhibitor, naltrexone

Introduction

The personality dimension impulsivity is central to many behavioral disorders of children and adults. Impulsive actions “appear poorly conceived, prematurely expressed, unduly risky, or inappropriate to the situation and... often result in undesirable consequences” (Daruna & Barnes, 1993, p. 23). Global excessive impulsivity in childhood is a defining feature, along with hyperactivity and inattention, of common attention-deficit/hyperactivity disorder (ADHD). In contrast, the less frequent childhood impulse control disorders (ICDs) are characterized by failure to resist focal maladaptive impulses, such as the urge to set a fire or pull out hair. Typically, increasing tension or arousal precedes, and is relieved by, the impulsive act in an ICD, although children may be less likely to endorse this tension/relief pattern. While negative consequences follow the impulsive act, such as property damage, financial loss, or personal injury, patients with ICDs persist in their maladaptive behaviors. The

ICDs have been conceptualized as existing on an impulsive-compulsive spectrum of psychiatric disorders (McElroy et al., 1992). Some DSM-IV-TR ICDs resemble more classic impulsive, externalizing disorders or addictions in symptomatology and may be associated with comorbid conduct disorder or ADHD. These ICDs include intermittent explosive disorder, pathological gambling, pyromania, and kleptomania. Pathological grooming ICDs, however, appear to lie closer to compulsive disorders on the impulsive-compulsive spectrum; these ICDs include trichotillomania and pathological skin picking, the latter an impulse control disorder not otherwise specified.

Typically, ICDs first appear in adolescence. In adult studies, ICDs are more common in adult men than women, with the exception of trichotillomania, which predominates in females. Adult treatment studies have investigated selective serotonin reuptake inhibitors (SSRIs), mood stabilizers, and opioid antagonists, as well as cognitive-behavioral

therapies, for the ICDs, with trichotillomania and pathological gambling receiving the most research attention (Dell'Osso et al., 2006).

This chapter focuses on the English-language literature from the last 20 years regarding childhood ICDs. Data, where available, regarding epidemiology, comorbidity, family and societal risk factors, and treatment will be reviewed for each ICD. Studies focusing on adolescents and adults are reviewed in other chapters.

Pyromania and Fire Setting

Like other ICDs, pyromania is associated with rising tension before the act of fire setting, followed by relief or pleasure after setting the fire or watching its aftermath. According to the DSM-IV-TR, individuals with pyromania must have set fires deliberately on multiple occasions and have exhibited fascination, interest, or curiosity about fire. This fire setting should not be done for secondary gain or political purposes. Pyromania is not diagnosed if fire setting is better accounted for by diagnoses of conduct disorder, mania, or anti-social personality disorder (American Psychiatric Association, 2000).

Pyromania is rarely diagnosed in children. In one case series of 250 pediatric fire setters, only 4 were found to meet criteria for pyromania (Sakheim & Osborn, 1999). Rising tension and relief are often not reported in juveniles who set fires repeatedly; in addition, many of the juveniles who set fires repeatedly meet criteria for conduct disorder and thus are excluded from the diagnosis of pyromania. In a series of child psychiatric outpatients, only 6.5% of juvenile fire setters met pyromania criteria, while 64.5% met criteria for conduct disorder (Heath et al., 1985).

However, while pyromania may be an uncommon pediatric diagnosis, juvenile fire setting is common and dangerous. In the United States, 49% of arsonists arrested are under the age of 18, the highest juvenile percentage among the serious crimes. In 2006, 9.1% of people arrested for arson in the United States were between 10 and 12 years old (FBI, 2006). Juvenile fire setting is similarly problematic across the world. Twenty percent of Australian fires were attributed to juveniles in a Queensland Fire and Rescue Service report (as cited in Dadds & Fraser, 2006). The average structural damage from a juvenile fire exceeded \$US20,000 in 2006 (FBI, 2006). Children are the primary victims of juvenile-set fires and comprise 85% of lives lost in those fires (U.S. Fire Administration, as cited in

Putnam & Kirkpatrick, 2005). Most pediatric fire injuries and deaths are directly related to the child's own involvement in the fire (Hall, 2000).

Childhood fire behaviors have been divided into fire play and intentional fire setting. Fire play is motivated by curiosity, such as playing with matches or lighters. Fire play has been reported in a large minority of children, including 45% of boys evaluated in kindergarten, second, or fourth grade, approximately ages 5, 8, and 10 years (Kafry, 1980). Unfortunately, in one-third of first fire play episodes, flames spread beyond the source of ignition, the match or the cigarette lighter, and cause fires. Eighteen percent of these fires are ignited by children under the age of 3 years. Very young children may be particularly likely to set dangerous fires because they lack awareness of combustibility and how to handle emergencies; they may hide after igniting a fire, increasing the risk of smoke inhalation and burn injuries (Kafry, 1980).

Unlike simple fire play, fire setting involves "systematic gathering of flammable materials with the fire being directed at a clearly defined target, either person or property, with anger or revenge as the most commonly reported motives" (Adler et al., 1994, p. 1195). The remainder of this section will focus on pediatric fire setting.

Prevalence and Gender

Large-scale prevalence studies of fire-setting behaviors in nonclinical samples of juveniles demonstrate male predominance across age groups. In a recent Italian study, 29% of 567 11- to 18-year-olds reported that they had set fires; 40% endorsed setting multiple fires. Boys were significantly more likely to set fires than girls, with 80% of those reporting fire setting being male (Del Bove et al., 2008). In a study of 1359 4- to 9-year-olds in Brisbane, Australia, boys were significantly more likely to report fire interest and match play than girls. Increasing age in boys, but not in girls, was significantly associated with match play, with match play rising from 2.5% of 4- to 6-year-old to 6.5% of 7- to 9-year-old boys (Dadds & Fraser, 2006). In a clinical sample, 18.9% of outpatients at a child guidance clinic had set fires, all of whom were male (Fitzgerald & O'Hanlon, 1991).

Comorbidity

Comorbidity with conduct disorder (CD) is frequent in pediatric fire setters. Conduct disorder was significantly more common in fire-setting children (31.3%) versus nonfiresetters (6.6%) in a study of

6- to 12-year-old children experiencing domestic violence (Becker et al., 2004). Living in a violent home also significantly increased the risk of cruelty to animals, while other factors such as sexual abuse did not influence either cruelty or fire setting. Even higher rates of CD, 53.3%–75%, are reported in outpatient and inpatient clinical samples of juvenile fire setters (Fitzgerald & O'Hanlon, 1991; Heath et al., 1985; Kazdin & Kolko, 1986; Kuhnley et al., 1982).

Attention-deficit/hyperactivity disorder is less commonly reported as a comorbid condition with fire setting. It was reported in none of 32 fire-setting pediatric outpatients, 16.1% of fire-setting pediatric inpatients, and 17% of children remanded to a fire-setting treatment program; in the first two series, CD was much more frequent (Franklin et al., 2002; Heath et al., 1985; Kuhnley et al., 1982). Untreated ADHD may increase the risk of burn injury to children. In a retrospective case series, 0.5% of pediatric burn unit patients had ADHD. While they composed a very small percentage of the children in the burn unit, their behaviors leading to the burns were particularly dangerous. Half of the patients with ADHD had engaged in impulsive fire play, often using a flammable liquid or aerosol can with the ignition source; 42.8% of the children were off medication when they burned themselves (Thomas et al., 2004).

An unusual apparently comorbid condition is Klinefelter syndrome, leading one group to recommend chromosomal analysis for young male arsonists (Kaler et al., 1989). At least four cases of fire setting have been reported in children with Klinefelter syndrome. One of these boys stopped setting fires after treatment for Klinefelter syndrome with testosterone (Miller & Sulkes, 1988). Even less common is coincident fire setting and photosensitive epilepsy, reported in a father and his young daughter. The daughter's fire setting, present since infancy, declined markedly after she was required to remain at least 3 meters away from the television set and was treated with anticonvulsants (Meinhard et al., 1988).

Motivation and Recidivism

Motivations for fire setting have been studied in children in order to clarify risk factors for recidivism and to target treatment. Fire setters have been subtyped, for instance, into low-risk *curious* fire setters, who lack motivation and set a fire during a lapse in adult supervision, versus higher-risk *thought-disordered, delinquent, thrill-seeking, revenge-based, compulsive/pyromaniac*, or the highest-risk

disordered-coping subtype. The last group uses fire setting as a way to respond to "any assault on the ego that produces emotional discomfort" (Williams & Clements, 2007, p. 69). This clinical model has not been subject to robust research verification.

In studies, excitement and thrill seeking are frequent motivations for children who set fires; finding fire exciting may be a risk factor for more severe and recurrent fire setting. Thrill seeking was significantly associated with fire setting in a study of 4- to 9-year-olds (Dadds & Fraser, 2006). In a study of 186 juvenile fire setters, most of whom were 12 years of age or younger, children often cited multiple motivating factors. The most popular motivations were curiosity (49%), excitement (30%), and revenge (17%; Showers & Pickrell, 1987). When Sakheim, Osborn, and Abrams (1991) compared children who had engaged in severe (deliberate, planned, persistent) fire setting to those who had engaged in minor fire setting, they found that almost half of the severe fire setters were excited by fire, but none of the minor fire setters were. In a later study, they found that minor fire setters were significantly more likely to experience guilt over fire setting than major fire setters (Sackheim & Osborn, 1999). Heightened interest in fire was a significant predictor of recidivism among 192 children and adolescents who completed the Fire Interest Interview (McKay et al., 2006).

Motivation may also predict fire-setting sites. In one study, fire setters motivated by revenge were more likely to set fires at home. Those motivated by excitement set fewer home fires, instead setting fires on public wastelands in groups, running through the fires to increase excitement (Fitzgerald & O'Hanlon, 1991). Similarly, group fire setting was endorsed by slightly more than half of 95 children who completed the Fire Incident Analysis for Children interview (Kolko & Kazdin, 1994). The most common motivation for setting fires was "playing around" or having fun; older children were more likely to endorse motivations of anger or revenge.

Parental Features

Many investigators have expressed concern about parental indifference to pediatric fire setting. In some studies, a substantial minority of parents, roughly 25%, has been unaware of the fire involvement of their children (Del Bove et al., 2008; Kolko & Kazdin, 1994). In an outpatient study, few parents of fire setters spontaneously reported fire setting as a symptom of concern, not volunteering information

about fire setting unless specifically asked (Fitzgerald & O'Hanlon, 1991). Severe pediatric fire setting has been associated with lack of parental response to previous fire setting (Kolko & Kazdin, 1994). Other juvenile fire setters may be particularly vulnerable to fire setting, not because their parents are unmoved by the fires, but because they are absent. A study of incarcerated children found that the fire-setting detainees were significantly less likely to have a mother living in their home than other detainees (Ritvo et al., 1983).

Other studies have focused on parental psychopathology and styles of discipline. Mothers of inpatient pediatric fire setters, compared to mothers of other psychiatric inpatients, endorsed more personal psychiatric symptoms and depression, poorer adjustment in dyadic relationships, and poorer monitoring and control of their children (Kazdin & Kolko, 1986). In another study, fire setters were more likely than non-fire-setting children to be exposed to a combination of harsh discipline and "less effective mild punishment for general misbehavior in the home" (Kolko & Kazdin, 1989, p. 173). In the study of pediatric detainees, 11% of the fire setters, but none of the other detainees, had been punished with fire (Ritvo et al., 1983).

Fire-Setting Treatment

In many jurisdictions, judges send pediatric fire setters to their local fire stations for educational intervention as their primary or sole treatment. Specially trained firefighters deliver information to the fire-setting child and family, and are responsible for determining which children have significant psychopathology and require mental health referral. This process has concerned mental health providers. Some seriously mentally ill children may be missed, as was reported in a retrospective review of 30 Child Behavior Checklists completed by the parents of juvenile fire setters. The checklists were scored after treatment referral, and only 41% of fire setters with pathological scores had been referred for mental health assessment (Pierce & Hardesty, 1997).

Some fire treatment programs have set up assessment protocols to determine the risk of future fire setting and evaluate psychopathology. The Burn Education Awareness Recognition and Support program (BEARS) in Chicago uses the Federal Emergency Management Agency (FEMA) Tool, which includes a standardized child interview, a family interview, and a child behavioral assessment form. Upon completion of the Tool, mental health referrals are made for the highest-risk group,

and fire safety education only is recommended for the curiosity/little-risk group (Bennett et al., 2004). Families may find firefighter referrals more acceptable and may resist mental health referrals (Webb et al., 1990).

For many children, brief educational interventions may be sufficient to stop fire-setting behaviors. Adler et al. (1994) studied the Australian Juvenile Fire Awareness and Intervention Program. A total of 138 child and adolescent recurrent fire setters (mean age 8.1 years, 97.1% male) were randomized to four treatment arms, which offered combinations of fire educational material, firefighter's intervention, and referral to a specialty clinic for fire setters. There was no significant difference between treatment groups. Children who received only an initial interview about fire setting and a safety pamphlet did just as well as children who received more intensive interventions. Due to these findings, the Intervention Program was modified so that more intensive behavioral interventions were offered only to older children who continued to set fires after initial fire safety training.

One-day multidisciplinary interventions have been designed for juvenile fire setters and have demonstrated efficacy in controlled and uncontrolled studies. In the Trauma Burn Outreach Prevention Program, 132 4- to 17-year-olds completed family visits to the burn unit, skin bank, and morgue and received fire safety equipment for home use (Franklin et al., 2002). Patients in the group had set serious fires, averaging more than \$4000 in property damage. One fire had resulted in the death of two of a fire setter's siblings. The treatment group was compared to a control group of fire setters who met criteria for the program but did not attend. Thirty-six percent of the untreated and 0.8% of the treated fire setters set further fires, according to review of fire department and court records at 8-month to 2.5- year follow-up.

Similarly, Colorado Juvenile Fire Offenders (JFO) has focused on a 1-day uncontrolled intervention (Carroll et al., 1986). A total of 220 children (as young as 4 years old) and adolescents completed the program, which consists of a half-day fire district training center intervention and a half-day burn center intervention. Parents were required to accompany their children to the program after it was determined that only 12% of families had provided fire prevention interventions at home following participation by the children only. Parental involvement increased reported home safety interventions to 65%. Overall program success was

assessed by monitoring referrals to social services following the intervention; none of the fire setters returned to social services in the first 3 years of the program.

Kolko (2001) investigated fire-setting outcomes in a controlled study that included more standardized mental health intervention. Two treatment arms were compared: 16 children who received home-visit treatment with a firefighter (HVF) versus 38 five- to 13-year-olds who attended one of two 8-week manualized interventions, either fire safety education (FSE) or cognitive-behavioral therapy (CBT). The HVF intervention was similar to that provided by the Australian program. In CBT, children practiced several skills, including self-control, problem solving, and conflict resolution. Their parents learned about fire-setting motivation, promoting prosocial activities, and providing consequences for fire setting. Families in the FSE intervention were trained in fire safety, evacuation, and reporting. The parents completed home safety education. At 1-year follow-up, all treatment groups had reduced fire setting and match play. However, families who had completed CBT reported significantly greater reductions in those dangerous behaviors. Children in the CBT group showed the greatest reduction in deviant fire behavior, while children receiving HVF showed an increase. The FSE intervention significantly reduced the number of children showing interest in fires. While HVF was not superior to the other interventions on any follow-up measure, it was inexpensive and adequate for many of the fire setters.

Intermittent Explosive Disorder

People with intermittent explosive disorder (IED) have abrupt aggressive outbursts that cause physical harm or property destruction. The aggression is grossly disproportionate to any provocation (DSM-IV-TR). Studies of IED, even in the adult population, are uncommon and suggest a mean age of onset of approximately 14 years (Kessler et al., 2006; McElroy, 1999). Studies focusing on the pediatric population are lacking. However, a structured interview, the Module for Intermittent Explosive Disorder, has been used to characterize explosive anger in a pilot study of 10- to 17-year-olds (Olvera et al., 2001). Youth meeting IED criteria differed from psychiatric controls, demonstrating significantly more weekly physically aggressive and lifetime aggressive episodes. The youth with IED scored higher on indices of ADHD and oppositional-defiant disorder but not conduct disorder;

the majority also screened positive for symptoms of mood disorders, especially depression.

Childhood-Onset Physical Aggression

Although the pediatric literature on IED is sparse, childhood-onset physical aggression has been subject to intense research scrutiny. Physical aggression is common in toddlers and preschoolers. Children demonstrate strategic use of physical force against peers by their first birthday (Baillargeon et al., 2007), with the most rapid onset of physical aggression seen between 11 and 15 months of age (Tremblay et al., 1999). Fifty-eight percent of preschoolers, both boys and girls, demonstrate modest physical aggression (Tremblay et al., 2004). However, a small group of children are markedly more aggressive than their peers. In this group, gender differences are clear by toddlerhood, with 5% of 17-month-old boys, but only 1% of same-age girls, demonstrating frequent physical aggression in a large birth cohort study (Baillargeon et al., 2007). When cohorts of children are followed into preadolescence, most of the older children demonstrate a pattern of decreasing physical aggression with age, especially girls, although a minority persists in stable use of physical aggression throughout childhood (Cote et al., 2006; Lee et al., 2007). This persistently aggressive minority has been followed into adulthood in several large-scale longitudinal studies. Men who were members of the most aggressive group in childhood demonstrate significantly more adult delinquency and lower educational and occupational achievement (Asendorpf et al., 2008). Childhood onset of aggression, as opposed to adolescent onset, may be a marker for persistence of serious aggression into adulthood. A New Zealand birth cohort study of 539 men found that those with conduct problems prior to adolescence were much more likely to be convicted of violent offenses by the age of 26 (Moffitt et al., 2002). While aggressive boys are more common and better studied, a recent longitudinal study focused on 881 girls. Physical aggression, as reported by teachers, declined between the ages of 6 and 12. However, at age 21, those women rated highly hyperactive and physically aggressive in childhood were significantly more likely to report that they continued to engage in physical aggression, became pregnant early, and required welfare assistance than their less aggressive and hyperactive peers (Fontaine et al., 2008). Another large-scale longitudinal study found that high parent ratings of aggression in 3-year-old girls strongly predicted continued aggression at age 12 (Pihlakoski et al., 2006).

Many risk factors contribute to childhood-onset physical aggression. In the Canadian National Longitudinal Study of Children and Youth, significant risk factors included male gender, low income, having a mother who did not complete high school, and hostile/ineffective parenting (Benzies et al., 2008; Cote et al., 2006). Other studies have identified risk factors such as birth weight exceeding 4500 grams, maternal prenatal smoking (Huijbregts et al., 2007), pregnancy and delivery complications, male gender, family antisocial behavior (Buschgens et al., 2009), neglect before the age of 2 (Kotch et al., 2008), and receptive vocabulary deficits (Seguin et al., 2009). The impact of media violence on childhood aggression is controversial; at most, media violence appears to increase the rate of aggression in children under the age of 10, but not in older children and teenagers (Ferguson et al., 2009; Manganello & Taylor, 2009; Paik & Comstock, 1994). Peer factors contributing to sustained physical aggression include early rejection by peers and association with other aggressive peers (Dodge et al., 2003; Van Lier et al., 2007).

Two temperament styles have been associated with persistent physical aggression. Children with persistently negative affects, who are unable to adapt and are irritable, struggle with early-onset aggression, particularly impulsive or reactive aggression. This style may be associated with impaired social informational processing, in which children “too readily impute bad intentions to others,” misreading the actions of others as hostile (Blader & Jensen, 2007; Lochman & Dodge, 1994). These children may also lack the ability to self-regulate, a skill that depends on the maturation of prefrontal-limbic connections and on modeling by flexible, supportive caregivers (Calkins & Keane, 2009). The other temperament style associated with persistent physical aggression is callous-unemotional. This style may be associated with proactive or instrumental aggression, which is calculated, covert, and not associated with anger dyscontrol. Children demonstrating prominent proactive aggression have been found to be more confident about aggressive acts and to have difficulty planning competent behavioral responses (Crick & Dodge, 1996).

Genetic studies of physical aggression are relatively new and await replication. Genes possibly associated with higher childhood-onset aggression include low-expressing variants of the serotonin transporter polymorphism (Beitchman et al., 2006), the serotonin 2A receptor Tyr 452 allele (Mik et al., 2007), and valine/valine homozygosity for the

catechol-*O*-methyltransferase (COMT) gene (Caspi et al., 2008; Monuteaux et al., 2009). The COMT gene modulates prefrontal cortex dopamine levels; valine carriers perform less well on measures of executive functioning (Tunbridge et al., 2006).

Recent medication treatment studies of childhood physical aggression have focused on children with developmental disabilities or ADHD. Stimulants have some efficacy for ADHD-associated aggression, although medications used for ADHD, including stimulants and atomoxetine, may also trigger aggression in less than 2% of patients (Connor et al., 2002; Polzer et al., 2007). A controlled study of valproic acid augmentation of stimulants demonstrated significant improvement versus placebo; prior to augmentation, 31 of 74 children had remitted on stimulant lead-in (Blader et al., 2009). A risperidone augmentation study of stimulants was also positive, although the placebo response rate was 77% (Armenteros et al., 2007). Risperidone as a single agent has demonstrated efficacy for aggression in children with below-average IQ and disruptive behavior disorders (LeBlanc et al., 2005) and in a multisite placebo-controlled study of children and adolescents with autism (Research Units on Pediatric Psychopharmacology Autism Network, 2002). Positive open-label studies have been published for quetiapine (Findling et al., 2006), aripiprazole, and ziprasidone (Bastiaens, 2009).

Psychosocial treatments for childhood-onset aggression have been delivered in a wide variety of formats, often including family, school, and individual therapy components. On meta-analysis, behavioral interventions demonstrated significantly larger effect sizes than family therapy; interventions were especially likely to be effective for younger children (Fossum et al., 2008). Among school programs, better-implemented programs, especially those targeting high-risk students, demonstrate higher efficacy (National Gambling Impact Study Commission, 1999).

Pathological Gambling

Pathological gamblers exhibit a preoccupation with gambling, need to use greater amounts of money to become adequately excited, and are unable to cut back or control their gambling. They may jeopardize relationships or employment, commit illegal acts, or borrow money to cover losses (American Psychiatric Association, 2000).

In many countries, access to legalized gambling was minimal before the 1970s, but it has expanded markedly in the last four decades. Many forms of

gambling are widely available, including casinos, scratch-off tickets, and national or state lotteries. In the United States, for instance, state lotteries were outlawed in the 1870s following a lottery scandal, but they reemerged in 1964 when New Hampshire established the first modern lottery. In the 1970s, the number of state lotteries expanded greatly (National Gambling Impact Study Commission, 1999; Olason et al., 2006; Wood & Griffiths, 2004). Gaming revenues have become important for balancing regional and national budgets, and advertisements may emphasize how gaming funds educational or infrastructure development (National Gambling Impact Study Commission, 1999; Volberg, 1994). Commonly, television programs announce the weekly lottery winners, and advertisements for gambling are widespread. Lottery tickets are sold in local grocery and convenience stores, often as an impulse item close to the candy display and the cash register (Moran, 1995). In the United Kingdom, as much as 90% of the adult population reports gambling at least once, and the majority play on a weekly basis (Camelot as cited in Wood & Griffiths, 1998).

Problem gambling is likely to manifest early, in adolescence. From 4% to 8% of adolescents meet criteria for pathological gambling, exceeding the rates reported in adult studies of 1%–3% (Deverensky & Gupta, 2000; Deverensky et al., 2003; Jacobs, 2000). Thus, very-early-onset gambling may be a particular risk factor for pathological gambling.

While gambling seems almost universally distributed, it is usually illegal for children to gamble. Despite this, children do gamble, often with the assistance and approval of their families (Wood & Griffiths, 1998, 2004). In a study of primary school students, 81% of 8- and 9-year-olds had already bet money, although a later study found that parents estimated that children's gambling did not begin until after the age of 11 (Ladouceur et al., 1994, 2001). Other studies of gambling in children and teenagers have reported an age of first gambling ranging from 7.95 to 11.2 years (Felsher et al., 2004; Westphal et al., 2000). Gupta and Derevensky (1997) reported that 81% of gambling 9- to 14-year-olds gambled while accompanied by family members.

Gambling in preteenagers appears quite common, and scratch-off tickets appear to be a common early form of gambling. In a 1996 Louisiana survey of 11,736 6th through 12th graders, scratch-off tickets were the most frequent form of weekly gambling (Westphal et al., 2000). A Canadian survey of 1072 6th through 12th graders reported results by grade level (Felsher et al., 2004). Most of the children in

the study, sixth and seventh graders, mean age 11.2, were unaware of the legal age for lottery ticket buying. However, 53.8% of them reported being at least occasional participants in scratch-off ticket gambling, the most popular form of gambling chosen. Scratch-off tickets were also the earliest form of gambling among this group of youngsters, who reported first playing at a mean of 7.95 years and first purchasing tickets at a mean of 9.03 years. Many did not perceive scratch-off tickets as a form of gambling; 55.3% reported that it was easy for them to buy tickets.

Studies of adult problem gamblers suggest that early onset of gambling increases the risk of later pathological gambling (Burge et al., 2006). For instance, in an American survey study, 23%–36% of adult problem and pathological gamblers reported they had started gambling before the age of 15, versus only 7%–8% of all gamblers (Volberg, 1994). A prospective study of youth gambling found that early onset of gambling (by sixth grade, or approximately 11 years of age) was a significant risk factor for problem gambling in young adulthood (Winters et al., 2002).

Impulsivity, measured by self-reports and by a history of childhood ADHD, is increased in adult pathological gamblers (Rodriguez-Jiminiz et al., 2006). Prospective studies of children have also demonstrated links between impulsivity and onset of gambling. A longitudinal study of 903 Canadian boys explored the relationship between the age of gambling onset, impulsivity, inhibition, and the risk of problem gambling (Vitaro et al., 2004). Gambling data were collected from ages 11 to 17. Most of the boys (61.7%) followed a trajectory of low gambling involvement. Two high-risk groups were also identified. The chronic high-gambling group, 22.1% of the sample, included boys who were likely to gamble by the age of 11 and remained highly likely to gamble throughout the study period. By the age of 17, more than 20% of the chronic high-gambling group fit the profile of high-risk or problem gamblers. These boys described themselves on Eysenck Impulsiveness and Venturesomeness self-rating scales as more impulsive than the other groups. They were rated by their teachers as less inhibited at ages 6 and 10. A third group did not start gambling before age 13 but rapidly increased the gambling behavior once initiated; 15% met criteria for problem or high-risk gambling at age 17. Boys in this late-onset gambling group rated themselves as intermediate in impulsiveness, and their teachers rated them as more inhibited than the chronic high-gambling group. Overall, this

study suggests that impulsivity and lack of inhibition, even as measured in childhood, may predict a higher risk for problem gambling later in life. Similarly, in another longitudinal study of children in Baltimore, parents reported high levels of impulsivity and hyperactivity in first graders who became gamblers by age 17 (Martins et al., 2008).

Peer behavior may significantly impact gambling behavior, especially in girls. A computer-simulated roulette game was used to study individual and group gambling behavior in 130 fourth through sixth graders (Haroon & Deverensky, 2001). Boys bet more money consistently, slightly increasing their wagers throughout the trials. Girls increased their wagers in group settings. It appeared that children tried to impress each other in group settings by betting more.

While gambling behavior appears common in children, pathological gambling is infrequent before adolescence, so treatment studies of children have not been reported. Rather, treatment models for children have focused on prevention. Children may be particularly appropriate candidates for primary prevention methods, which are aimed at preventing the onset of risky gambling. Proposed objectives for primary prevention of gambling include increasing knowledge in youth, parents, and the general public about the risks and consequences of gambling. Goals would include changing media depictions of gambling and modeling by parents and peers. Other methods could include limiting availability and visibility of gambling materials to youngsters in retail stores and enforcing age restrictions on gambling (Messerlian et al., 2005).

Kleptomania

People with kleptomania do not resist their recurrent impulses to steal; they are motivated more by a desire to take or have an item than to sell or use it (American Psychiatric Association, 2000). Kleptomania is not well characterized in childhood. Adults with kleptomania typically report onset of shoplifting at around the age of 16 years (Feeney & Klykylo, 1997; Grant & Kim, 2002). One case report has detailed the treatment of an 11-year-old with ADHD and kleptomania. The combination of sertraline and methylphenidate was successful in stopping her recurrent stealing (Feeney & Klykylo, 1997).

Childhood-Onset Theft

Childhood theft is slightly better studied than childhood kleptomania, but prevalence data are lacking

for both. Developmental studies have demonstrated that children have a different moral response to theft than adults. Small children expect that thieves will feel happy after successful completion of dishonest acts, even though other studies find that adults would not predict this expectation in children (Barden et al., 1980; Zelko et al., 1986). Four-year-olds linked positive emotions to material gains from theft in one study, while 8-year-olds believed that thieves would have less positive emotions due to growing understanding of the inherent harm and unfairness associated with theft (Arsenio & Kramer, 1992).

Theft is a covert antisocial behavior (Loeber & Schmalin, 1985) often seen in conjunction with other covert behavior problems such as lying, defiance, and wandering off without permission. Serious acts of theft are more common in youth with early-onset behavior problems (Taylor et al., 2001). Early theft is linked to ultimate diagnosis of disruptive behavior disorders and earlier commission of armed robbery (Gaber et al., 1987; Lahey et al., 1999). A follow-up study of children referred for behavior problems found that theft, more than aggression, significantly predicted juvenile court involvement at 2- to 9-year follow-up. Fifty-six percent of children with at least four parent-reported episodes of theft (at a mean age of 9.8 years) versus 13% of aggressive peers without a theft history had juvenile court records at follow-up (Moore et al., 1979). A juvenile detention facility study found that theft was the most frequently reported offense among young detainees. Young people arrested for theft were more likely than those arrested for other crimes to be arrested before the age of 12, to have multiple charges, and to be charged later with other offenses, including assault (Taylor et al. 2001). Despite the concern that theft may be a *gateway crime*, treatment and prevention research for juvenile theft is minimal; it remains unclear what constitutes normative versus pathological theft behavior in childhood (Taylor et al., 2001).

Pathological Grooming: Trichotillomania and Pathological Skin Picking

Trichotillomania

People with trichotillomania (TTM) repeatedly pull out their hair, causing visible hair loss. They experience rising tension prior to pulling, followed by “pleasure, gratification, or relief” after pulling (American Psychiatric Association, 2000). However, in multiple pediatric case series, 20.6%–90% of children with TTM denied feeling rising tension

followed by relief. The highest denial rate was reported in the youngest series, mean age, 9.9 years (Hanna, 1997; King et al., 1995; Reeve et al., 1992; Tolin et al., 2007). Interestingly, three boys who developed secondary TTM following stimulant treatment all endorsed tension and relief (Martin et al., 1998).

DIAGNOSTIC FEATURES: PULLING SITES AND BIOPSY FINDINGS

Children with TTM most commonly pull scalp hair, typically in a diffuse pattern. Hair loss may be more severe on the side of the dominant hand (Malhotra et al., 2008). Some children may demonstrate the *Friar Tuck sign* from pulling primarily at the crown (Tay et al., 2004). Generally, eyebrows and eyelashes are reported to be the second most common sites of pulling, and 26%–54% of children report pulling from multiple sites (Hanna, 1997; Reeve et al., 1992; Santhanam et al., 2008; Tolin et al., 2007).

Occasionally, punch biopsy is necessary to diagnose TTM. Some children are very secretive, pull in private, especially at night, and deny pulling as the cause of hair loss. In case series of children and adolescents with hair pulling, 40%–53% initially denied this behavior (King et al., 1995; Santhanam et al., 2008). Parents may find the diagnosis difficult to believe (Stroud, 1983), especially when multiple family members or pets are also missing hair. One 7-year-old boy presented with focal hair loss, which was also demonstrated by two cats and a dog that liked to sleep with him. Biopsy of the little boy and one of the cats established that the hair loss was from pulling, not a shared fungal infection (Zone et al., 2003). Another 7-year-old was reported to pull and eat her own hair and that of her dog (Weller et al., 1989). Some children with TTM may pull hair from other children, and very young children may pull it from their parents (Kao et al., 2005; Oranje et al., 1986).

On examination, areas of hair loss (alopecia) from TTM may resemble alopecia areata, androgenic alopecia, traction alopecia (i.e., from tight hair elastics), or tinea capitis (Adams & Kashani, 1990; Bruce et al., 2005). In TTM, hair may be twisted before it is pulled, leaving hair of assorted lengths distributed over the bald spot, which is typically oval or linear (Stroud, 1983). On biopsy, normal hairs are seen growing “among empty hair follicles in a non-inflammatory dermis” (Bruce et al., 2005, p. 367). Trichomalacia and pigment casts have been described as major diagnostic features of scalp biopsy

in TTM (Bergfeld et al., 2001). The scalp may appear brown from rubbing (Stroud, 1983). Biopsy can establish that hair is being pulled, but not by whom, as demonstrated in one case in which a tutor was eventually demonstrated on surveillance video to be pulling hair from three young students who apparently had been afraid to report the abuse (Sarawasat, 2005).

EPIDEMIOLOGY

Trichotillomania has been described in the dermatological literature as being seven times more common in children than adults (Mehregan, 1970); however, data supporting that assertion are lacking. A large survey study of college students found a 0.6% lifetime prevalence of TTM and a 1.5% (male)/3.4% (female) lifetime prevalence of hair pulling causing visible hair loss (Christenson et al., 1991b). Mean age of onset of hair pulling in a series of 60 adults was 13 ± 8 years (Christenson et al., 1991a; approximately 9% reported that they had been pulling since they were 5 years of age or younger, and approximately 62% reported the onset of pulling by the age of 20 years. In three large case series in mental health clinics, less than 1% of pediatric patients presented with hair pulling (Fung & Chen, 1999; Mannino & Delgado, 1969; Ranga Rama Krishnan et al., 1985). Muller (1987) reported on a series of 145 patients with TTM seen over a 10-year period, ranging at age of presentation from 2 to more than 60 years of age. Thirty percent of these patients were children under the age of 10 years. In that series, the peak age of presentation was between 11 and 17 years; the age of onset was not reported. In Hanna's series of 11 children with TTM, age of onset ranged from 2 to 13.5 years (Hanna, 1997).

Overall, it appears likely that trichotillomania is not common in any age group, but may be more commonly seen in patients under, rather than over, the age of 18 years. Adolescent versus pediatric predominance has not been established.

A benign, self-limited variant of TTM has long been reported in children under the age of 6. However, long-term studies have not confirmed that very-early-onset TTM resolves spontaneously. Clear gender predominance has not been established in this young population, with some studies citing male and others female (Wright & Holmes, 2003) predominance. In Muller's population of patients with TTM, males predominated only in the 2- to 6-year-old age group. Muller noted, “in small children [TTM] is usually a minor problem and clears up spontaneously or with minor encouragement

and explanation by the primary physician" (Muller, 1987, p. 597). Wright and Holmes reported that "most" of ten toddlers treated with behavioral and family interventions to reduce stress had reduced or stopped hair pulling at 3-month follow-up.

COMORBIDITY

Comorbidity with other psychiatric disorders has been investigated in small case series of children with TTM; many of these series include adolescents. The majority of children with TTM appear to have psychiatric comorbidity, especially anxiety, mood, or habit disorders. Those studies assessing habit disorders report comorbid habits such as nail biting, thumb sucking, or skin picking in 10%–50% of children with TTM (Hanna, 1997; Malhotra et al., 2008; Oranje et al., 1986; Reeve et al., 1992). While TTM has been conceptualized by some researchers as an obsessive-compulsive spectrum disorder, obsessive-compulsive disorder (OCD) has been found to be comorbid with TTM in only a small minority of pediatric patients, ranging from 4.8% to 13.3%. Nor is OCD seen in the majority of their parents (King et al., 1995; Oranje et al., 1986; Tolin et al., 2007). Chronic tic disorders also appear uncommon in children with TTM, with no series demonstrating comorbid Tourette's disorder, and chronic tic disorders have been reported in only 0%–13.3%. Much more common are non-OCD anxiety disorders, particularly overanxious disorder (which has been subsumed into the diagnosis of generalized anxiety disorder in the DSM-IV-TR), in 20%–60%, and depressive disorders (especially dysthymia), in 8.7%–36% (Hanna, 1997; King et al., 1995; Reeve et al., 1992; Tolin et al., 2007). Significant comorbid anxiety was reported in 50% of a series of toddlers with TTM (Wright & Holmes, 2003). Comorbid mental retardation was reported in only one study, in 19% of patients (Oranje et al., 1986).

TRICHOBEOZARS

Some children with TTM engage in *trichophagy*, chewing and swallowing pulled hair. Most case series have not investigated the frequency of this behavior in children; one series found that 20% of children and adolescents reported it (King et al., 1995). Trichophagy can trigger the development of trichobezoars, large mats of swallowed hair combined with food particles and even fibers from carpeting or fabric. These hairballs can obstruct the stomach and extend into the intestine. Children as young as 2½ years of age have required surgical

removal of bezoars (Aleksandrowicz & Mares, 1978; McGehee & Buchanan, 1980). Children with trichobezoars are often chronic hair pullers, who may or may not have apparent hair loss at presentation with the trichobezoar. Symptoms of pediatric trichophagy include failure to gain weight, a painless abdominal mass, flatulence, halitosis, constipation, and nausea (De Backer et al., 1999; Phillips et al., 1998; Ramadan et al., 2003). Iron deficiency anemia has been reported in toddlers with trichobezoars, triggering speculation that TTM in this age group may resemble iron deficiency-triggered pica (McGehee & Buchanan, 1980).

TREATMENT AND OUTCOME

Many children with TTM do not admit to hair pulling. They may be oddly oblivious of their hair loss; as King et al. (1995) comment, their parents' concern "stood in marked contrast to the child's stated indifference" (p. 1454). These authors noted that "patients' own degree of distress, however, varied both over time and across subjects and was sometimes at variance with the observed degree of cosmetic impairment" (p. 1454) even in the presence of near-total baldness. While this denial may seem an insurmountable obstacle to engagement in psychotherapy, many pediatric treatment studies describe children who insisted that they were not pulling their hair but who completed therapy regardless.

HYPNOTHERAPY

Hypnotherapy has been described as a treatment modality for at least seven children, ranging from ages 3 to 12 years at the time of treatment (Cohen et al., 1999; Kohen, 1996). When possible, therapists started treatment by teaching the children to monitor their own hair pulling in order to increase autonomy in dealing with their symptoms. Older children were then taught relaxation/mental imagery and self-hypnosis. The hypnotherapist provided direct suggestions for one hand to "help" the other to stop pulling. The technique was adjusted to fit each child's age and interests. For instance, preschoolers were not expected to close their eyes for hypnosis, and the therapist used more playful language, teaching the child to use the nonpulling hand as the "friend" of the pulling hand. All of the pediatric patients were reported to stop hair pulling with the treatment, although one patient required later treatment for a brief relapse. Cohen et al. (1999) noted that this treatment method requires responsible parents and highly motivated patients.

PSYCHODYNAMIC FAMILY THERAPY

Brief hospital-based psychodynamic interpretation was used to treat a 27-month-old girl who had required surgical removal of a trichobezoar (Aleksandrowicz & Mares, 1978). The little girl was too fearful and tense to tolerate hypnotherapy, but she responded well to brief family interventions during her recovery from surgery. She demonstrated significant improvement at 6-month follow-up, although the extent of hair regrowth and subsequent pulling was not specified.

HABIT REVERSAL THERAPY AND BEHAVIORAL THERAPIES

Brief behavioral interventions have been successful for toddlers and preschoolers with TTM. Often, these interventions are paired with *time in*, or increased parental attention at nonpulling times. One strategy targeted thumb sucking in young children who suck their thumbs while pulling their hair, as thumb sucking is easier to extinguish than hair pulling. Applying a bitter substance to the thumb was reported to dramatically reduce thumb sucking and associated hair pulling in four children ages 2 to 5 years (Altman et al., 1982; Friman & Hove, 1987; Knell & Moore, 1988). Another family tried three interventions with a thumb-sucking, hair-pulling 5-year-old: a bitter substance, a thumb alarm (similar to a bedwetting alarm), and a leather thumb post. The thumb post proved the most successful and easiest intervention, stopping hair pulling and thumb sucking at day 14 (Watson & Allen, 1993).

Other behavioral interventions have been used for children with TTM who lack comorbid thumb sucking. For younger children, parents have placed socks on their children's hands at night and also when pulling was observed. This method demonstrated efficacy in an ABAB case study of a 3-year-old (Blum et al., 1993). A 27-month-old required double socks early in treatment; she was gradually weaned to single socks, then to socks of decreasing thickness. Hair pulling had resolved when the child was reassessed as a 4-year-old (Byrd et al., 2002).

School-age children have most commonly been treated with behavioral therapies, sometimes in combination with pharmacotherapy. In a review of 10 cases in which behavioral therapy alone was used to treat school-age children with TTM, 90% were reported to be successful (Bruce et al., 2005). Behavioral interventions used have included self-monitoring, token economy, response cost, facial screening (typically for children with mental retardation), and habit reversal therapy. In this age group,

habit reversal therapy may be simplified. A 9-year-old girl responded positively to a combination of increased physical nurturing by her mother and her teacher and simple response prevention. She would sit on her hands or hold a pencil with both hands if she was seen touching or pulling her hair. She had a full head of hair at 1-year follow-up (Blum et al., 1993).

Three children, ages 10 to 12½ years, completed a more intensive habit reversal regime (Vitulano et al., 1992). Over six sessions, the children learned self-monitoring, collecting pulled hair each day in labeled envelopes and logging hair-pulling episodes as well as urges to pull. In habit interruption and prevention training, they were taught to extend their arms down with fists clenched for 90 seconds in response to pulling or pulling urges, and also practiced the technique twice daily regardless of whether they had the urge to pull. They also brushed their hair daily as a form of overcorrection. Following the example of Azrin et al. (1980), the children recorded their reasons to stop pulling in an annoyance review (Moore et al., 1979). The children developed self-messages, such as "I want pretty long hair to brush," which they used frequently. Parents were instructed to give praise and tangible rewards for efforts to follow the treatment plan. The outcome was rated via several instruments, including self-reports and clinician-rated impairment and improvement scales, as well as photographic records of hair growth, which were rated blindly.

The children disliked collecting their hair in envelopes, and conflict with parents regarding pulling and monitoring by the children was problematic in all three cases. One child demonstrated improvement following an interlude of family therapy to improve parent-child interaction. By the end of the study, two of the children had demonstrated reductions in pulling and improved hair growth on photographs, while a third child, with significant depression, continued to pull, despite the use of clomipramine in addition to behavioral therapy.

MEDICATION TRIALS

Medication trials for TTM are few in the pediatric population, with most reports focusing on SSRIs. Treatment for children with TTM using SSRIs has been described in individual case reports and one retrospective review. In the review, which involved 20 patients, the age of clinical presentation ranged from 2 to 14 years (Malhotra et al., 2008). Fourteen of the children were treated with a variety of medications; nine children also received behavior

therapy. Half of the medicated children took fluoxetine, 10–20 mg daily, with others taking sertraline or the nonselective SRI clomipramine. The authors reported a 50% dropout rate; 80% of those completing treatment were rated as improved. Results for individual medication trials were not reported, and it was unclear if any medication was more effective than the others.

Fluoxetine treatment of four individual children has been reported, with the two who responded improving on doses of 10 mg daily (Palmer et al., 1999; Sheikha et al., 1993; Vitulano et al., 1992; Wilens et al., 1992). One of two children treated for depression and TTM responded to imipramine (Sheikha et al., 1993; Weller et al., 1989). A pair of 12-year-olds who were not responsive to fluoxetine were later treated with clomipramine; one child did not respond on 150 mg daily, while the other was reported to be doing well at 6-month follow-up on 250 mg daily (Vitulano et al., 1992; Wilens et al., 1992).

While neuroleptics, especially as augmentation agents, have been reported in several series and trials in adolescents and adults with TTM, only one child has been reported to have been treated with neuroleptics. This 11-year-old with severe mental retardation, autism, and TTM did not obtain relief on thioridazine but responded well to haloperidol 1 mg BID, relapsing on 1.5 mg daily and returning to remission of hair pulling on return to 1 mg BID (Ghazziudin et al., 1991).

Naltrexone, an opioid antagonist, has been investigated in TTM. People with TTM continue hair pulling despite the self-inflicted pain, suggesting that the pain response as mediated by the opioid system may be impaired. An open pilot study investigated naltrexone treatment in 14 children (mean age 9 years) with TTM (De Sousa, 2008). Patients reached a mean final dose of 66.07 mg daily in the 10-month study. Adverse effects were denied by the patients, and liver function test results were unchanged throughout the study. Of these children, 21.4% reported no hair pulling at the end of the study; 57% reported improvement.

Pathological Skin Picking

Pathological skin picking (PSP) is classified as an impulse control disorder not otherwise specified in the DSM-IV-TR. People with PSP engage in ritualistic or impulsive skin picking, sometimes spending hours a day on the habit (Odlaug & Grant, 2008; Wilhelm et al., 1999). Mean age of onset has been reported to range from 12 to 16 years (Flessner &

Woods, 2006; Grant & Christenson, 2007; Keuthen et al., 2001; Odlaug & Grant, 2008; Simeon et al., 1997; Wilhelm et al., 1999).

Odlaug and Grant (2007) compared adults with childhood-onset PSP to patients with later onset. In this study of 40 patients, 47.5% reported onset of picking before the age of 10 years. Childhood-onset patients were more likely to report subconscious picking, and reported a mean age of onset of 5.6 years. Symptom severity was similar in early- and late-onset patients.

Patients with the neurodevelopmental disorder Prader-Willi syndrome (PWS) appear particularly vulnerable to developing PSP in childhood, even when compared to mentally retarded controls (Akefeldt & Gillberg, 1999; Kim et al., 2005). In a Japanese questionnaire study, 35.3% of 2- to 5-year-olds and 56.1% of 6- to 11-year-olds with PWS were reported to exhibit excessive skin picking (Hiraiwa et al., 2007).

Treatment of pediatric PSP has rarely been reported in the research literature. A 9-year-old boy with PWS and severe skin picking causing deep wounds was treated with fluoxetine to a maximum dose of 60 mg daily without change in skin picking. Clomipramine augmentation exacerbated his overeating. When the child was hospitalized due to severe skin picking, naltrexone 50 mg daily was added to fluoxetine in an ABA model; each trial of the naltrexone-fluoxetine combination was associated with a significant decrease in skin picking within 7–10 days of treatment (Benjamin & Buot-Smith, 1993).

Behavioral therapy was the primary intervention used to treat a 9-year-old with borderline intellectual functioning, ADHD, and PSP (Lane et al., 2006). The little boy would pick and scratch at his skin in school. At baseline, he was observed to pick his skin during the majority of his school hours. He was given a box of three soft balls to handle during high-risk times. His skin picking was found to decrease from 67% of the time to 22% when he was taking his stimulant medication and using the soft balls to keep his hands occupied; skin picking increased on the days when the stimulant was not administered. Long-term follow-up was not possible.

Conclusion

Current data on childhood ICDs are incomplete, but suggest that at least a substantial minority of adults develop ICD symptoms or even full-blown ICDs as children. Some criteria for ICDs in the DSM-IV-TR appear less likely to be endorsed by children, such as rising tension and relief, while other exclusionary

criteria, such as the exclusion of comorbid conduct disorder in pyromania, do not reflect common presentations of highly symptomatic children. Pyromania/fire setting and TTM are the best-characterized childhood ICDs; however, large-scale epidemiological and treatment studies are lacking for all ICDs in this age group. Likewise, reproducible rating instruments appropriate for assessing children are not widely used in childhood ICDs, making objective assessment of treatment outcomes problematic.

Future Directions: Questions for Future Research

1. Does onset of TTM before the age of 6 years truly predict a benign, self-resolving course? Are there genetic and/or other biomarkers predictive of the outcome for TTM?
2. Can an objective rating instrument be developed for assessment of the recurrent risk of fire setting in children? Ideally, such an instrument should be accessible to non-mental-health assessors in the juvenile justice system.
3. Can effective public policy regarding gaming advertising and availability reduce the marked recent increase in gambling among children and adolescents?
4. Can behavioral techniques, including modified habit reversal therapy, be applied successfully to children with PSP?
5. Are there distinct syndromes of kleptomania and IED in children?
6. Can childhood ICDs be treated successfully with medications that have helped adolescents and adults, such as SSRIs, opioid antagonists, or mood stabilizers? Is neuroleptic augmentation reasonable and safe in this population?
7. Hormone therapy (testosterone) may help reduce fire setting in patients with Klinefelter syndrome. Will hormone therapy (typically human growth hormone) used to treat patients with PWS help skin picking?

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Impulsivity in Adolescents

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Abstract

Adolescence is the time between the beginning of sexual maturation and adulthood, typically bounded by the ages of 13 to 19 years. One construct that holds a central place in many theories of development and psychopathology is impulsivity. Impulsivity has been considered to play an important role in normal behavior as well as linked to several problematic behaviors that are present or arise during adolescence. Impulsivity, considered to be a multidimensional construct, has been defined and measured in a variety of ways. This chapter will discuss the definitions of impulsivity, measurement (including self-report and behavioral tasks), developmental course, behaviors and disorders in which it is implicated, and future directions.

Keywords: adolescence, impulsivity, measurement, development, externalizing behaviors

The word *adolescence* is from the Latin *adlescere*, which means “to grow into adulthood” (Lerner & Steinberg, 2004). Adolescence is the time between the beginning of sexual maturation (puberty) and adulthood, roughly considered to be the period between 13 and 19 years of age. It is a transitional stage of physical and mental development; the transition involves biological (e.g., body shape and composition change, menarche/spermache begins), social (e.g., struggle for greater autonomy, intense peer group involvement, initiation of romantic relationships and sexual activity), and psychological (e.g., emergence of abstract thought, questioning of mores, increasing introspection) changes (Marcell, 2007). Hormones together with social structures, designed to assist the transition from childhood to adulthood, set the stage for this developmental program (Marcell, 2007).

Adolescents are often portrayed as rebellious, distracted, thoughtless, and daring, that is, they appear to take greater risks with less appreciation or understanding of the consequences of their risk

taking. Indeed, because of a relative imbalance between risk taking and consequence appraisal, a number of problem behaviors begin to emerge during adolescence, including those often associated with increased risk taking such as substance use, sexual activity, and delinquent behaviors (DiClemente et al., 1996). Adolescence is also a period of increased incidence of several major mental illnesses, including major depression, bipolar disorder, panic disorder, and schizophrenia (Marcell, 2007). Suicide is the third most common cause of death in adolescence (CDCP, 2004) following unintentional injury (e.g., motor vehicle accidents) and assault (e.g., homicide).

One construct that holds a central place in many theories of development and psychopathology is impulsivity. Impulsivity has been considered to play an important role in normal behavior as well as linked to several problematic behaviors that are present or arise during adolescence, including risk-taking behavior (Luengo et al., 1994), substance use (e.g., Fillmore & Rush, 2002; Reynolds, 2006;

Zuckerman et al., 1990), suicide (Brodsky et al., 1997; McGirr et al., 2007), and an array of other clinical diagnoses (e.g., Barkley, 1997a; Gauggel et al., 2004; Heerey et al., 2007; Swann et al., 2002). Impulsivity may be especially key when considering the balance between adolescent risk taking, reward seeking, and consequence appraisal.

Defining Impulsivity

While the term *impulsivity* is commonly used, it is a construct that has poor universal meaning. Several definitions of impulsivity have emerged, including poorly conceived and/or prematurely performed actions that are either inappropriate or unduly risky for a particular situation, a tendency to act without forethought (i.e., respond quickly and without reflection), excessive discounting of rewards and punishments as a function of their delay, and an inability to inhibit inappropriate behavior (Ainslie, 1975; Barkley, 1997b; Barrat & Patton, 1983; Dickman, 1990; Douglas, 1972; Eysenck, 1993; Murrary, 1938; Rachlin & Green, 1972; Winstanley et al., 2006). This multitude of definitions has led to confusion and disagreement as to the exact nature of the construct and the appropriate means of measurement. One compromise, given the multiple definitions, has been to treat impulsivity as a multi-dimensional construct, although there still has been little agreement as to how best to characterize the different components (Ivanov et al., 2008).

Lorr and Wunderlich (1985) stated that there are two major components to the dimension of impulsivity: (1) resisting urges versus giving in to urges and (2) responding immediately to a stimulus versus planning before making a move. Barratt (1994) defined impulsivity in terms of three subfactors: acting on the spur of the moment (motor activation), not focusing on the task at hand (attention), and not planning and thinking carefully (lack of planning; Barratt, 1994). More recently, Dawe and Loxton (2004), in their review of the literature, concluded that impulsivity is best conceptualized as a two-dimensional trait. The first trait, labeled *Reward Drive*, is said to reflect individual differences in sensitivity to incentive motivation and engagement in appetitive behavior upon detection of reward cues. The second trait, *Rash Impulsiveness*, is proposed to reflect individual differences in the ability to modify or inhibit prepotent (Reward Drive-initiated) behaviors in light of potential negative consequences.

These definitions, and the literature in general, tend to treat impulsivity as a trait rather than a state, that is, not as a momentary event in an individual's

life. Further, it is treated as dysfunctional rather than as a functional trait that varies little with the demands of a given context or environment. Despite the definitional cacophony, across the different definitions and usages, the core features appear to be (1) the tendency to execute actions too hastily or in a thoughtless manner, (2) difficulty withholding or inhibiting actions, and (3) the tendency to seek immediate gratification at the cost of longer-term gains (Schachar et al., 1993).

Measurement

Various measures have been developed to assess impulsivity, including self-report measures that rely on self-perceptions of behavior and behavioral tasks that measure overt behavior. Below are descriptions of both self-report and behavioral tasks that are commonly used to measure impulsivity in adolescents.

Self-Report Measures

BARRATT IMPULSIVENESS SCALE

The Barratt Impulsiveness Scale (BIS) is one of the most widely used self-report measures of impulsive personality traits. The scale has been through several revisions, with version 11 being the most recent (BIS-11; Patton et al., 1995). The scale consists of 30 self-descriptive items, with responses in a 4-point Likert-type scale ranging from "Rarely/Never" to "Almost Always/Always." The questionnaire contains three subscales: Attentional Impulsiveness (actions precipitated by lack of attention), Motor Impulsiveness (hyperactivity due to need of movement), and Nonplanning Impulsiveness (attitudes and conclusions precipitated by lack of reflection). Although the BIS was originally developed for use with adults, it has been used commonly with adolescents (e.g., Paaver et al., 2008; Steinberg et al., 2008; von Diemen et al., 2008). Recently the BIS-11-A, an adaptation of the adult BIS-11, was developed for use with adolescents (Fossati et al., 2002). Analyses with the BIS-11-A reveal six first-order factors, which include Motor Impulsiveness, Cognitive Complexity, Self-Control, Lack of Delay, Attention, and Perseverance. However, intercorrelations among these factors for adolescents are considerably higher than those for factors of the BIS-11 with adults (Fossati et al., 2002). Therefore, it has been stated that the BIS-11-A total score may be the most appropriate index of impulsivity for research with adolescents, with a higher score reflecting greater impulsivity.

Some age and gender differences on the BIS have been found among adolescents. For example, older

compared to younger adolescents and males compared to females have shown higher impulsivity (Fossati et al., 2002; Stanford et al., 1996; Wittmann et al., 2008). Adolescents compared to college students have shown higher impulsivity on the BIS (Stanford et al., 1996). Of note, gender differences have not been consistently observed (nonsignificant gender differences: Li & Chen, 2007). Zaparniuk and Tayler (1997) have identified advantages and disadvantages of using the BIS with adolescents. Advantages includes that it is short and easy to administer, requires a third- or fourth-grade reading level, and provides a broad assessment of impulsivity with separate subscales. Identified disadvantages include the dependence on a subject's honesty and awareness of his or her behavior patterns.

EYSENCK IMPULSIVENESS SCALE

The Eysenck Impulsiveness Scale (Eysenck & Eysenck, 1980; Eysenck et al., 1984) is one of the scales that are part of the Junior I.6 Questionnaire, a widely used self-report measure. In conceptualizing the scale, the Eysencks (Eysenck, & Eysenck, 1978, 1980) distinguished impulsiveness from venturesomeness, and separate scales were constructed to assess these constructs. The Eysenck Impulsiveness Scale is a 23-item yes-no scale that is face valid for impulsivity (e.g., "Do you generally do and say things without stopping to think?"). A total score is created by summing the responses indicative of impulsivity. Norms are available for ages 8 to 15 (Eysenck et al., 1984). Scores have been found to rise between the ages of 8 and 15 and decline from the age of 16 (Eysenck & Eysenck, 1985). Gender difference findings have been somewhat inconsistent; girls have been found to report higher impulsivity (Heaven, 1991). Internal consistency is generally adequate (Eysenck et al., 1984; White et al., 1994). Zaparniuk and Tayler (1997) have identified advantages and disadvantages of the Eysenck Impulsiveness Scale; like the BIS, it is quick and easy to administer but requires a subject's honesty and awareness of his or her own behavior.

UPPS IMPULSIVE BEHAVIOR SCALE

In response to the lack of consensus on the definition of impulsivity, Whiteside and Lynam (2001) developed the UPPS Impulsive Behavior Scale. Drawing on the five-factor model of personality (e.g., Costa & McCrae, 1992), the researchers (Whiteside & Lynam, 2001) have partitioned this construct into four personality traits representing distinct pathways to impulsive behavior: (1) urgency

(U; i.e., compromised ability to resist impulses that are driven by negative affect), (2) premeditation (P; i.e., the tendency to think and reflect on the consequences of an act before engagement), (3) perseverance (P; i.e., the ability to sustain attention on a task that may be boring or difficult), and (4) sensation seeking (S; i.e., the tendency to enjoy and pursue activities that are exciting as well as being open to trying new experiences). This four-dimensional model has been replicated in a sample of teenagers (d'Acremont & Van der Linden, 2005). Specifically, d'Acremont and Van der Linden found, based on exploratory and confirmatory analyses, that the four-factor model is replicated in girls and boys and in the whole sample (mean age 15.57 years; SD 2.04). Girls had a higher score for urgency and boys had a higher score for sensation seeking. Although more work is needed (e.g., concurrent and predictive validity, further validation, understanding of which dimensions are most predictive of specific outcomes, such as lack of premeditation with antisocial actions and lack of perseverance with inattention or hyperactivity), the study suggests that the UPPS is a promising tool for studying impulsivity in adolescence.

In considering these different self-report measures, it is important to address their divergent validity from related constructs. One construct that is commonly confounded with impulsivity, especially within adolescent research, is sensation seeking. *Sensation seeking* refers to the tendency to seek out novel, varied, and highly stimulating experiences and the willingness to take risks in order to attain them (Zuckerman, 1979). Steinberg and colleagues (2008) help differentiate these two often interrelated constructs by showing that not all impulsivity leads to stimulating/rewarding experiences and not all sensation seeking is done impulsively (e.g., one can plan to engage in a sensation-seeking behavior like sky diving). Therefore, when considering measures of impulsivity, it is important to consider overlap. For example, one of the scales in the UPPS Impulsive Behavior Scale (Whiteside & Lynam, 2001) is Sensation Seeking. Likewise, in Zuckerman's commonly used Sensation Seeking Scale (SSS; Zuckerman et al., 1978), the Disinhibition subscale, which is typically defined in terms of behavioral disinhibition or undercontrol, involves a component of impulsivity. In order to increase conceptual and measurement purity, it is important for researchers to recognize that although impulsivity and sensation seeking can both be related to the same outcomes (e.g., risk taking behavior) and are often related to each other,

they are not the same conceptually and may indeed function independently in individual adolescents.

Behavioral Measures

Several behavioral measures have been used to measure impulsivity. Ivanov and colleagues (2008) described how the behavioral paradigms used to measure impulsivity and the various subconstructs resulting from these measures can be broadly divided into two categories: (1) those that measure impulsive choice or impulsive decision making and (2) those that measure impulsive action or motoric impulsivity. This categorization of tasks is also supported by work done by Reynolds and colleagues (2006) with healthy adult volunteers. Conducting a principal components analysis on data from four behavioral tasks, two components emerged, labeled *impulsive decision making* and *impulsive disinhibition* (Reynolds et al., 2006). Impulsive decision making includes measures that involve participants making decisions about delayed versus immediate or probabilistic versus definite outcomes, while behavioral disinhibition includes measures in which participants are expected to inhibit prepotent motor behaviors—measures that assess what is often termed *response inhibition* or *behavioral inhibition*.

IMPULSIVE CHOICE OR IMPULSIVE DECISION MAKING

The tasks that fall into this category measure the propensity for risky decision making when the participant is asked to choose between different rewards. The common principle among these tasks is that the subject has to choose between a safer strategy that will produce a greater final gain versus a strategy of bigger immediate wins paired with possible penalties that could result in a smaller final reward. Ivanov and colleagues (2008) stated that these tasks are developed to separate the cognitive components that underlie impulsivity, such as sensitivity to consequences as well as risk taking. Although multiple tasks exist to study impulsive decision making, common measures used with adolescents are the delay discounting procedures and the Balloon Analogue Risk Task.

The concept of delay discounting (Ainslie, 1975; Kirby et al., 1999) offers a well-known operationalization of impulsivity. The delay discounting model is based on the tenet that as a reward is delayed, its perceived value is systematically discounted, such that impulsive individuals prefer smaller immediate rewards over larger delayed rewards. Consequently, highly impulsive persons have the propensity to

choose the course of action that maximizes immediate gains, as they are either unlikely to take into account or give value to future gains or give up immediate satisfaction. A common means to assess delay discounting is by the delay discounting procedure, a paper/pencil version of the original monetary-choice questionnaire (Kirby & Marakovic, 1996). The questionnaire consists of a fixed set of 27 choices between smaller immediate rewards and larger delayed rewards (Monterosso et al., 2001). For example, participants are asked, “Would you prefer \$54 today or \$55 in 117 days?” Participants are instructed to show their preference by choosing one option. Previous research has shown that individuals’ discount curves are well described by the hyperbolic discount function (Mazur, 1987) $V = A/(1 + kD)$, in which V is the present value of the delayed reward A at delay D and k is a free parameter that determines the discount rate. As k increases, the person discounts the future more steeply. Therefore, k can be thought of as an impulsiveness parameter, with higher values corresponding to higher levels of impulsiveness. Relatively steeper discounting indicates that the point at which the participant prefers the immediate reward to the delayed reward occurs at lower values of the immediate reward and at shorter delay times (i.e., less preference for the larger delayed reward; Steinberg et al., 2009).

There are several versions of the delayed discounting procedure that are used with adolescents including: Question Based Delay Discounting Measure (DDQ; Richards et al., 1999) and Experiential Discounting Task (EDT; Reynolds & Schiffbauer, 2004). Delay Discounting tasks have been used primarily as a research tool. In terms of reliability and validity, over the trials, internal consistency of responding has been high (White et al., 1994). Findings from research with adolescents have demonstrated that delay discounting rates are elevated in adolescent smokers (Audrain-McGovern et al., 2004) and adolescents with ADHD (Barkley et al., 2001). As with other measures of impulsivity, gender differences have been somewhat equivocal; yet, a number of studies have reported nonsignificant gender differences (Reynolds et al., 2007, 2009; Steinberg et al., 2009). In terms of age differences, younger adolescents compared to those 16 and older have demonstrated greater willingness to accept a smaller reward delivered sooner than a larger one that is delayed (Steinberg et al., 2009).

The Balloon Analogue Risk Task (BART; Lejuez et al., 2002) concerns the behavioral measurement

of potentially impulsive risk taking. Within this laboratory paradigm, repeated performance of a risk-taking behavior is usually reinforced with monetary gain but sometimes (unpredictably) punished with monetary loss. The participant can choose to perform conservative behavior that terminates further risk taking and saves accumulated money. Specifically, the participant inflates a computer-generated balloon. Each pump is worth 1 point, but if the balloon is pumped past its explosion point, then all points accrued for that balloon are lost. The task was developed to provide a controlled setting in which to model risk taking in the natural environment, where risk taking up to a certain point leads to positive consequences, with further excessive risk taking leading to greater negative consequences that outweigh the positives. The BART has been found to have excellent reliability (test-retest) and validity (including convergent and divergent; Harrison et al., 2005). In an initial study with adults, BART responding was related to self-report measures of disinhibition (Lejuez et al., 2002). Subsequent adolescent studies using the BART have found relations similar to those reported in adult work (Aklil et al., 2005; Lejuez et al., 2005, 2007). Among adolescents, significant age and gender differences have not been shown on the BART (Lejuez et al., 2007).

IMPULSIVE DISINHIBITION

Behavioral disinhibition includes measures in which participants are expected to inhibit prepotent motor behaviors; these measures assess what is often termed *response inhibition* or *behavioral inhibition*. For example, the *stop and go task* (Logan et al., 1997) begins with the presentation of either an *X* or an *O* in the center of the computer screen. Subjects are instructed to press the *z* key when the *X* appears and the */* key when the *O* appears. The letters are presented at 2-second intervals, and reaction times (RTs) are recorded. On 25% of the trials (25% of the *X* trials and 25% of the *O* trials), a tone (stop signal) sounds after the presentation of the *X* or *O*. Subjects are instructed to refrain from pressing any keys when they hear the sound. The delay from the onset of the letter presentation to the onset of the tone (stop-signal delay) is systematically adjusted in 50-millisecond increments. If the subject fails to refrain from pressing a key after hearing the tone, the stop-signal delay is decreased by 50 milliseconds on the following stop-signal trial. If the individual successfully refrains, the stop-signal delay is increased by 50 milliseconds on the next trial. Eventually, the stop-signal delay will reach a duration at which the

subject will inhibit his or her key press responses on approximately 50% of trials. Individuals characterized by greater impulsivity should require a shorter delay to reach the point at which they are able to inhibit their responses 50% of the time. Related tasks targeting response inhibition have been developed and are commonly used with adolescents, for example, the go/no-go task (Newman et al., 1985) and the Conners' Continuous Performance Test-II (CPT; Conners, 2000; of note, one of the hallmarks of this task is the length that accentuates a decrement in attention and performance, with the number of errors increasing with fatigue). Of note, while these tasks conceptually fall under the umbrella of impulsive disinhibition, the go/no-go task is also thought to target passive avoidance (i.e., withholding a response to avoid punishment) and the CPT is thought to also target sustained attention.

The go/no-go task (Newman et al., 1985) is a learning task designed to assess the ability to inhibit inappropriate responses. In this task, participants are presented with eight numbers, of which four are designated correct and four incorrect. They are instructed to respond only to the correct numbers. They are often rewarded monetarily for correct responses (e.g., 10 cents) and penalized for incorrect responses. The outcome measures are errors of omission (withholding a response when a correct stimulus is presented) and errors of commission/false alarms (responding to an incorrect stimulus). Impulsivity on the task is defined as the number of errors of commission, which indicate an inability to inhibit inappropriate responses.

On the CPT, participants are instructed to respond as quickly as possible by left-clicking a computer mouse when a recurring target stimulus is present (e.g., any letter other than the letter *X*) and to refrain from responding to more rarely occurring nontarget stimuli (e.g., the letter *X*). The time between each stimulus (target and nontarget) is varied. Inattention is indicated by high numbers of omission errors (not responding to target stimuli, i.e., failing to press the key when the target is presented) and/or commission errors (responding to nontarget stimuli, i.e., pressing the key when the nontarget is presented).

Laboratory tasks targeting inhibitory control are commonly used in studies of attention-deficit/hyperactivity disorder (ADHD). For example, research has demonstrated that youth with ADHD have significantly more difficulty inhibiting their responses than do controls (for review, see Lijffijt et al., 2005). Further, task performance has been

found to correlate well with both laboratory and teacher ratings of interaction and hyperactivity (Pliszka et al., 1997). Few gender differences have been reported on response inhibition tasks (e.g., Dougherty et al., 2003; Liotti et al., 2007).

Limitations of Measurement

Lack of consensus about the definition of impulsivity has led to difficulty in its measurement (Gorlyn, 2005). Both self-report and behavioral tasks have some limitations. Impulsivity self-report scales exhibit low intercorrelations and are subject to response bias. Specifically, with self-report measures, participants must recognize and report on their own behavioral tendencies in various contexts relative to other individuals, and these self-perceptions may not always accurately reflect their behavior. Self-reports require insight or cognitive ability to understand questions in order to provide an accurate report of behavior. In other words, an impulsive individual may not always be sufficiently reflective to perceive his or her own impulsivity. In contrast, performance on behavioral tasks is potentially more objective and thus less sensitive to biased self-perceptions. On the other hand, the behavioral tasks typically measure only one specific dimension of behavior (e.g., the value of delayed rewards or response inhibition), which may have limited generalization to broader behavioral contexts or to the multidimensional construct of impulsivity.

Measure of Impulsivity: How Are They Related?

Although both self-report and behavioral measures have been studied extensively in separate research contexts, they are rarely used together in the same study, and relatively little is known about their relation to each other (Lane et al., 2003; White et al., 1994). The question arises as to whether self-report and behavioral tasks are measuring the same or different constructs. Findings from studies of adults are mixed; some studies indicate that performance on delay discounting tasks is correlated with self-report measures (Kirby et al., 1999; Richards et al., 1999; Swann et al., 2002). But in other studies, self-reports are not related to behavioral indices (Crean et al., 2000; Lane et al., 2003; Mitchell, 1999; White et al., 1994). There also is relatively little information about interrelations among different behavioral measures of impulsivity. Addressing this issue, several recent studies suggest that different behavioral measures may reflect separate underlying processes (as suggested above). For example, Lane

et al. (2003), using a principal components analysis to examine relations among behavioral measures in 32 healthy adult volunteers, found that behavioral inhibition was not related to intolerance of delays and concluded that delay intolerance and behavioral inhibition are separate processes.

Specific to adolescents, Reynolds and colleagues (2008) recently conducted a study designed to identify different dimensions of impulsive behavior in adolescents from a battery of laboratory behavioral assessments. In one analysis, correlations were examined between two self-report and seven laboratory behavioral measures of impulsivity. The correlation between the two self-report measures was high ($r = .41$) compared to the correlations between the self-report and laboratory behavioral measures (r values ranged from .02 to .21). In terms of relationships between behavioral tasks, all measures of discounting were positively correlated and the measures of sustained attention were positively correlated. However, performance on the response inhibition task was not correlated with any of the other laboratory behavioral measures. In a second analysis, a principal components analysis was performed with just the laboratory behavioral measures. Three behavioral dimensions were identified: (1) impulsive decision making, (2) impulsive inattention, and (3) impulsive disinhibition. Using the same sample, these dimensions were further evaluated with a confirmatory factor analysis, which did support the hypothesis that these are significant and independent dimensions of impulsivity. The researchers concluded that there are at least three separate subtypes of impulsive behavior when laboratory behavioral assessments are used with adolescent participants.

Thus, it seems that adolescents are similar to adults, both in terms of weak associations between self-report and laboratory behavioral assessments of impulsivity and in terms of the different dimensions of impulsive behavior identified with laboratory behavioral assessments. The fact that different dimensions of impulsivity appear to be identified by behavioral tasks may have significant implications for research involving conditions with impulsive characteristics (Reynolds et al., 2008). That is, specific subtypes of impulsive behavior (or a combination of subtypes) may be predictive of some clinical conditions and not others (e.g., ADHD, substance use). Currently, despite the recognized multidimensionality of impulsivity, most studies examining the construct focus on one dimension of the construct in isolation without considering the contributions of other components (for an exception, see Lane et al.,

2003). Thus, it is difficult to speculate on the generalizability of the results across other dimensions of impulsivity and, more importantly, on how specific components of impulsivity are related to specific outcomes. It may be that correlations between self-report measures and behavioral tasks would be greater if the tasks measured more general impulsive behaviors or if the questionnaires assessed the specific processes identified by the behavioral procedures. Future work should (1) assess correlations among measures across different dimensions of impulsivity in order to support the notion of impulsivity as a multidimensional construct, (2) have a clear understanding of why one dimension is chosen for a particular research question, and (3) delineate the core conceptual factors and their specific contributions to the variety of impulsive behaviors.

Now that the limitations of the measurement and definition of impulsivity have been recognized, the remainder of the chapter will be devoted to the presence of impulsivity in adolescence both in normative development and in psychopathology.

Developmental Course

Although there is individual variability in the trait of impulsivity across the lifespan, it is also important when examining this variable in adolescents to understand normative development. In considering the developmental course of impulsivity, studies of age differences in impulsivity that span adolescence and adulthood are relatively rare. Yet, the available research suggests that, in general, impulse control continues to mature over the course of adolescence and early adulthood. For example, in a sample of individuals ranging in age from 7 to 29, Galvan and colleagues (2007) reported a significant negative correlation between chronological age and impulsivity (using the Connors Impulsivity Scale). Similarly, Leshem and Glicksohn (2007) reported a significant decline in impulsivity from ages 14–16 to 20–22 on the Eysenck Impulsiveness Scale and the BIS. This was recently replicated in a large socioeconomically and ethnically diverse sample of individuals between the ages of 10 and 30 (Steinberg et al., 2008). Specifically, using both self-report and behavioral measures, impulsivity followed a linear pattern, declining steadily from age 10 on (a note of caution: these are cross-sectional findings). Delay discounting was also examined in this sample; results demonstrated that younger adolescents were more willing to accept a smaller reward delivered sooner than a larger delayed one than were individuals age 16 and older (Steinberg et al., 2009).

Similar delay discounting findings have been found with other between-group cross-sectional comparisons of young adolescents (age 12 years), young adults (average age 20 years), and older adults (average age 70 years). These studies demonstrate that young adolescents discount monetary rewards more steeply than young adults, who discount at a faster rate than older adults (e.g., Green et al., 1994). Together these findings suggest a decline in impulsivity from childhood through adolescence and into adulthood.

These findings parallel evidence showing structural and functional maturation over the course of adolescence in brain regions that subserve impulse control and other aspects of self-regulation (Paus, 2005). The development of the prefrontal cortex is believed to play an important role in the maturation of higher cognitive abilities such as decision making and cognitive control, processes inherently related to impulsivity (Casey et al., 1997a, 2002). A number of paradigms, including response inhibition tasks, have been used, in conjunction with functional magnetic resonance imaging (fMRI), to assess the neurobiological basis of these abilities (Casey et al., 1997b, 2000; Durston et al., 2003). Together these studies show that children recruit distinct but often larger, more diffuse prefrontal regions when performing these tasks than do adults. The pattern of activity becomes more fine-tuned with age, with regions not correlated with task performance diminishing in activity with age (see Casey et al., 2008, for review).

In considering these findings in relation to observed increased risk-taking behavior in adolescence, recent neuroimaging studies have examined reward-related processing (e.g., Bjork et al., 2004; Ernst et al., 2005; May et al., 2004), focusing primarily on the region of the accumbens (a portion of the basal ganglia involved in predicting reward; Casey et al., 2008) in conjunction with top-down control regions (i.e., prefrontal cortex). For example, Galvan et al. (2006) examined behavioral and neural responses to reward manipulations across development. Results suggested enhanced accumbens activity to rewards in adolescents relative to children and adults. Adolescents showed an exaggerated accumbens response in anticipation of reward. Further, both children and adolescents showed a less mature response in prefrontal control regions than adults. The authors concluded that these findings suggest different developmental trajectories for these regions that may underlie the enhancement in accumbens activity and, in turn,

relate to the increased risky behaviors observed during this period of development.

Thus, two core neurobiological networks appear to underlie human decision making and are important in the emergence of risky behavior observed in adolescence (Steinberg, 2004). The first, a cognitive control system, which consists of prefrontal and parietal regions, as well as the anterior cingulate, facilitates executive functioning. The second, an affective system, includes regions that are important to processing reward and social and emotional salience, including but not limited to the amygdala, ventral striatum, orbitofrontal cortex, medial prefrontal cortex, and superior temporal sulcus. Empirical evidence supports the notion that it is the affective network that dominates during adolescence, with heightened reward sensitivity throughout this developmental period (e.g., Green et al., 1999).

The emergence of this affective network at puberty, with the cognitive control network developing across a longer developmental trajectory (Steinberg, 2004), further supports the dominance of the affective system in adolescent decision making and increasing risky behavior. This dissociation between an affective and a cognitive system has also been conceptualized as a dissociation between an activational system and an inhibitory system, with delayed development of the inhibitory system and the dominance of the activational system being responsible for the onset of risky behaviors (e.g., Bardo, 2004; Chambers et al., 2003). In sum, the staggered development of these two brain regions is thought to result in heightened reward drive and cognitive disinhibition (Gullo & Dawe, 2008).

Impulsivity and Adolescent Dysfunction

In considering impulsivity's role in abnormal behavior, the current psychiatric diagnostic system, the revised fourth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR; American Psychiatric Association, 2000), lists impulsivity as a symptom of several disorders evident in adolescence (e.g., attention-deficit/hyperactivity disorder, conduct disorder, substance use disorders) as well as diagnostic categories that are characterized as adult disorders (e.g., borderline personality disorder and antisocial personality disorder). In addition to specific diagnoses, impulse control is related to a number of other problem behaviors in adolescence, including suicide. The following discussion concerns some of the areas

in which impulsiveness in adolescent behavior has been implicated in psychopathology.

Attention Deficit Hyperactivity Disorder

Attention-deficit/hyperactivity disorder (ADHD) is characterized by developmentally inappropriate inattention and impulsivity that causes impairment in social and academic functioning. Fewer studies of adolescents with ADHD have been conducted than with school-age children, but much of what is known about children with ADHD can be extrapolated to adolescence. Barkley (2004) notes that there is no compelling evidence that ADHD symptoms in adolescents are different from those in children with the disorder, although adolescents tend to display fewer symptoms of overt hyperactivity than their school-age counterparts. The impairments and consequences associated with these symptoms, however, do change and often become more serious during adolescence.

It has been theorized that the symptoms of ADHD arise from a primary deficit in executive functioning. There are a number of theories that attempt to elucidate the precise nature of this deficit; Barkley's theory of behavioral disinhibition stands out as being the most highly developed and widely tested. Barkley theorizes that inhibition is primary to other executive functions in that a response must be inhibited long enough to allow other executive functions to occur (Barkley, 1997a, 2001). Inhibition, according to Barkley, encompasses the processes of response inhibition (inhibiting a prepotent response or stopping an ongoing response) and interference control. The inhibitory deficit found in individuals with ADHD causes those with the disorder to behave impulsively and to have difficulty discontinuing actions that others are able to easily stop. Problem-solving tasks in which no preconditioned response is immediately available are particularly reliant on the process of response inhibition, as are tasks requiring resistance to temptation or deferred gratification.

Barkley's theory of response disinhibition has been studied extensively in laboratory settings. Individuals with ADHD have consistently been found to perform poorly relative to controls across multiple studies on tasks such as the stop-signal task (Barkley, 1997a; Fischer et al., 2005; Nigg, 1999; Oosterlaan et al., 2000; Schachar et al., 2000). Further, those with ADHD have been found to show a preference for smaller, sooner rewards over larger, later rewards in delay discounting paradigms (Luman et al., 2005). Of note, ADHD groups

continue to perform poorly relative to control groups when comorbid conduct disorder is excluded (Nigg, 1999).

Substance Use Disorders

Despite intensive public health efforts to inform adolescents about the dangers of substance abuse and thereby reduce the incidence of usage, substance use remains an issue of grave importance among today's youth. The Monitoring the Future (MTF) surveillance of risk behaviors in youth (Johnston et al., 2006) estimates that 39% of 10th graders have smoked cigarettes, 63.2% have used alcohol, and 42.1% have used an illicit drug. Adolescence is a critical developmental period in which there is a rapid increase in substance use initiation and subsequent misuse (Kelley et al., 2004). Considerable concern exists about the potential outcome of such early substance use, as adolescent users run the risk of engaging in more dangerous use of illicit substances as they enter adulthood (Chen et al., 1996) as well as future impairments in social functioning and physical and mental health (Friedman et al., 2004; McGue & Iacono, 2005). Adolescent problematic substance use is also associated with other dangerous behaviors, including condom nonuse that risks exposure to human immunodeficiency virus (HIV; Brook et al., 2004), comorbid psychopathology (Galaif et al., 1998), suicidal ideation and/or attempts, and deliberate self-harm (Hufford, 2002).

Impulsivity has been linked to substance use vulnerability, frequency, severity (including social and emotional consequences), and dependence (Allen et al., 1998; Fishbein et al., 1989; King et al., 1991; Moeller & Dougherty, 2002; Patton, 1995; Petry, 2001) and has been considered as part of a larger externalizing spectrum that shows a clear relationship to substance use and related psychopathology, including antisocial behavior (Krueger et al., 2002). Specific to adolescents, one of the key findings is that measures tapping the impulsivity construct are associated with both the initial use and later development of substance abuse in adolescents (Gullo & Dawe, 2008). Research suggests that disinhibition predates the development of substance abuse (Sher & Trull, 1994). For example, higher impulsivity is associated with the onset of alcohol consumption at earlier ages (Kollins, 2003; von Diemen et al., 2008). Further, impulsive individuals are known to be at higher risk for substance-related disorders (Coffey et al., 2003; Mitchell, 2004; Poikolainen, 2000).

Impulsivity may influence which adolescents continue to use substances and develop dependence disorders as young adults in multiple ways. For example, longitudinal studies of early alcohol use suggest that young adults who continue to be problem drinkers are more likely to have been rebellious, nonconformist, deviant, or isolated during high school (Newcomb, 1997). School failure has been shown to increase the likelihood that a child will use drugs, with some reports indicating failure at the elementary school level and others pinpointing academic difficulties in later grades (Haynes, 1998). If a child has had problems with emotional distress and impulsive behavior in the early years and has not adapted well to the school environment, cumulative risk may mediate his or her affiliation with a deviant or delinquent peer group that does not adhere to conventional societal norms, accepts drug use and early sexual involvement, engages in disruptive or delinquent behavior, and/or pressures its members to continue drug use. Membership in a deviant peer group may further influence the child's attitudes about school, drug use, social conventions, and social expectations, further altering the trajectory toward initiation and later dependence. Alternatively, a child who has had several negative responses to school during the early years may simply be rejected by adolescent peers, becoming socially isolated and alighting on a different but equally adverse trajectory toward initiating substance use.

Conduct and Externalizing Disorders

Conduct disorder refers to a group of behavioral and emotional problems and is part of the antisocial spectrum. Antisocial behavior in adolescence is predictive of later antisocial behavior and adverse outcomes in adulthood. In individuals with antisocial personality disorder, antisocial behavior manifests in childhood as conduct disorder and continues throughout adulthood (Gelhorn et al., 2007). Children and adolescents with conduct disorder have great difficulty following rules and behaving in a socially acceptable way. Children or adolescents with conduct disorder may exhibit some of the following behaviors (American Psychiatric Association, 2000): aggression to people and animals (e.g., bullies, threatens or intimidates others), destruction of property (e.g., deliberately destroys others' property), deceitfulness, lying, or stealing (e.g., has broken into someone else's building, house, or car), and serious violations of rules (e.g., often stays out at night despite parental objections). Many children with a conduct disorder

may have coexisting conditions such as mood disorders, anxiety, posttraumatic stress disorder, substance abuse, ADHD, and learning problems. Impulsivity has long been viewed as a critical element of disruptive behavior disorders in children and adolescents. Impulsivity is correlated with aggressive (Bettancourt & Miller, 1996; Hollander et al., 2002; Seroczynski et al., 1999) and violent behavior. For example, Seguin and colleagues (1995) found that aggressive boys performed more poorly than controls on tasks assessing disinhibition.

Links between inhibitory deficits and antisocial behavior may be evident in individuals who cannot stop their actions when circumstances indicate that it is no longer appropriate to act. They may lack the ability to inhibit their impulses or an inappropriate loss of temper in response to a provoking stimulus, which may lead to violent responses (Hawkins & Trobst, 2000). Moffitt (1993) argued that an impulsive personality style serves to maintain antisocial behavior across the lifespan through a variety of person–environment interactions. Accordingly, impulsivity increases the risk of long-term antisocial behavior through direct and indirect means. In terms of direct pathways, deficits in impulse control produce delinquent behavior by interfering with adolescents' ability to control their behavior and to anticipate the future consequences of antisocial acts. In terms of indirect pathways, deficits in impulse control may also lead to delinquency by disrupting adolescents' success in school, leading to termination of education with the attendant lack of job opportunities and, thus, economic disadvantage. Consequently, the adolescent and young adult lacks opportunities to succeed in conventional ways and thus may be more likely to rely on the rewards associated with antisocial activities. Together these developments lead to "life-course-persistent" antisocial behavior (Moffitt, 1993).

Theoretical and empirical research consistently shows that various types of problem behaviors co-occur in adolescents. It has long been noted that substance use and delinquent behaviors occur together (Elliott et al., 1985; Welte & Barnes, 1985). This co-occurrence of behaviors has been labeled *problem behavior syndrome* (e.g., Donovan & Jessor, 1985; Jessor & Jessor, 1977) and is considered an externalizing factor. Krueger (1999), in a national survey of subjects ages 15–54, showed that alcohol dependence, drug dependence, and antisocial personality disorder formed a single externalizing factor, distinguished from a separate (although correlated) factor representing internalizing disorders. Considering this

coherent spectrum of externalizing disorders, these disorders are likely linked at an etiological level. Impulsivity may serve as the common underlying mechanism.

Borderline Personality Disorder

Borderline personality disorder (BPD) is a serious mental health problem with great public health significance. It is found at rates of 1%–3% in the general population (Swartz et al., 1990; Torgerson et al., 2001). Borderline personality disorder is associated with severe functional impairment (Skodol et al., 2005), high rates of co-occurring psychiatric disorders (including mood, anxiety, substance abuse, and eating disorders; McGlashen et al., 2000), and an elevated risk for completed suicide (occurring at a rate of 10%; Work Group on Borderline Personality Disorder, 2001).

Historically, identification of BPD-related pathology in children and adolescents was discouraged; yet, recent findings highlighting the early developmental origins of BPD suggest the importance of examining potential antecedents of BPD earlier in the lifespan (Crick et al., 2005). For example, retrospective studies of adult patients with BPD indicate that many had experienced BPD-related symptoms long before they were diagnosed with the disorder in late adolescence/young adulthood (Zanarini et al., 2001), and clinical literature indicates that many patients with BPD describe significant psychiatric difficulties prior to adolescence (Paris, 2003). Theories of BPD have emphasized the role of particular personality traits in the pathogenesis of this disorder. In particular, researchers have suggested that BPD likely results from the interaction of emotional dysregulation (e.g., emotional lability) and disinhibition (e.g., Nigg et al., 2005). That is, during highly emotionally aroused states, in general, individuals are more likely to act quickly and sometimes impulsively, with less regard for consequences. Individuals with BPD have more emotion regulatory difficulties and are more often in states of high emotional arousal. Studies have found that adults with BPD report heightened levels of impulsivity (e.g., Bornovalova et al., 2006; Henry et al., 2001; Hochhausen et al., 2002).

Suicide

Studying suicide in adolescents is imperative, as within the past 60 years, the rate of suicide for people between the ages of 15 and 24 has dramatically increased, doubling for females and quadrupling for males (American Association of Suicidology,

2004). Suicide is the third most common cause of death in adolescence (CDCP, 2004). Impulsivity has consistently been identified as an important factor in suicide risk among studies that examine correlates of suicidal behavior. For example, higher levels of impulsivity have been related to various indices of suicidal behavior, including suicidal ideation (Hull-Blanks et al., 2004) and suicide attempts (Dougherty et al., 2004). Longitudinal support is also available. For example, Pfeffer et al. (1995) conducted a longitudinal study on the role of impulsivity in child and adolescent suicide attempts among psychiatrically hospitalized children followed up 6 to 8 years later. Children and adolescents with a good course (i.e., had no history of suicidality before study entry and did not make a suicide attempt during the follow-up period) were found to have significantly better impulse control than those with an improved (i.e., reported suicidal ideation or behavior before study entry but demonstrated no suicidality during the follow-up period) or poor (i.e., made a suicide attempt during the follow-up period) course. In other prospective work, impulsivity has been identified as a trait predicting further suicidal acts (Oquendo et al., 2004).

Although the relationship between impulsivity and suicidal behavior has been well documented in the literature, the mechanism by which impulsivity confers risk is not entirely clear (Witte et al., 2008). One hypothesis is the stress-diathesis model of suicidal behavior in which the hypothetical diathesis (i.e., the tendency to experience more suicidal ideation and to be more impulsive), in the presence of a stressor (i.e., mental illness), makes people more likely to act on suicidal feelings (Mann et al., 1999). Thus, according to the stress-diathesis model, impulsivity is an important diatheses for suicidal behaviors by predisposing one to act on suicidal feelings (Mann et al., 1999). Thus, impulsivity plays a proximal role in that it is thought to lead to a disinhibited state that is more conducive to impulsive suicidal behavior (Witte et al., 2008). In contrast, Joiner's theory (Joiner, 2005) posits that the relationship between impulsivity and suicide is indirect; impulsive individuals may be more likely to have experiences associated with the process of acquiring the capability to die by suicide (i.e., painful/provocative experiences). The theory posits that impulsivity alone does not necessarily increase the likelihood that someone will impulsively engage in suicidal behavior in the face of a stressor, but rather that an individual's impulsivity level leads to attainment of the capability for suicide (i.e., through exposure to painful/provocative stimuli) that makes suicidal behavior possible should the

desire arise (Witte et al., 2008). More research is needed to clarify the mechanisms by which impulsivity confers the risk for suicide.

Conclusion

In 1993, Fink and McCown wrote a chapter on impulsivity in adolescents, focusing on its measurement, causes, and treatment. They concluded the chapter by listing unanswered questions regarding impulsivity in children and adolescents: "How does one define the construct of impulsivity? What is the most appropriate way to assess impulsivity? Why do some children and adolescents evidence impulsive behaviors?" (p. 302). Reflecting on these questions almost two decades later, it is clear that some progress has been made. For example, impulsivity is now better understood and measured as a multidimensional construct. There is now more understanding of how self-report and behavioral tasks relate and do not relate to each other and within each category. Seminal work has been done on brain regions that subserve impulse control and other aspects of self-regulation—namely, the identification of the staggered development of affective and cognitive control brain regions that are linked to heightened reward drive and cognitive disinhibition in adolescents (Gullo & Dawe, 2008).

Future Directions

A number of future directions are indicated in the domains of measurement, larger models, and intervention. In terms of measurement, as mentioned above, future work would benefit from assessing correlations among measures across different dimensions of impulsivity in order to explore the notion of impulsivity as a multidimensional construct and to delineate the core conceptual factors and their specific contributions to the variety of impulsive behaviors. As measurement improves, future work should attempt to build larger models that incorporate variables such as emotion and context. Research suggests that maladaptive styles of emotional coping and impulsivity interact with the experience of negative emotions to predict the general propensity to engage in risky or problem behaviors (Cooper et al., 2003). Research is needed to address questions such as this: Are highly impulsive individuals, and those who rely on dysfunctional styles of coping with negative emotions, less able to regulate their negative mood states effectively, thus leaving them more vulnerable to the immediate relief promised by various risky behavioral choices? Further, developmental models argue that characteristics of an

adolescent's environment are expected to mitigate or exacerbate the manifestation of individual traits as risk factors. Thus, how do environmental factors (e.g., the neighborhood context) influence the relationship between impulsivity and outcomes in adolescence? One could hypothesize that the relationship between impulsivity and an outcome variable such as risk-taking behavior varies as a function of neighborhood context such that impulsivity exerts more influence in neighborhoods with "hot spots" for alcohol, drugs, and violence than in better-off neighborhoods. Finally, a fertile area for future work is in intervention strategies for impulsivity. It will be important to develop interventions to increase youths' awareness of and regulation strategies for impulsivity while simultaneously offering alternative sources of reinforcement through engagement in healthy, prosocial, and novel activities.

Yet, perhaps most pressing in the field are studies addressing the basic developmental mechanisms of the relationships among domains such as reward seeking, impulsivity, and consequence appraisal and how there may be a normative risk-taking window for all adolescents. As described above, impulse control has been found to continue to mature over the course of adolescence and early adulthood. These findings parallel evidence showing structural and functional maturation over the course of adolescence in brain regions that subserve impulse control and other aspects of self-regulation (Paus, 2005). Normative heightened vulnerability to risk taking in adolescence has been proposed to be due to the combination of a relatively high inclination to seek excitement and a relatively low capacity for self-control that are typical of this period of development (Steinberg et al., 2008). Yet, there is also individual variability in the trait of impulsivity across the lifespan, as observed in the descriptions of impulsivity in adolescent psychopathology. Studies are needed to address whether or not impulsivity assumes a maladaptive cast with a greater likelihood of psychopathology depending on a combination of experiential, environmental, and genetic factors. Research that (1) examines multiple pathways to maladaptive impulsivity, (2) identifies protective factors that can alter adverse trajectories, and (3) tests hypothetical causal mechanisms of risk is likely to be the most fruitful

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Abstract

Impulse control disorders (ICDs) are not well studied in the elderly, as the development of ICDs tend to decrease with age. Although less prevalent than younger patients, older adults with ICDs—psychological gambling in particular—may have unique assessment and treatment challenges as a result of their age, elaborate social community, comorbid medical conditions, and attitudes about mental health treatment. This chapter discusses the phenomenology of excessive gambling and other ICDs in elders, unique risk factors for this older population, and some potential treatment options. The chapter concludes with some thoughts on the future directions for research in this field.

Keywords: impulse control, gambling, substance abuse, older adults, assessment, treatment

Introduction

Adults over age 65 represent a large and growing demographic group in the U.S. population (Administration on Aging, 2009), and health concerns in this population are becoming increasingly important to understand. Impulse control disorders (ICDs) in the elderly are not well studied. However, as these disorders are also increasingly recognized as a source of considerable disease burden, it is important to understand what effect they may have on the health of elderly individuals. Older adults are clearly a key target market for the gambling industry (NOR Center, 1999). However, relatively little is known about the effects of gambling on older adults, and the evidence for the effects of other ICDs is even more scarce. This chapter will first review what is known about the prevalence and risk factors for problem and pathological gambling, as well as other ICDs, in the elderly. Second, the relatively sparse data on the health effects of gambling and other ICDs on the elderly will be described. Finally, treatment issues that may differ among, or be unique

to, older versus younger adults with ICDs will be explored.

Prevalence Estimates for ICDs in the Elderly

Problem and Pathological Gambling

There are two sources of data on the relationship between age and problem/pathological gambling: the Gambling Impact and Behavior Study (GIBS; NOR Center, 1999) and the National Epidemiologic Study of Alcohol and Related Conditions (NESARC; Grant et al., 2003a). Both studies are national community studies that include extensive information on gambling. Both studies divided respondents into categories based on gambling experiences: those who had not gambled, those who had gambled and experienced none of the DSM-IV symptoms of problem and pathological gambling (sometimes referred to as *recreational gambling*), those who had experienced one or two symptoms, and those who had experienced three or more symptoms. The latter category is often referred to as *problem/pathological*

gambling since five or more symptoms are required for a diagnosis of pathological gambling (American Psychiatric Association, 2000).

Prevalence rates for disordered gambling decrease as age increases. In the GIBS, those over age 65 had the lowest rates of pathological gambling (0.2% versus 0.3%–0.9% for younger age groups) and among the lowest rates of problem gambling (0.6% versus 1.0% for those ages 18–29; NOR Center, 1999). The NESARC study found a prevalence rate of 0.3% for problem/pathological gambling for those over 65 compared to 0.6% for those under age 65 (Desai & Potenza, 2008).

Other ICDs

There are few reliable population-based estimates of the prevalence of other ICDs. The NESARC study found a lifetime prevalence of 1.13% for fire setting (Blanco et al., 2010), and the National Comorbidity Study Replication found a prevalence of 5.4% for intermittent explosive disorder (IED; Kessler et al., 2006). In both studies, the rates are much lower in older adults. For example, only 4% of fire setters were over age 65, and the prevalence of IED in respondents over age 60 was 2.1% less than half the population rate. The prevalence of kleptomania has been estimated at 0.6% in adults (Goldman, 1991); however, no data on those over 65 are available. It would be reasonable to expect that the rate would be substantially lower among older adults, as this would be consistent with patterns seen for addictive disorders such as drug and alcohol abuse/dependence (Compton et al., 2007; Hasin et al., 2007). In addition, many of the ICDs are recognized as disorders of adolescence or early adulthood (Coccaro et al., 2004; Kessler & Wang, 2008), probably in part because so few cases are seen among elderly patients.

One group of disorders, hoarding and Diogenes syndrome, though not categorized as ICDs, share some features with disorders that are conceptualized to fall on an impulsive-compulsive spectrum (Jefferys & Moore, 2008; Reyes-Ortiz, 2001). These problematic behaviors, including problematic shopping, Internet addiction, and sexually compulsive behavior, have features of both impaired impulse control that results in initiation of the behavior and compulsive urges that serve to maintain the behaviors over time, even in the face of adverse consequences to health or functioning (Grant & Potenza, 2006). Hoarding behavior may also have both impulsive and compulsive features (Snowdon et al.,

2007) and may be associated with problematic shopping (Winsberg et al., 1999) or with kleptomania (Maier, 1997). Hoarding is not considered an ICD. However, it is worth mentioning here because it is more often seen in older adults than in younger adults and may be incompletely understood in the elderly.

Phenomenology

There are no data indicating that the expression of ICDs is markedly different in older adults than in younger adults. However, this may be due to limited research. Given that prevalence rates are so much lower in older adults, and that these disorders are unlikely to have a substantial impact on mortality, it is likely that, consistent with other addictive behaviors, individuals may “grow out” of an ICD as they age. It could thus be hypothesized that those disorders that persist into older age, or have a later age of onset, may be substantially different in nature (e.g., secondary to a decline in brain function with age) or expression (e.g., severity), or may be accompanied by different comorbid disorders. For example, it is possible that the presence of personality disorder may reduce the likelihood of growing out of an ICD over time. Future research on the nature of ICDs in the elderly will be helpful in determining whether these disorders have a different course or expression in older adults.

Problem and pathological gamblers experience many of the same symptoms as people with other addictions: tolerance (the need to gamble more to achieve the same effect), symptoms of withdrawal, and loss of control over the addictive behavior (Potenza, 2006; Wareham & Potenza, 2010). This pattern has not been demonstrated to be unique among elderly gamblers (Custer, 1984).

The development of problem gambling has been suggested to follow a predictable course, described by Custer (1984), that starts with a “winning phase,” progresses to a “losing phase,” and ends with a “desperation phase,” which continues either indefinitely or until extreme events force a change in gambling behavior. There is little indication that the course is substantially different among the elderly (Custer, 1984). However, there may be some correlates of gambling difficulties that are different in the elderly. These include sources of income, spending behaviors, borrowing behaviors, and engagement in criminal behavior (Desai, 2004).

The financial repercussions of problem gambling may be more severe in the elderly. Older adults are

more likely to be retired and surviving on annuity income, savings, and investment income. Since they are less likely to have employment income, either the patterns of spending on gambling or the impact of that spending may change. Older adults with no employment income have more difficulty recovering from financial losses and may be more likely to exhaust their savings, cash in investments, and spend annuity income than younger gamblers (Desai et al., 2004; Pavalko, 2002). The amount of money spent by a younger working individual may have less overall impact than the same amount spent by a retired gambler with limited ability to recover from a loss.

While the effects of financial losses may be more noticeable in the elderly, the effects of gambling on work and family relationships may be less pronounced. Difficulties related to either work or family relationships, which are symptoms included in the DSM diagnosis of pathological gambling, may be less relevant for older adults who are retired and may be widowed. For example, retired gamblers are less likely to commit work-related white-collar crimes to obtain gambling money. They may also have fewer family members than younger gamblers from whom to borrow money to continue gambling (Desai, 2004). Since there may be less opportunity to engage in certain behaviors that constitute symptoms of pathological gambling, the impact of gambling on these aspects of life for older adults may appear underestimated.

Unique Risk Factors for ICDs

With the exception of pathological gambling, there has been very little research on risk factors for ICDs. Genetic research has shown that there is both a genetic and an environmental component to these disorders (Shah et al., 2004), so family history is a clear risk factor for many reasons. Imaging studies have found similar brain function and dysfunction in people with ICDs and with other addictive disorders (Shah et al., 2004), so it is likely that these groups of disorders would have risk factors in common. However, none of these risk factors is likely to be different in older adults than in younger ones.

The one area where there is moderate evidence for unique risks to older adults is among those with cognitive decline and those being treated with medications that affect the dopaminergic systems in the brain. Dopamine agonists have been used for many years as a front-line treatment for Parkinson's disease (Fan et al., 2009) and they are increasingly used for other purposes, such as treatment of restless leg syndrome

(Abler et al., 2009) and fibromyalgia (Holman, 2009). Case reports, and more recently larger clinical studies, have found that these drugs associate with impaired impulse control, including ICDs (Dodd et al., 2005; Giladi et al., 2007; Isaias et al., 2008; Stamey and Jankovic, 2008). Since the biologies of these disorders are different, and since symptoms may remit quickly after the medications are discontinued, it has been suggested that the symptoms are directly related to the effect of the medications on reward systems in the brain (Drapier et al., 2009; Holman, 2009). Restless legs syndrome and Parkinson's disease are also disorders almost exclusively of older adults. Therefore, they may constitute a risk factor unique to this demographic group.

There are a number of possible risk factors for the onset of problem and pathological gambling, and some of these risk factors may also be either stronger in or unique to older adults. These include gambling opportunities and the marketing practices of the gambling industry, social isolation, depression and anxiety, and biological changes that are associated with both older age and gambling behavior (Desai, 2004).

Research has suggested that opportunity plays an important role in the prevalence of problem and pathological gambling. All age groups have been impacted by the increased availability of state-sponsored lotteries and the expansion of casinos. However, since retirees have more disposable time, their opportunities to gamble are thereby increased (McNeilly & Burke, 2002). States that have large numbers of retired citizens (e.g., Florida, Arizona) have seen particularly steep increases in the availability of gambling activities, and many such venues have incentive programs targeted specifically to elders (e.g., free bus rides, discounted meals). Finally, many extended-care facilities offer various forms of gambling (e.g., bingo games for money or casino day trips) to promote social interaction and activity. All of these increases in the opportunity to gamble, which are relatively unique to older adults, may place this age group at greater risk for the development of gambling-related problems.

As adults age, they often experience the loss of traditional social roles associated with being parents, having employment, and being married (McNeilly & Burke, 2002; Pavalko, 2002). The loss of such roles, along with the increased likelihood that children will not live nearby, can lead to feelings of social isolation, boredom, and even depression and anxiety. Gambling may help to alleviate these feelings by providing

entertaining sensory stimulation, a chance to socialize, and a chance to escape from everyday problems and feelings (McNeilly & Burke, 2000, 2001, 2002; Pavalko, 2002), although there is no direct evidence that geriatric depression leads to gambling (Grant et al., 2001).

In addition, while elderly people may be restricted in the types of physical activity they are capable of performing, gambling venues are handicapfriendly, and many forms of gambling (e.g., slot machines) are relatively passive forms of entertainment requiring little cognitive ability (McNeilly & Burke, 2002). This is cause for some concern, particularly since slot machines appear to be a favorite of older casino gamblers (Grant et al., 2001), and some research has shown that progression to addiction is particularly fast among slot machine players (Breen & Zimmerman, 2002).

It is unlikely that individuals with advanced forms of dementia are capable of gambling. However, early forms of dementia and other forms of cognitive decline may place older adults at greater risk for gambling-related problems by reducing their ability to weigh risks, impairing the memory of past losses or the ability to determine cause and effect, or causing paranoid or magical thinking that could affect gambling behavior (Grant et al., 2001). For example, there have been anecdotal reports of excessive sweepstakes participation among patients with cognitive dementia (Mendez et al., 2000).

Health Correlates of ICDs

The health correlates of ICDs are understudied in all age groups. Some research has begun to explore associations with other psychiatric disorders such as depression, obsessive-compulsive disorder, and personality disorders (Afifi et al., 2010; Dell'Osso et al., 2006; Winslow et al., 2010); however, there has been no exploration of these correlates specifically among older adults. Since older adults are more likely to be living with chronic diseases such as diabetes and heart disease, future research should examine whether such physical conditions have an effect on ICDs. At a minimum, the presence of comorbid chronic conditions and ICDs may complicate treatment efforts for both disorders.

The health impact of gambling, whether recreational or pathological, has been examined in slightly more detail. Early research suggested that gambling increased elderly individuals' self-esteem (Campbell, 1976) by allowing them to participate more fully in a society that tended to hide them away and exclude them from everyday activities.

More recent data from the GIBS found that older gamblers reported having better subjective health than their nongambling peers (Desai et al., 2004), suggesting that gambling activities may confer some limited health benefits if practiced responsibly. Later data from the NESARC study also found that older gamblers were significantly more likely to report that their health was excellent compared to that of nongamblers; they also reported having better daily physical and mental functioning (Desai et al., 2007). However, they were also more likely than nongamblers to be smokers, to have alcohol abuse/dependence, and to be obese. Taken together, these data indicate that those elders who feel better and have better daily functioning are more likely to gamble, but that they also have a significant risk of developing poor physical health secondary to poor health behaviors.

There may be some health effects of gambling that are unique to the elderly, particularly those who engage in casino gambling. First, there may be poor health effects associated with sitting for long periods of time, often in smoke-filled environments, eating either less frequently or larger amounts than normal, and participating in games that increase heart rates and excitement levels. While none of these factors would be an immediate concern for younger gamblers, they may be of greater concern for elderly gamblers who may have diabetes, heart disease, or otherwise poor circulation.

Second, it is possible that the health consequences of large financial losses may be greater among the elderly, who have limited ability to recoup those losses through work. Large financial losses may be associated with consequences such as poor health management due to the inability to purchase medications, loss of independence due to inability to live on less money, or increased social isolation resulting from borrowing money or strained family relations resulting from gambling. However, no data have directly assessed these hypotheses, and further examination is warranted.

Third, one group of the elderly, women, may be at particularly high risk for the development of gambling-related problems and health problems related to gambling. Women make up a majority of the elderly due to their longer life expectancy, and their proportion will likely continue to increase as the population ages. Older women may be at even greater risk for the above-mentioned health effects due to having, in general, even lower incomes than older men, a higher likelihood of being widowed and thus socially isolated, and a higher likelihood

than elderly men of living with chronic diseases such as diabetes and hypertension (Administration on Aging, 2009). There is also some evidence that women of all ages may be more vulnerable than men to the phenomenon of telescoping, in which they begin gambling later in life but develop gambling-related problems faster than do men (Potenza et al., 2001; Tavares et al., 2001), though this has not been a consistent finding (Grant et al., 2001).

Assessment and Treatment of ICDs in the Elderly

Because the ICDs have such low prevalence in the elderly, they are less likely to be assessed and treated than other disorders, such as depression and anxiety, that have a higher prevalence and wider understanding among clinicians. Many of the ICDs, such as problematic shopping, gambling, and sexual behavior, may have higher levels of stigma associated with them, thus reducing the likelihood that they will be reported to health care providers unless impairment is severe. Although it is true of all age groups, the elderly may be particularly vulnerable to underdiagnosis due to the presence of competing diseases, reluctance to seek mental health care, and possible increased stigma compared to younger adults.

Elderly patients with problem or pathological gambling may be less likely than younger patients to be identified clinically for several reasons. First, most people with pathological gambling problems, regardless of their age, do not present in clinical settings for treatment of gambling, and the elderly may be even less likely to seek treatment. Most studies of problem or pathological gamblers in treatment report mean ages in the mid-40s, suggesting that older gamblers are less likely to seek treatment specifically for gambling.

Second, instead of seeking specialized treatment, patients with problem and pathological gambling tend to present in primary care settings with more psychosomatic complaints such as back pain, depression, anxiety, or stress-related problems (McGowen & Chamberlain, 2000; Pavalko, 2002; Stewart & Oslin, 2001). Among the elderly, who naturally have more physical aches and pains, it may be more difficult to detect an underlying gambling-related problem in a primary care setting (McGowen & Chamberlain, 2000; Stewart & Oslin, 2001). In addition, primary care clinicians are often not very proficient at identifying psychiatric and substance abuse disorders, particularly in the elderly (Stewart & Oslin, 2001). This tendency likely extends to problem and pathological gambling (McGowen &

Chamberlain, 2000) as well as other behavioral addictions and ICDs.

Third, the elderly may report fewer problems related to gambling, even when asked, for several reasons. First, older respondents are generally thought to attach more stigma to disorders such as depression and alcohol dependence, and thus downplay their experience of such symptoms (Sirey et al., 2001a, 2001b). The same may be true for gambling-related problems such as stress over large losses or family strain due to excessive borrowing. Second, an exaggerated sense of independence, or the need to retain what limited independence they still have, may prompt more resistance to recognizing certain symptoms, such as financial strain or disruption of family relations (Pavalko, 2002). Finally, early cognitive decline may interact with the normal course of pathological gambling to create an even more distorted impression of cause and effect: while the majority of pathological gamblers begin to see their gambling as a result, not a cause, of their life difficulties, elderly pathological gamblers may have an even more distorted sense of the causes of their behavior and its effect on other people (Pavalko, 2002).

No treatments for pathological gambling, or for other ICDs, have been developed specifically for elderly patients. In general, treatment recommendations involve psychosocial rehabilitation models, possibly in combination with medications, several of which are being tested for effectiveness. However, some of these recommendations may be particularly difficult to apply to older patients. One recommendation is that patients should try to avoid gambling cues and involvement with other gamblers, and find suitable leisure activities to substitute for gambling (Blaszczynski & Silove, 1995). This may be particularly problematic for older adults who may have developed an elaborate social structure around gambling. Older adults often have fewer social ties (LaVeist et al., 1997; Thompson & Heller, 1990), and asking a patient to cut those ties and attempt to replace them could prove to be very challenging in this age group. In addition, if social activities were traditionally heavily gambling-related, finding suitable substitute activities might be difficult, particularly for persons with limited physical and/or cognitive abilities.

A second recommendation is to treat depressive and anxiety symptoms that occur during recovery with appropriate medication (Blaszczynski & Silove, 1995). Again, this may be more challenging in older versus younger adults: older adults may be more resistant to such medication (Chiam, 1994) and may be taking other medications that would negatively

interact with psychotropic medications. A third recommendation is that erroneous beliefs, attitudes, and expectations concerning gambling need to be challenged and corrected (Blaszczynski & Silove, 1995). In older patients with diminished cognitive abilities or early symptoms of dementia, Alzheimer's disease, or paranoia, challenging such beliefs might be difficult (Mendez et al., 2000; Unger, 1999). Finally, many care providers suggest that attending Gambling Anonymous (GA) meetings, whether independent of or in conjunction with other types of treatment, is important, although this view is not universally endorsed (Blaszczynski & Silove, 1995; Pavalko, 2002). However, elderly patients with limited capacity to obtain transportation may have difficulty attending such meetings, although better attendance has been reported among elderly patients, possibly due to more free time or boredom (Grant et al., 2001).

Although several pharmacological treatments have shown promise, there are no clearly superior drugs for problem and pathological gambling (Grant et al., 2003b). Most treatment studies have not separated the effects of treatment by age, and there is little evidence that treatment effects would differ by age, other factors being equal (e.g., medication interaction effects). However, future treatment studies may want to explore the specific effects of medications over the lifespan. In addition, some case reports suggest that there may be different sensitivities to pro-dopaminergic drugs that may play a role in the treatment of elderly pathological gamblers (Shah et al., 2004). Further research may explore the beneficial effect of such drugs specifically for this age group.

Summary

Impulse control disorders are increasingly being recognized as important sources of disease and may have an important impact on the health of the population as it ages. Gambling among the elderly, in particular, has increased dramatically over the past few decades, and older adults represent a major target market for the gambling industry. This puts them at potentially increased risk for the development of problem and pathological gambling. The prevalence rates of ICDs decrease with age. However, this may also mean that older adults with such disorders may have more severe forms of the disorders, may have a late-onset disorder secondary to changes in brain function related to aging or medical treatment, or may have more severe comorbidity with other psychiatric disorders. Although ICDs are less

prevalent among the elderly than among younger patients, older adults with ICDs may have unique identification and treatment challenges as a result of their age, comorbid medical conditions, and attitudes about mental health treatment.

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Gender and Impulse Control Disorders

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Abstract

Although women and men (and girls and boys) share similarities, they also exhibit differences that may contribute to the etiology and development of impulse control disorders. Such differences may hold significant implications for the generation of optimal prevention and treatment strategies. In this chapter, we review data on gender-related differences in impulse control disorders, considering epidemiological, clinical, biological, and therapeutic perspectives. Implications for improving prevention and treatment interventions are discussed.

Keywords: female, male, men, women, telescopng

Sex differences exist in brain structure and function (Cahill, 2006), and these differences have implications for psychiatric disorders (Brady et al., 2009; Grant & Potenza, 2007). Gender-related differences exist for the formally defined impulse control disorders (i.e., pathological gambling [PG], kleptomania, pyromania, trichotillomania, intermittent explosive disorder) as well as for those not otherwise specified (i.e., compulsive Internet use, compulsive buying, compulsive sexual behavior). Examining and defining these gender-related differences and their clinical relevance should help develop more effective prevention and treatment strategies for males and females across the lifespan. This chapter reviews research on the influence of gender with respect to the phenomenological, neurobiological, and clinical aspects of impulse control disorders.

Pathological Gambling

Prevalence rates of PG are estimated to range from 0.2% to 2% in the United States (Desai & Potenza, 2008; Petry et al., 2005; Potenza et al., 2001a). Data from the National Epidemiologic Survey of Alcohol and Related Conditions (NESARC) estimate the prevalence rates for lifetime PG in men

at 0.64% and for women at 0.23% (Blanco et al., 2006). This approximately 2:1 male:female ratio has been observed in multiple cultures at various times, suggesting a relatively stable sex difference in adults. Similar and perhaps more pronounced differences exist with respect to gambling problems in youth, in which an approximate 4:1 male:female ratio has been estimated (Brezing et al., in 2010). These findings may in part reflect sex differences in age of gambling onset, with boys more frequently reporting earlier ages of gambling onset than girls (Brezing et al., 2010).

Multiple factors may contribute to sex differences in prevalence estimates for PG. It has been suggested that cultural norms prohibiting gambling may be more salient for women than men (Hing & Breen, 2001). Although data on the association of gambling availability and PG are mixed, access to specific forms of gambling might influence the propensity of men and women to gamble and to develop PG, given the differences in forms of gambling preferred and practiced by men and women (Potenza et al., 2001b, 2006). Specifically, among recreational gamblers, women typically report favoring nonstrategic forms of gambling (e.g., electronic gambling

or slot machine), whereas men typically report favoring strategic forms of gambling (e.g., card or sports gambling; Potenza et al., 2006). These patterns are reflected to a lesser extent in the behaviors of recreational gamblers and to a greater extent in the types of gambling problems that men and women develop (Potenza et al., 2001b, 2006). Together, these data suggest that individuals may develop problems with gambling through impaired behavioral control over specific forms of gambling that are preferred in a sex-specific manner.

Gender differences have been reported in the progression of gambling problems. Although women report on average a later onset of gambling, they develop gambling problems more quickly than men (Grant & Kim, 2002b; Potenza et al., 2001b; Tavares, 2001). This pattern, termed *telescoping*, was previously described for alcoholism and later for drug dependence, and may hold for other impulse control disorders (Piazza et al., 1989). This phenomenon may also be relevant for women seeking treatment more quickly than men (Nelson et al., 2006), a finding observed across multiple psychiatric conditions (Perlick & Manning, 2007).

The specific factors underlying the progression of gambling problems are not well understood. Differences in early gambling experiences have been reported, with women with gambling problems reporting having first gambled with family members and men having gambled as a social interaction (Nelson et al., 2006). Among youth, boys and girls exhibit different patterns of gambling behaviors, with boys tending to report higher wagers, exhibit more risktaking, spend more time and money gambling, and develop more gambling-related problems (Derevensky et al., 1996; Desai et al., 2005; Jacobs, 2004). This pattern may in part reflect cultural differences; boys may be encouraged to participate in a wider range of risk-taking behaviors (including gambling) than girls (Huxley & Carroll, 1992). Among adults, gender-related differences in responses to gambling cues have been reported, with women showing more reactivity than men to gambling advertisements (Weiss & Petry, 2008). The extent to which these and other factors contribute to the development of gambling in males and females requires additional investigation, particularly in longitudinal studies.

Motivations for gambling may differ across the genders, and these differences may relate to differences in psychopathologies observed in males and females. Among adults with PG, women are more likely than men to report gambling to escape from

dysphoria or negative emotional states (Blanco et al., 2006). Gambling problems in women are more closely linked to major depression than they are in men (Desai & Potenza, 2008), and clinical or treatment-seeking samples of women with gambling problems are more likely to report having anxiety, depression, and suicidality (Dannon et al., 2006; Potenza et al., 2001b). While women reportedly gamble to escape from negative moods, men reportedly gamble for excitement, leading to the colloquial terms of *escape gambling* and *action gambling* being attributed typically to women and men, respectively. In support of this finding, in a study of recreational adult gamblers, those gambling for excitement were more likely to be men, and gambling for excitement was associated with heavier gambling (more frequent, greater maximal wins and losses, and more forms of gambling) as well as with externalizing behaviors and disorders (substance abuse and incarceration; Pantalon et al., 2008). These findings suggest that internalizing and externalizing tendencies typically demonstrating female and male predominance, respectively, may underlie gender-related differences in gambling behaviors and related problems. Multiple studies of individuals with PG have found that men are more likely to report problems with externalizing disorders and behaviors including substance abuse and dependence, arrest, and incarceration (Grant & Kim, 2002b; Ladd & Petry, 2002; Potenza et al., 2000). Importantly, some of these differences appear to extend to youth populations and subsyndromal levels of gambling. For example, a significantly stronger association between depression and adolescent gambling was found in girls (odds of about 4) than in boys (odds of about 1), suggesting that some clinically relevant gender-related differences are apparent prior to adulthood (Desai et al., 2005).

Additional gender-related differences are reflected in sociodemographic and clinical measures. Women suffering from PG are more likely than men to have dependent children and be married (Crisp et al., 2004; Tavares, 2001). Women also typically report lower debts than do men (Crisp et al., 2004), consistent with data from adolescents showing that boys gamble more heavily than do girls while reporting fewer mental health problems (Desai et al., 2005). Chinese women compared to Chinese men seeking treatment for PG are more likely to be older, married, less educated and unemployed, and to have started gambling at an older age (Tang et al., 2007). Women compared with men exhibiting subclinical PG are more likely to be widowed/separated/divorced and

to have a lifetime history of mood and anxiety disorders (Blanco et al., 2006).

Gender, TreatmentSeeking, and Self-exclusion

Although some studies indicate that women may be less likely than men to admit that they have a gambling problem (Clarke et al., 2006), others find women more likely to seek treatment and enter into programs like self-exclusion from casinos (Nower & Blaszczynski, 2006). Female compared with male self-excluders are more likely to be African American, retired, unemployed, and older when they apply for self-exclusion. Additionally, these women report a later age of onset for gambling problems and experience a shorter period between onset and self-exclusion than do men. Women are also more likely to self-exclude for reasons including bankruptcy, suicide prevention, and regaining control of their lives. Both men and women are motivated to apply to a self-exclusion program to improve marital relations (Nower & Blaszczynski, 2006).

Gender, Impulsivity, and Adolescent Brain Function: Implications for PG and Impulse Control Disorders

Investigations of brain structure and function suggest differences related to impulse control, with many differences observed in childhood and adolescence. For example, sex differences in adolescent brain function have been observed in the relationship between white matter integrity and impulsivity, with impulsivity related to integrity of the corpus callosum in adolescent boys and impulsivity related to integrity of the splenium in girls (Silveri et al., 2006). Adolescent girls and boys also show structural differences, including differences in volumes of the caudate, amygdala, and hippocampus (Giedd, 2004; Giedd et al., 1996). Although these differences may hold important implications for the development of PG and other disorders characterized by impaired impulse control (Cahill, 2006; Chambers & Potenza, 2003), additional research is needed to investigate directly adolescent brain structure and function in individuals affected by these disorders.

Neuroimaging, PG, and Gender

Most brain imaging studies of PG have involved men, either predominantly or exclusively, creating a deficit in our understanding of the neural correlates of brain functioning in PG in women compared to men. In studies of PG, relatively diminished

activation of ventral striatum and ventromedial prefrontal cortex (PFC) has been observed across multiple tasks, including those assessing cognitive control, gambling urges, simulated gambling, and decisionmaking (reviewed in Potenza, 2008). In subjects without PG, sex differences have been observed in the neural correlates of some of these neurocognitive processes. For example, in studies of decisionmaking using the Iowa Gambling Task (IGT), women and men demonstrated differences in task performance and displayed different patterns of regional activation in the PFC and orbitofrontal cortex (OFC; Bolla et al., 2004). Specifically, men performed better than women, and during task performance, men activated the right and left lateral OFC and the right dorsolateral PFC, and women activated the left medial OFC and the left dorsolateral PFC (Bolla et al., 2004). Additional research is needed to examine directly sex differences in brain structure and function in PG and determine the clinical relevance of such differences.

PG, Gender, and Genetics

Like brain imaging studies, most early studies of the genetic basis for PG involved men, either predominantly or exclusively. Twin studies allow for the estimation of genetic and environmental contributions to disorders (Shah et al., 2005). Studies of male twins have identified substantial genetic contributions to PG, with models estimating that about 50% or more of the variance is genetic in nature (Eisen et al., 1998; Potenza et al., 2005). Recent data indicate a similarly substantial genetic contribution to PG in women (Slutske et al., 2010). Early molecular genetic studies suggest gender-related allelic differences in multiple genes, including those encoding the serotonin transporter, the D4 dopamine receptor, and the monoamine oxidase A enzyme (Ibáñez et al., 2003a, 2003b; Perez de Castro, 1997; Perez de Castro et al., 1999). The extent to which these early examinations can be replicated requires additional investigation, particularly given the small samples studied and other limitations (Ibáñez et al., 2003b). How these findings may relate to clinically relevant measures, and how they can be used to advance prevention and treatment strategies, also warrant additional investigation.

Trichotillomania

Trichotillomania (TTM) involves excessive, interfering, or distressing patterns of hairpulling. Although the precise prevalence of this disorder is not well-established, estimates range from 0.6% to 2.5%,

depending on the geographic location (Christenson et al., 1991b; Vythilingum et al., 2002). There is a female predominance in TTM, with female: male ratios ranging from about 2:1 to 5:1 (Chamberlain et al., 2007; Christenson et al., 1991b). Sex differences in TTM may be developmentally sensitive, as relatively greater rates of TTM appear to exist in boys (Reeve et al., 1992; Santhanam & Rogers, 2008), perhaps related to differences in age at onset. Some studies demonstrate onset in the toddler years, with the majority of children who develop TTM before the age of 6 being boys (Muller, 1987). Therefore, the view of TTM as a predominantly female disorder has been questioned. There may be more males with the disorder early in the course of development, and they may either grow out of it or do not seek treatment as often as females (Swedo & Rapoport, 1991). The majority of people who seek treatment for TTM are female, and typically they develop the disorder between the ages of 11 and 16 years (Minichiello et al., 1994; Swedo & Rapoport, 1991). Hormonal changes might influence the phenomenology of TTM. The disorder can begin concurrently with menarche, and symptom severity in females may increase during the premenstrual period (Bohne et al., 2005; Keuthen et al., 1997).

Gender differences have also been observed in the clinical phenomenology of TTM. Men with TTM have been reported to spend about twice as much time as women pulling or plucking, and for men this appears to occur typically in a relatively reflexive, automatic, and unfocused fashion during contemplative tasks such as writing, reading, driving, or watching television (Grant & Christenson, 2007). Men with TTM appear to experience greater functional impairment than women, as well as co-occurring anxiety disorders (Grant & Christenson, 2007). On the other hand, women with TTM frequently experience major depression, and hair pulling in women may reflect a mechanism to relieve negative affect (Christenson et al., 1994b).

Brain Imaging and Gender

Unlike PG, most neurobiological studies of TTM have involved women, either exclusively or predominantly. Among women with and without TTM, multiple volumetric differences have been observed in such regions as the putamen, PFC, cuneus, and cerebellum (Grachev, 1997; Keuthen et al., 2007; O'Sullivan et al., 1997). Functional studies have also identified differences in women with and without TTM in cortical and cerebellar regions (Swedo et al., 1991).

Treatment and Gender

The majority of individuals with TTM who seek treatment are female. One possible reason for the failure of men to seek treatment could be that baldness is more socially acceptable for men (Minichiello et al., 1994). However, this reason alone is insufficient to explain the finding, as more females than males tend to pull hair from their scalp (Christenson et al., 1994; Minichiello et al., 1994). Additional considerations, including gender differences in treatment-seeking related to culture and stigma, also warrant consideration (Perlick & Manning, 2007).

Kleptomania

Like TTM, kleptomania typically affects more women than men, with prevalence estimates suggesting a 2:1 or 3:1 female:male ratio (Grant & Kim, 2002a; Grant & Potenza, 2008; McElroy et al., 1991; Presta, 2002). In kleptomania, the age of onset is typically later for women than men, with the first symptoms appearing in adolescence or early adulthood (Grant & Potenza, 2008; McElroy et al., 1991, 1996). For both men and women, initial symptoms of kleptomania may continue into later adulthood, with either episodic periods or a continued course of this behavior (McElroy et al., 1996).

Gender-related differences in clinical aspects of kleptomania have been reported. Some gender-related differences are thought to reflect underlying differences in the motivation to steal, with the suggestion that women may be more likely to steal items in an attempt to cope with lost object relations in their lives (Goldman, 1991). With respect to specific items, men are more likely to steal items from an electronics store and women are more likely to steal household goods and hoard their stolen items (Grant & Potenza, 2008; McElroy et al., 1996). Patterns of co-occurring disorders also demonstrate gender-related differences, with men typically demonstrating more problems with substance abuse and other impulse control disorders and women demonstrating borderline personality disorder and bulimia (Grant & Potenza, 2008; Presta, 2002). These findings are consistent with gender-related differences in impulsivity observed in individuals with kleptomania, with men scoring higher than women on the Barratt Impulsiveness Scale (Baylé et al., 2003). Among women, severity of kleptomania has been associated with poorer neuropsychological performance on the Wisconsin Card Sorting Task, a measure of cognitive flexibility.

(Grant et al., 2007). These findings suggest that a compulsive element of behavior is related to the symptoms of kleptomania in women; additional studies are needed to examine the extent to which these findings extend to men with kleptomania.

Biology and Gender

Few investigations have focused on gender-related differences in the biology of kleptomania. One study found that men with kleptomania had significantly more birth traumas than healthy controls (Presta, 2002). A separate case report found that OFC subcortical circuits were damaged in a man with kleptomania (Nyffeler & Regard, 2001). These findings raise the question of whether neurological trauma might influence the development of impulsive behavior, particularly in men. Women, with kleptomania, compared to those without it, demonstrated decreased white matter microstructural integrity in inferior frontal brain regions (Grant et al., 2006). The extent to which these findings extend to men with kleptomania warrants direct investigation.

Pyromania

Few investigations of gender-related differences in pyromania have been performed. Of the relatively few existing studies of pyromania, a significant number have examined arsonists; only a fraction of these individuals meet criteria for pyromania. For example, among 90 arsonists, only 12 fulfilled criteria for pyromania (Lindberg et al., 2005). There is a male predominance among arsonists, with a 3:1 or greater male:female ratio reported (Dickens et al., 2007). Female compared to male arsonists have been found to be older, to have additional psychiatric disorders, and to have a history of sexual abuse (Dickens et al., 2007). In contrast, male arsonists have been more likely to report criminal offenses and substance abuse problems (Dickens et al., 2007). A recent examination of NESARC data found a lifetime prevalence of firesetting of 1.13% in the adult U.S. population (Blanco et al., 2010). Firesetting was associated with specific sociodemographic features (young age, male gender, never married, U.S.-born, annual income below \$70,000, non-Asian, non-Hispanic) and psychiatric disorders, particularly those related to impaired impulse control (antisocial personality disorder, drug dependence, bipolar disorder, and PG).

Gender-related differences have been identified in impulse control disorders in adolescents hospitalized for psychiatric problems (Grant et al., 2007).

Of the impulse control disorders assessed, the most robust statistical differences were found for pyromania; all individuals diagnosed with pyromania were girls, representing 6.9% of the entire sample and 12.5% of the girls (Grant et al., 2007). As other studies have suggested that male adolescents may be at higher risk for fire-setting behavior than female adolescents (Dell'Osso et al., 2006; Strachen, 1981), more research is needed into the prevalence of pyromania in the community and how best to prevent, identify, and treat it.

Problematic Internet Use

Prevalence estimates suggest that about 2% of adolescents and less than 1% of adults experience problematic Internet use (Brezing et al., 2010; Koran et al., 2006; Liu & Potenza, 2007). Like PG, problematic Internet use appears to be more prevalent in males, with some studies reporting a 4:1 male:female ratio (Morahan-Martin & Schumacher, 2000), although other studies have found more equal gender distributions (Johansson & Göttestam, 2004; Kaltiala-Heino et al., 2004).

As with PG, motivations underlying problematic Internet use may have important gender-related differences. While males are more likely to use the Internet for entertainment and to gather information, females are more likely to use it for communication and social interactions (Hamburger & Ben-Artzi, 2000; Shaw & Gant, 2002). Additionally, males tend to score higher than females on levels of comfort and self-efficacy in using the Internet (Schumacher & Morahan-Martin, 2001; Sherman et al., 2000; Wolfradt & Doll, 2001). The extent to which these gender-related differences may lead to problematic Internet use or influence its course requires additional investigation.

As with PG, a link between depression and problematic Internet use has been observed (Brezing et al., 2010; Liu & Potenza, 2007). The extent to which this link is more relevant to females, as appears to be the case in recreational gambling in youth and PG in adults (Blanco et al., 2006; Desai & Potenza, 2008; Desai et al., 2005), or extends to males with problematic Internet use requires additional investigation. Problematic Internet use may overlap with PG, problematic gaming, and compulsive sexual behavior because of their similar characteristics and the use of the Internet for gambling, gaming, and sexual experiences (Beard & Wolf, 2001; Delmonico et al., 2001). Males appear more likely than females to use the Internet for these purposes (Brezing et al., 2010; Johansson & Göttestam,

2004), although this notion has been debated by some (Delmonico et al., 2001).

Compulsive Sexual Behavior

The adult prevalence rate of compulsive sexual behavior (CSB) in the United States has been estimated to range from 3% to 6% (Carnes, 1991; Coleman, 1992), although data from systematic epidemiological studies are lacking. This disorder appears to be male predominant (Kuzma & Black, 2008), with men typically scoring higher on structured measures of CSB than women (Schneider & Schneider, 1996). However, CSB may be as frequent, or more frequent, among females in certain populations (e.g., adolescents hospitalized for psychiatric reasons; Grant et al., 2007).

Men and women tend to participate in different forms of CSB. Men tend to engage more in masturbation, paraphilic activities, exchanging money for sex, multiple sexual encounters, and sexual encounters with strangers (Bancroft & Vukadinovic, 2004; Black et al., 1997). Women are more likely to participate in seductive rituals to obtain multiple sex partners, use sex practices for employment purposes, and engage in sadomasochistic relationships (Black, 2000). Women may also use CSB as a coping mechanism (Turner, 2008). Women with CSB may more often establish emotional attachments to achieve gratification, whereas men with CSB may focus more on the physical aspects of sex (Black et al., 1997). In addition, CSB is frequently found in homosexual and bisexual men (Grant & Potenza, 2006; Warner et al., 2004). It has also been observed in individuals with Parkinson's disease, in whom an association with dopamine agonist treatment has been seen (Weintraub et al., 2010), and the vast majority of individuals with CSB and Parkinson's disease are male. As most CSB research has focused on men, additional research is needed to better specify specific gender differences within CSB.

Intermittent Explosive Disorder

Intermittent explosive disorder (IED) is characterized by isolated episodes of aggressive impulses that are out of proportion to the precipitating psychosocial stressor and result in serious criminal acts against people or destruction of property (Olvera, 2002). Intermittent explosive disorder has an approximately 2:1 male:female ratio (Kessler et al., 2006; Potenza et al., 2009). Among the multiple neurochemical systems implicated in human aggression, several (e.g., testosterone) exhibit sex differences and

suggest a possible etiology for the gender-related differences observed in the prevalence of IED (Potenza et al., 2009). Additional research is needed on the gender-related differences in the pathophysiology, prevention, and treatment of IED.

Compulsive Shopping

Approximately 6% of adults in the United States are estimated to experience compulsive shopping, with a slightly higher prevalence in women than men (Koran et al., 2006). Among clinical samples of adults, a more robust female predominance has been observed, with some female:male ratios as high as 9:1 (Black, 1988; Christenson et al., 1994a; Dittmar, 2005; Koran et al., 2003; McElroy et al., 1994). A similar female predominance has been observed among adolescents with mental health concerns (Grant et al., 2007). Differences in the female: male ratios of clinical and population-based samples may in part reflect a tendency for men to report feeling that they collect items and women to identify the shopping behavior as pathological (Black, 1998). Alternatively, these differences in prevalence could relate in part to more general gender-related differences in treatment-seeking for mental health concerns (Perlick & Manning, 2007).

Multiple factors could influence the development of compulsive shopping differently in men and women. Such factors could include differences in the use of chronic purchasing to relieve negative affect (O'Guinn & Faber, 1989), cultural factors related to advertising and the promotion of a specific lifestyle in female-oriented media (O'Guinn & Faber, 1989), or biological differences in neurochemical and neural systems underlying motivated behaviors (Potenza & Hollander, 2002). Additional research on the factors influencing compulsive shopping in men and women is needed to better improve prevention and treatment strategies for the disorder.

Conclusion

The understanding of gender-related influences on the development and course of impulse control disorders is relatively immature. Significant differences related to gender have been identified in impulse control disorders, with some of these differences similar across disorders and others specific to individual disorders. Consideration of gender in research on the etiology, prevention, and treatment of impulse control disorders will help improve the health and well-being of those individuals directly and indirectly affected by the disorders.

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PART **7**

Special Issues

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Prevention of Impulse Control Disorders

Neda Faregh and Jeffrey L. Derevensky

Abstract

There is currently no unifying model of prevention for all impulse control disorders. As with many other mental health problems, individuals with impulse control disorders frequently share a host of comorbid disorders with common antecedents, symptoms, and risk factors. It is argued that a comprehensive approach to the prevention of impulse control disorders rests in the adoption of a general mental health prevention framework associated with other psychiatric disorders. Specific programs currently available for the prevention of impulse control disorders are discussed. The adoption of prevention programs targeting multiple and general risk behaviors and the promotion of programs that enhance protective factors leading to resiliency for children and adolescents are advocated. Universal prevention programs designed to enhance self-regulation and promote positive development are presented within a risk protection framework.

Keywords: prevention, impulse control disorder, harm minimization, risk and protective factors, mental health

Introduction

The current version of the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition, Text Revision (DSM-IV) outlines five disorders subsumed under Impulse-Control Disorders Not Elsewhere Classified (ICD). They include Intermittent Explosive Disorder, Kleptomania, Pyromania, Trichotillomania, Pathological Gambling, and ICD-Not Otherwise Specified (ICD-NOS). Each of these disorders is characterized by experiences of increased tension and arousal leading to the relevant action that is harmful to the individual or others, followed by pleasure and relief, which may or may not include feelings of guilt and self-reproach. Impulse control disorders may not only occur by themselves but may co-occur with other mental disorders such as substance abuse and mood disorders. In spite of their prevalence, ICDs are frequently hidden disorders that are commonly underdiagnosed and under-treated (Dell'Osso et al., 2006).

This is a particular group of disorders that, despite a long and controversial history of inclusion among psychiatric disorders, is fraught with difficulties in terms of diagnosis and differentiation from common and related human behavior. For example, the differences between shoplifting and true kleptomania, revenge-motivated arson and true pyromania, frequent recreational gambling and pathological gambling, intermittent explosive disorder and antisocial personality disorder, and other common impulsive behaviors versus the pathological version of the behaviors are not always easy to discern (Amara et al., 2007; Lacey & Evans, 1986). A number of other disorders have been suggested as potentially belonging to this category of ICDs, and the DSM-V task force is currently debating and considering their inclusion. It is expected that the release of DSM-V may include a category of Compulsive-Impulsive (C-I) disorders separate from anxiety disorders that would subsume four

new disorders, in addition to those currently under the ICD category. Specifically, the four new C-I disorders would be C-I Internet Usage Disorder, C-I Sexual Behaviors, C-I Skin Picking, and C-I Shopping (Dell'Osso et al., 2007). All are preceded by some form of arousal and consist of a compulsion to maintain the behavior over time. These additions to DSM-V are expected to allow for better reliability of diagnoses and more useful diagnostic categories, notwithstanding the as yet inadequate appreciation of disorder etiology (Regier et al., 2009). The potential new inclusions in the category are supported by past research and based on the impulsive features of the disorders. Impulse control disorders share many features. By definition, all ICDs are characterized by the performance of repetitive behaviors, increased tension before committing these acts, and pleasure and relief experienced during or shortly after these acts. Other similarities across ICDs include the presence of comorbid addictions, common risk factors, and early age of onset. These disorders are fully described elsewhere; hence, we will not review them here and refer the reader to other chapters in this volume. In this chapter, we focus on prevention models and examine appropriate prevention strategies, as well as how prevention research from other fields of mental health can instruct efforts in the prevention of ICDs.

Disorders of impulse control entail multiple costs to communities and society in the broader sense in addition to individual and family consequences. Treatment of these disorders is challenging and often costly, with a majority of affected individuals remaining undiagnosed or undertreated; hence, prevention takes on an overriding importance. Yet, the health, education, and policy professionals have only recently acknowledged the need to prevent these disorders.

Little is known about the best method of prevention for the ICDs, and few programs have been implemented. No best practice models have been recognized. The implemented programs often are deficient in scientific foundation, and frequently lack evaluation and assessment of their efficacy. However, there are numerous commonalities in the risk factors underlying many mental health disorders. These commonalities can lend support to the development of prevention programs for disorders that have been largely unexplored. For example, the field of alcohol and substance abuse prevention has a well-established literature with empirically based knowledge of best practices. Much of what has been

learned in these fields may be applicable to prevention efforts aimed at ICDs.

In general, repetitive behaviors normally do not constitute a disorder until they become problematic in terms of self-regulation. When repetitive behaviors become maladaptive and lead to significant functional drawbacks, or are persistent despite repeated attempts to stop them, they are in some ways similar to an addiction. There are many similarities between the behavioral excesses of some ICDs and those of chemical dependence. This is primarily the reason why some use the term *addiction* when referring to gambling disorders or excessive Internet use that are classified under ICDs rather than addictions. The craving, tension, relief upon acting on the urge, repetition and increase of the urge, rapid sense of relief, the hedonic nature of the counterproductive act, habituation, withdrawal, and high comorbidity with other addictions are all characteristics of substance use disorders and addictions that are similar to ICDs (Marks, 1990). As well, the role of external cues seems to be similar for substance and behavioral addictions. The routines, sounds, and sights associated with gambling in casinos, or the mental association made between drinking an after-meal coffee and inhaling tobacco smoke, are examples of external cues that prime addictive behaviors. These cues are often as powerful as the actual physiological effects of behavior such as the inhalation of tobacco or the euphoria of gambling. External cues vary according to the disorder. However, the similarities point to the possibility of interrelated brain mechanisms and physiology, suggesting that common brain mechanisms are likely involved in both behavioral pathologies and chemical addictions (Marks, 1990).

Although most prevention efforts are aimed at children and adolescents because of the vital importance of early intervention, prevention is germane to all age categories. The research community has been notably urged to include elderly persons in mental health research and prevention efforts and large-scale interventions (Jeste et al., 1999; Katz & Coyne, 2000). Older adults are frequently neglected in prevention programs despite the current pervasiveness of aging populations and the expected rise in the number of older adults by the year 2011, when the baby boom generation reaches the typically defined "senior" age of 65. The lack of attention to the potential mental health needs of this growing subpopulation and the scarcity of mental health service providers with geriatric training is considered a crisis in geriatric mental health fueled

by the general exclusion of the elderly from psychiatric epidemiological studies (Jeste et al., 1999).

Rationale for Prevention

Given the prevalence and disabling nature of ICDs, these disorders are considered one of the most common classes of mental health problems (Dell'Osso et al., 2007). The high prevalence assumes a substantial number of individuals with one or multiple ICD diagnoses, and many more who remain undiagnosed representing a fairly large proportion of the population. The adverse consequences are significant and far-reaching, providing compelling grounds for the initiation and maintenance of effective preventive strategies.

The adverse effects of ICDs are profound and the social and psychological impact is not directed solely at the affected individuals, but also at their families and communities. An earlier Australian study estimated that country's cost for arson at \$157 million annually, in addition to loss of life and injury (Doley, 2003). The costs of problem gambling are both public and private, relating to employment losses, academic achievement, quality of life, and family discord, as well as those relating to productivity losses due to absenteeism, unpaid debts and bankruptcy, compensation for frequent unemployment and welfare payments, and ancillary crime-related expenses. An Australian study estimated the aggregate cost of problem gambling at \$1.8 to \$5.6 billion per year (Eadington, 2003). Such costs are usually unequally distributed among communities, as the stakes vary for certain subpopulations (e.g., Aboriginal and First Nations peoples; Hayward & Colman, 2004). The cost of shoplifting has been estimated at more than \$10 billion per year in the United States (Yaniv, 2009), although the exact proportion relevant to kleptomania is not known. The impact of pathological behavior on the health service delivery system is also substantial. Hence, the nature and the direct impact of ICDs, as well as the unintended consequences of pathological behaviors, are no longer considered insignificant and have become a major concern with public health implications.

In general, only 20% of those in need of mental health services receive appropriate care (Tuma, 1989). It has also been suggested that among children and adolescents, 20% experience symptoms of a mental disorder, with the majority (75%–80%) failing to receive appropriate treatment (Weissberg et al., 2003). Often, appropriate empirically based treatment programs are unavailable, are in short supply, or are not offered for the appropriate target

groups. One of the primary difficulties involved with treatment is the high dropout rates observed among treatment seekers. Often, those who drop out of treatment programs are individuals with the most severe form of the disorder (Crawford et al., 2009). Therefore, proactive efforts toward prevention are often deemed the most effective solution to address problems and to help minimize costs. Given the costs and consequences of problems associated with ICDs, the incentive to work on preventive measures, while challenging, should be a priority.

Prevention Theory

The particular approach taken by a given prevention effort depends on the health status of the target group. Prevention initiatives are predicated on the public health axioms of primary, secondary, and tertiary prevention. Primary prevention aims to decrease the incidence of a particular disorder by preventing its onset; secondary prevention concerns early detection and the resolution of initial problems before they escalate to more severe pathology; and tertiary prevention involves efforts to diminish the severity and the burden of the disorder and improve the patient's quality of life through treatment (Caplan, 1964).

In the early 1990s, the Committee on Prevention of Mental Disorders and the Institute of Medicine were mandated by the U.S. Congress to launch a program aimed at understanding the ways in which risk factors for mental disorders can be reduced. The result was a conceptual framework of prevention emphasizing risk reduction as part of a wide range of intervention efforts for mental disorders, including treatment and the recognition of resilience along with its maintenance, and underlining unique prospects for intervention (Weissberg et al., 2003). This framework draws upon previous successful models of prevention work on both physical and mental disorders and offers recommendations for developing effective prevention programs (Mrazek & Haggerty, 1994).

According to this model, three different modes of prevention can be used, separately or simultaneously, to target distinct groups. At the broad level of prevention, universal methods target the entire population, regardless of risk levels. At the intermediate level, selective interventions have a narrower focus and are aimed at populations or groups known to be at risk but in whom there is no indication of ill health or problem behavior. At the specific level, indicated preventions are directed at those already identified with symptoms of a disorder and

considered to be at high risk of developing the disorder but not yet been diagnosed with the disorder (Mrazek & Haggerty, 1994). At all three levels, the intervention occurs prior to the onset of the disorder. It is focused on an entire population rather than an individual and is derived from prior epidemiological information. At each stage, the interventions are based upon known risk factors and strive to prevent the development of the disorder by either strengthening the health of the population or averting the onset of the disorder (Gullotta, 2005).

Durlak and Wells (1997) described prevention at a broader conceptual level. Accordingly, there are two dimensions of prevention. The first concerns the individual or population, and the second involves the intervention. The level of population is one in which decisions regarding the selection of target populations are made based upon risk characteristics or attributes. At the intervention level, programs affect primarily either the person or the environment. Those that affect the person offer person-centered projects and include those that involve the individual directly (e.g., social learning and a direct instructional approach). Alternatively, programs can target communities in order to bring about pervasive social change (e.g., altering the home through parenting education, or the school environment through changes in school policy or specific teacher education).

Prevention of ICDs

A review of the literature reveals that there are no unifying prevention strategies for all ICDs. There are few disorder-specific prevention programs, but there is limited published literature pertaining to prevention of ICDS and most related information is frequently deficient in empirically based research and rigorous evaluation. Several disorders have received little attention from the research community, and the general lack of information has possibly led to their being overlooked in prevention initiatives. For others, including fire setting, prevention measures are not specifically directed at the compulsive behavior. Prevention programs against fire setting aim at safety education and the deterrence of excessive interest in fire that could lead to arson rather than the prevention of pyromania. Similarly, prevention programs against shoplifting target the retail industry more effectively than shoplifters, and the literature generally does not distinguish shoplifters from kleptomaniacs. The area of problem gambling prevention is further developed both by the

mental health sector and by the industry, though these programs have certain shortcomings and commonly lack long-term systematic evaluation of their effectiveness.

Kleptomania

Prevention methods for shoplifting are generally directed at retailers to create environments that primarily deter and detect theft. Such strategies include two-way and convex mirrors, closed-circuit cameras, electronic detectors at gates, guards, and so on. Interventions targeting potential shoplifters mainly consist of educational materials, but it has been suggested that such materials (films and documentaries) on shoplifting both inform and teach methods of shoplifting and are not a reliable form of intervention (Glasscock et al., 1988). A study of the effectiveness of signage against theft examined a method of public signposting that warned against shoplifting and additionally used a token economy with elementary school children to deter theft of candy from a convenience store. Although the program reduced candy theft by approximately 50%, a 2-week postintervention assessment found increased theft of the candy items above baseline level, suggesting the need for a maintenance program (McNess et al., 1980). There are support groups for shopaholics such as Shopaholic Anonymous, but their emphasis is on treatment through peer support rather than disorder prevention. Available treatments are provided mostly through mainstream health service delivery. These use methods of desensitization and aversion therapy, and some target arousal sensations prior to shoplifting. However, these treatments generally lack evaluation and for the most part are not evidence-based, and some may even be unethical and iatrogenic (Glasscock et al., 1988).

Pyromania

The literature pertaining to prevention of pyromania is scarce, and the little that exists tends to discuss prevention strategies and services that involve programs related to juvenile fire setting. Although fire setting with the intention to cause harm is considered a pathological disorder, juvenile fire setters often come from troubled backgrounds and have a range of behavioral and psychological disorders (Adler et al., 1994). However, they are not necessarily pyromaniacs. The major role of fire-setting prevention programs is focused on education. Prevention programs for fire setting involve teaching children about fire safety and the circumstances under which children's natural curiosity about fire

can be safely explored. The aim of these programs is to reduce the probability of arson by educating children and reducing the incidents of unsupervised child fire play. Such prevention programs are usually coordinated by fire departments and offered through firefighters' visits to schools, fire drills, fire protection information, television programs geared to the young regarding fire safety and the consequences of burns, and other similar initiatives. Other community prevention efforts consist of information leaflets, class curricula, and workshops (e.g., stop-drop-and-roll; home fire escape plans) intended for preschoolers and elementary school children. There are also programs for adolescents and adults on safety topics including methods of storing matches and other flammable material and proper maintenance of fire alarms in homes. Programs intended for adolescents encourage appropriate role modeling and becoming fire safety educators (Gaynor & Hatcher, 1997). Although the programs mentioned above are mainly concerned with delinquent fire activity and do not specifically pertain to pyromania, they can be potentially adapted to specifically target pyromania.

Gaynor and Hatcher (1997) developed a predictive model for pyromania based on risk and protective factors that can identify groups in need of intervention. Accordingly, factors that interplay to produce a given outcome (e.g., arson) are elements that represent individual characteristics, social circumstances, and environmental conditions. Individual characteristics are those that describe the demographic, physical, cognitive, emotional, motivational, and psychiatric attributes of the person. Social circumstances pertain to the family, peers, and the school, including academic achievement. The environment concerns life events and trauma, both eustress and distress, various behavioral expressions, and their consequences (Gaynor & Hatcher, 1997). In general, children and adolescents are at heightened risk of being implicated in pathological behavior when their normal course of development in their particular environments is interrupted by a set of biopsychosocial factors. Gaynor and Hatcher's predictive model of pyromania is based on this premise; hence, it is applicable to other prevention efforts given its comprehensive stance on risk and protective factors.

Gambling

Unlike other ICDs, pathological gambling typically begins as an innocuous social and public activity. Gambling has become a socially acceptable pastime that is gaining in popularity not only for adults but

for children and adolescents as well. In most regions, gambling is supported by governments (often the recipient of its proceeds) and is encouraged by many charitable organizations. Mass media advertising plays a significant role in glamorizing gambling, portraying it as a fashionable and desirable pursuit. Young, successful gamblers are held in high esteem through movie and television portrayals of players. Hence, gambling is ego-syntonic (Blaszczynski & Nower, 2002) and lacks the anticipatory anxiety seen universally among the ego-dystonic acts of fire setting, aggression, theft, or hair pulling. These acts are never seen as desirable, whereas gambling has been portrayed as not only desirable but also beneficial to society through its economic benefits (e.g., increase in employment, the influx of tourists that could create secondary commerce in the vicinity of the gambling venues). The increased government revenues used to foster community programs and increase social benefits are also seen as direct benefits of gambling. However, in most jurisdictions, pathological gambling follows a spatial pattern that mimics the geography of gambling availability and accessibility. Unlike the prevalence of other ICDs, that of pathological gambling is impacted by legislation and municipal policy that influence the launch and expansion of casinos and other gambling venues, the introduction of electronic gaming machines, availability of Internet wagering, and so on (Welte et al., 2004, 2009). The prevention of pathological gambling might require measures that are distinct from those required for other ICDs. However, the similarities would engender a shared degree of underlying interventions focusing on preventing problem behaviors, as ICDs tend to co-occur (e.g., pathological gambling and C-I Internet use disorder; Dell'Osso et al., 2006). It is suggested that the sought tension release often felt prior to committing one ICD act can be achieved through behavioral substitution in which, for example, the suppression of gambling urges is accomplished by committing a different compulsive act. This *dually directional symptom substitution* is noticed among several types of impulse disorders, such as kleptomania and bulimia (Lacey & Evans, 1986), gambling and substance abuse, and pyromania and self-cutting (Coid, 1993). The field of gambling prevention is still considered underdeveloped, but it has borrowed from the relatively well-developed field of alcohol and substance abuse prevention built on the public health models for prevention of infectious diseases (Erickson, 1999). In addition, gambling public policy and government legislation implicating

the gambling industry in the responsible provision of gambling have led to the development of several programs, making this area comparatively more developed than other ICD prevention efforts. Advances in this field could inform prevention efforts in co-occurring and other ICDs.

A number of gambling prevention programs have been proposed and undertaken. Many are designed as primary prevention programs intended for school-age children and youth. Some are aimed at vulnerable groups such as the elderly and those with impulse and addiction disorders. These programs tend to focus on multiple risk behaviors, reducing risk factors, and increasing resiliency. Substance abuse and gambling prevention programs aim to prevent or delay initial use, for example by increasing the legal drinking or gambling age, and by educating the target groups about the harms associated with use and indulgence. Despite laws that prohibit underage gambling, it is well documented that school-age children and adolescents generally gamble among themselves using nonregulated forms of gambling (Gupta & Derevensky, 1998). As with other risky behaviors, 70%–80% of children and adolescents report frequent engagement in both regulated and unregulated forms of gambling. Examples include playing cards for money, betting on sports events, and buying lottery tickets and betting on horse races. Because of the inherent vulnerability in younger age groups, the prevalence of problem gambling among adolescents is two to four times that of adults (Jacobs, 2000). As a group, adolescents are especially susceptible to developing significant gambling problems. This idea is not well understood, as it is generally assumed that because underage gambling is illegal, it is not taking place (Derevensky et al., 2004). Yet, adolescents have shown a rapid transition from recreational to problem gambling (Gupta & Derevensky, 2000). In addition, the nature of the gambling-related problems and the negative and long-lasting corollary for adolescents are considerable and severe, representing both a mental health and a public health issue (see Korn & Shaffer, 1999, for a comprehensive review, and see Messerlian et al., 2003, for a public health perspective on adolescent gambling).

In contrast to traditional prevention strategies, government policy and industry practices have adopted a harm reduction–harm minimization strategy toward gambling that does not call for abstinence from the activity, although abstinence is theoretically imposed on youth through prohibition of underage play, with variable rates of success.

The harm reduction model aims at reducing the potential for developing pathological gambling, in contrast to the traditional models of prevention of adolescent risky behaviors, which generally prescribe abstinence and nonuse (Dickson et al., 2004a). The adoption of such harm reduction strategies is predicated on the notion that it is difficult to prevent individuals from engaging in gambling or other risky activities, and that the choice to gamble is a matter of individual right and freedom (Shaffer, 2002). Also, it would not be possible to stop all forms of illegal or underage gambling, especially given the widespread social acceptance of gambling as a reasonable recreational pursuit and its ease of access. The purpose of harm reduction strategies is therefore to minimize any negative consequences that may be associated with gambling rather than targeting the risky behavior itself.

The few prevention programs aimed at gambling that currently exist are implemented sporadically, and most have no or few empirically based principles. Most of these programs are mainly aimed at reducing the incidence of problem gambling rather than addressing the risk factors associated with it. The majority have a harm-reduction approach, commonly encourage responsible gambling, and attempt to raise awareness of problematic issues related to gambling as an addiction. To raise awareness, programs generally provide information on recreational gambling and problem gambling, motivations to gamble, forewarning indications of problem development, consequences of excessive gambling, and how to obtain help for individuals who have a gambling problem. Programs that target specific at-risk populations (e.g., First Nations) tend to advocate abstinence given the associated harm and the ease of problem development. However, the majority of problem gambling programs are not based on existing data on the role of risk or protective factors, few have had systematic evaluations, and most generally fall short of standards associated with best practices (Derevensky et al., 2004). Although attempts to establish prevention programs are worthy and commendable, these examples demonstrate the difficulty of instituting preventive measures and gauging their usefulness.

In general, the specific type and form of prevention approach that could best protect youth and other segments of the population is not obvious, and empirical evidence for the use of harm-reduction strategies for gambling or other ICDs, including the C-I disorders, is still insufficient. Furthermore, there are few program evaluations of harm-reduction

initiatives; therefore, it remain unclear whether harm-reductions strategies have resulted in positive and/or negative outcomes (Derevensky et al., 2004).

Harm-reduction strategies seem to be favored by governments, especially with regard to gambling, because by not demanding abstinence, which would be difficult to enforce, they also benefit from the gambling-generated revenues. Although little is known about the relevance or effectiveness of this approach for other ICDs, the harm-reduction strategies for gambling are better understood in terms of their relevance and potential outcomes. Most governments have implemented policies that aim at reducing or minimizing the negative impact of gambling without placing any limit on gambling revenues or access to gambling for the general public, the majority of which does not suffer adverse consequences from their gambling behavior. The harm-reduction strategy assumes either that adolescents do not gamble or that the strategies aimed at adults can be effective among adolescents who engage in both regulated and unregulated forms of gambling.

It is well established that an early age of onset, gambling behavior is a significant predictor of later gambling problems (Gupta & Derevensky, 2000; Jacobs, 2000). The harm-reduction strategy by itself does not attempt to reduce the age of onset of gambling problems and therefore cannot be a successful prevention paradigm for the entire population. There is currently even less evidence of the effectiveness of harm-reduction strategies potentially aimed at the prevention of other ICDs and C-I disorders. In fact, the relevance of a strict version of harm-reduction strategies to rare but disturbing disorders such as pyromania or other, more common disorders in the ICD category is doubtful (for a review of harm-reduction prevention programs see Derevensky et al., 2004).

A number of harm-minimization practices have been adopted by the gambling industry. These include practices that attempt to modify electronic gambling machines and their availability and those that increase responsible gambling. Responsible gambling is defined by policies and practices intended to prevent and reduce harms associated with gambling. In general, these include self-exclusion options, responsible gambling messages, cash-out policies, the availability of counselors in casinos, toll-free problem gambling hotlines, the use of smart card and biometric technology with preset limits, and media campaigns. The self-exclusion programs are generally aimed at problem gamblers

and are intended to limit the gamblers' losses (Nower & Blaszczynski, 2008). The practice is initiated by gamblers who voluntarily exclude themselves from gaming venues for an indefinite period of time. Occasionally, third-party exclusion is possible in some jurisdictions. The process involves identifying oneself to the security office of a gaming venue and following a set of formalities that include having a photograph taken (for future identification purposes) and signing promissory documents wherein the gambler agrees to refrain from entering any gaming venue, and the venues agree to prevent the gambler's admission to their premises by monitoring, removing them, and reporting potential infractions to law enforcement officers. The self-exclusion programs have been criticized and their efficacy has been questioned; the issue of whether a signed self-exclusions form constitutes a binding contract has also been debated (Faregh & Leth-Steensen, 2009). However, findings in several jurisdictions have concluded that the practice is effective in reducing gambling among self-excluders (Ladouceur et al., 2007).

Modifications to electronic gambling machines include on-screen clocks, reduced speed of games, mandatory cash-out that terminates machine play after a predetermined amount of time has elapsed in order to encourage players to self-assess, and modifications to sounds and lighting. On-screen messages that cause interruptions in play time have been shown to have small harm-reduction impacts (Ladouceur & Sevigny, 2003). There is some evidence that reducing the maximum amount of wagers and reducing the number of electronic gambling machines minimize the risk of harm associated with heavy gambling involvement. Training of gambling venue staff to recognize signs of gambling problems and identify those at risk has been undertaken by certain jurisdictions (Hobson, 2005), but evidence of their effectiveness is weak (Blaszczynski et al., 2006).

Other services provided to gamblers include Internet or paper-based brochures with an explanation of responsible gaming and its code of conduct, as well as information on problem gambling treatment. The use of media to this effect includes responsible gaming radio ads and advertising of helplines established by most jurisdictions. In addition, funding for research institutions, such as the Ontario Problem Gambling Research Centre, that are mandated to examine gambling problems is considered a long-term prevention strategy.

There have also been community prevention strategies involving player and public education

aimed at minimizing gambling-associated harm. The International Centre for Youth Gambling Problems and High-Risk Behaviors at McGill University has designed several public service announcements (PSAs) for parents, focusing on the importance of parent-child communication and parental awareness of children's gambling activities. The Centre has also been actively involved in prevention efforts through school-based awareness and prevention workshops, the design and production of gambling-related games and tools (e.g., the *Know Limits* board game), computer-based interactive games (e.g., *The Amazing Chateau, Hooked City*), and adolescent-focused documentaries (e.g., *Clean Break*). The programs are predicated on increasing children's knowledge of the risks and problems associated with problem gambling, modifying inappropriate attitudes, and changing and correcting false cognitions and erroneous beliefs, ultimately to modify behavior by preventing excessive gambling and altering inappropriate patterns of behavior. The results of short-term follow-up studies are encouraging for increasing knowledge, modifying attitudes, changing cognitions, and dispelling erroneous beliefs—all prerequisites for modifying behavior. Unfortunately, funding restrictions have prohibited long-term longitudinal studies.

As yet, there is no direct literature on prevention of specific ICDs, with few exceptions. In examining the psychiatric literature, it is evident that a host of comorbid disorders are found among individuals with an ICD diagnosis, indicating the troubling incidence of severe mental health problems. Studies on psychopharmacological treatment of ICDs are relatively rare, and systematic controlled trials are for the most part lacking. Available psychopharmacological treatments of ICDs are carried out primarily with individuals seeking treatment (ascertainment bias) and often on small samples, not providing the necessary evidence base for a treatment rationale. Pharmacological treatments have included the use of serotonergic agents for problem gambling, mood stabilizers and antipsychotic medication for intermittent explosive disorder, and none for pyromania (Dell'Osso et al., 2007). The choice of pharmaceutical agent is often influenced by the presence of comorbid psychiatric conditions (e.g., mood disorders and addictions), which provides the rationale for the use of specific medications or a combination of medications (Grant et al., 2003a). Many of the treatments (e.g., for problem gambling) still have no Food and Drug Administration approval, and the published literature provides no clear treatment

guidelines (Grant et al., 2003a, 2003b). For a comprehensive review of the pharmacological management of ICDs, the reader is referred to Grant and Potenza (2004) and the chapters in Part 4 of this volume.

In spite of the lack of a unifying model of prevention for ICDs, as well as the lack of clear treatment guidelines and evidence of efficacy, and given the large number of common antecedents and symptoms of these disorders, it is argued that the best comprehensive approach to the reduction of ICDs is to adopt a general framework of mental disorder prevention, along with intervention methods that thwart recidivism.

More recent views on reasons why abnormal behavior might develop consider a few additional factors. Other than physiological (genetic, biochemical, and hormonal), psychological (personality, mood, and stress mechanisms), and environmental (the family/work/school units, the larger community, and the quality and quantity of relationships with colleagues, family, and friends) factors, a great deal of attention is paid to the interaction among any number of these variables and the interplay between the factors (Plante, 2006). The interplay between factors is considered to be the source of problems that lead to mental disorders. On a more global level, Plante (2006) outlines seven factors as a nonexhaustive catalog of prevention principles: (1) adopting a policy of intolerance to abuse and neglect of children, (2) minimizing poverty, (3) curtailing exposure to violence, (4) developing effective interventions including drugs for at-risk groups, (5) changing the cultural expectations of behavior through public policy, (6) avoiding exposure to risk factors for abnormal psychology, and (7) maximizing ethics through the development of social responsibility and concern for others.

The key elements that contribute to the expression of behavioral pathology are not unique to all individuals and all disorders, though there are a number of shared factors. An understanding of the shared factors can guide prevention efforts directed at those deemed at risk of developing any one of the ICDs. In spite of the fact that a well-developed prevention framework can be applied to any disorder, the programs that target specific pathologies can vary considerably, with certain levels of overlap. As already discussed, no specific prevention program have been developed for all ICDs. Prevention programs exist for specific ICDs, with sporadic implementation, though many are not scientifically based and fewer still have had systematic evaluation of

their effectiveness. As described earlier, ICDs share several common traits and antecedents, yet differ in significant ways. Some of the ICDs are less well understood than others, and the role of genetics and the interplay of the environment and biology are even less clear. Lacking explicit knowledge of etiology and origin, the concept of universal prevention would be the core approach to reducing the risk for the development of mental disorders including ICDs, as well as antisocial and high-risk behaviors. In addition, the field can draw on the knowledge base of other prevention fields, such as substance abuse and addictions prevention (Dickson et al., 2004b).

Universal psychosocial factors implicated in mental disorders and substance abuse are negative patterns of family life and maladaptive family interactions, lack of role models, poor scholastic achievement, and life traumas such as physical and sexual abuse and child neglect. Engagement in high-risk-behaviors generally gives rise to negative outcomes and a predisposition to further risky behaviors. Under all circumstances, there is an interplay of factors pertaining to an individual's psychology, biology, and environment that determine future behaviors, their course, and their outcome. Furthermore, the etiologies of ICDs and most psychiatric disorders are not yet fully identified, and the developmental period when a disorder, determined through multiple factors, occurs is generally unknown. This suggests the need for multifaceted programs beyond the prevention of specific disorders to involve adaptation of systems that work at the individual, family, and community levels to facilitate alterations of risk behaviors and the emotional and psychological problems that ensue (Durlak & Wells, 1997). These efforts generally include strategies that modify the "risk status" of youth and enhance protective factors (Dickson et al., 2009).

The enhancement of protective factors involves the development and maintenance of important competencies that promote wellness. This strategy, often referred to as the *enhancement model*, assumes that individuals with well-developed competencies are more capable and hence more proficient at adjusting to and withstanding stressors or factors that could lead to maladjustment (Durlak & Wells, 1997). In effect, prevention models have evolved from focusing on single disorders and preventing specific problems to much broader perspectives that include the prevention of general psychological and behavioral dysfunctions and the espousal of general mental health.

Prevention of Mental Health Disorders

Where prevention involves reducing risk factors and increasing resiliency, efforts must concentrate on (1) reducing problematic behaviors and (2) expanding the patient's positive sense of self and the acquisition of a repertoire of socially desirable activities and choices. The former leads to increasing the number of individuals who might enjoy acceptable levels of healthy living but lack what is necessary to flourish and create prosperous communities. The latter helps to generate the development of social competence, autonomy, and a sense of purpose, all of which are indispensable in reducing mental health disorders.

It is generally accepted that risky behaviors tend to lead to poor immediate and long-term outcomes, to have detrimental consequences, and to compromise well-being. However, there can be considerable variability in outcomes and consequences within populations and across contexts. Risk behaviors themselves are often considered risk factors, especially in the developmental course of adolescents. Another category of risk factors includes those that lie at the base of risky behaviors. Variables that motivate and induce engagement in risky behavior are considered antecedent risk factors with both immediate and long-term impacts, and with direct and indirect pathways to negative outcomes (Jessor, 1999). Factors that mediate the outcome of such risks are considered protective factors. Protective factors both directly and indirectly impact the effect of exposure and engagement in risk. Both risk and protective factors have been the subjects of numerous prevention studies in an attempt to reduce one set and increase the other.

Exposure to risk factors and their antecedents, and involvement in risky behaviors, do not necessarily result in unfavorable outcomes, showing heterogeneity across time, age, gender, environment, and context. This heterogeneity of outcomes is the reason behind the need for identifying and understanding protective and mediating factors with added levels of factor complexity, interrelatedness, and interactions that result from the dynamic nature of behavior and context.

The dynamic nature of interaction among risk-protective and antecedent-consequent factors highlights the vital role of development and change for an individual within his or her environment and context. The structure and makeup of variability and the shifting of directions and causality call for more careful approaches to prevention. Such prevention approaches must accommodate two sides of the

same coin. First is the need to tailor prevention programs to differential trajectories of specific target groups based on their context (environment, background, and circumstance). The second is the need to accommodate the corollary of risky behavior within a prevention strategy.

The risk-protection framework is an approach to prevention advanced by public health researchers. Risk and protection-focused approaches have been successful in diverse domains, including cardiovascular disease and safe driving (Howell, 2009). Risk factors are conceptualized as variables that increase the risk of developing a problematic behavior or disorder, and protective factors are conceptualized as those that either avert problems or interact with other variables to mediate the effect of risks. This approach recognizes that individuals experiencing a psychological disorder have no control over the risk or protective factors to which they are exposed. Family environment, the surrounding community, and the majority of individual psychological and physiological variables are not under the control of children and adolescents whom the prevention efforts aim to target. In essence, the risk and protection-focused prevention does not place responsibility for the behavioral disorders on the youth (Howell, 2009). Rather, it seeks to reduce risks and increase protective factors. This approach is at the base of a prevention strategy known as *Positive Youth Development* (PYD).

The PYD initiative aims to support adolescents and youth in their daily endeavors to achieve their full potential, thereby increasing protective factors and decreasing risk factors related to mental health disorders. The basic tenet of PYD is that by achieving their full potential, individuals are less likely to engage in risky behaviors. To use this strategy, families, the community, and grassroots organizations must be mobilized and become involved in promoting productive activities that build life skills and emphasize social mindfulness. The youth themselves are involved and active participants in the process of PYD as they engage in paid and volunteer activities and services that benefit their communities and their own positive growth as they explore their roles as valuable and productive citizens. The PYD strategy mediates social skills, respect for diversity, engagement, problem solving, peaceful resolution of conflict, and the formation of lifelong learning habits (Carnegie Council on Adolescent Development, 1995).

Positive development requires youth to cultivate an amalgam of skills, attitudes, and dispositions

that will enable them to engage in life with the skills needed for agency and initiative. In a study of youth disposition, Larson (2000) collected data on reported inner experiences of a representative sample of middle-class white adolescents who detailed their thoughts at randomly selected times of the day. The participants reported boredom and ennui for 27% of the random moments, a finding that Larson describes as incongruent with their privileged lives and evidence of the absence of engagement in positive life trajectories. Larson cautions against a lack of self-determination and want of motivation among youth. Furthermore, the absence of initiative and intrinsic motivation has shown significant correlations with failure to achieve self-directed goals and intentions among adolescents (Gollwitzer, 2008). Along the same lines, Deci and Ryan (1993) suggest that the capacity for autonomous action and self-determined motivation is the cornerstone of positive development and civic engagement. They also argue that the social environments of the school and the family play a crucial role in healthy development (Deci & Ryan, 1993).

The associations between impulse control and self-regulation, and between self-regulation and goal pursuit, as well as the role of goal pursuit in the development of resilience, are interrelated topics worthy of note. The essence of resilience and its promotion is the development of a positive sense of self and of human flourishing. There is a strong relationship between goal pursuit and self-regulation. Self-regulation is conceptualized as the struggle between impulses and the exercise of willpower (Vohs et al., 2009), an often explored topic in the investigation of ICDs given that those with such disorders are usually deficient in self-regulation. Self-regulation insufficiencies are highly related to most forms of *impulsivity*, which Strayhorn (2002) defines as the failure to consider the consequences of one's actions. In fact, most of the literature regarding self-regulation is concerned with impulsivity and impulse control problems. Self-regulation is also referred to as *self-discipline*, *self-control*, or *the ability to delay gratification*. Deficits in self-regulation are associated with a majority of psychopathologies and tend to impede goal pursuit (Strayhorn, 2002).

The relevance of self-regulation to ICD prevention is best seen in the work of Vohs and colleagues (2009), who have demonstrated that the principles of self-regulation are played out in goal pursuit and on occasions in which self-regulation and restraint of impulse are essential. Domain-specific applications of the self-regulation theory extend to impulsive

spending, interpersonal functioning, sexual behavior, and all spheres of interaction that involve dealing with emotions and stressors (Muraven et al., 1998).

Self-regulation is exercised when effort expenditure is required to accomplish a goal, especially a long-term goal. It is the effort expenditure toward self-regulation that allows an individual to deliberately overlook the pleasures and rewards of various stimulants or urges (drugs, gambling, spending money, sex, aggression) in order to adhere to rules or achieve goals. There are countless circumstances requiring self-regulation. Strayhorn (2002) outlines its use in curbing impulses to follow rules, in reducing hostile inclinations to engage in peaceful conflict resolution, in pacifying one's mind after frustration rather than indulging in tantrums, in keeping appointments, in complying with medical treatments, and so on. In essence, for the majority of people, self-regulation is used continuously on a daily basis.

Research on self-regulation has demonstrated that it is a limited resource subject to depletion. The exhaustion or reduction of self-regulation is hastened by use, including the use of emotional regulation. In laboratory settings, individuals who had to exert self-control in one task performed poorly on subsequent unrelated tasks requiring self-control (Muraven, 2008). These findings have been corroborated by several studies using an assortment of measures and experimental paradigms, supporting the notion that self-regulation is an exhaustible resource. However, it is not clear whether it is the exertion of effort toward self-discipline or frustration in response to failure in attempts at self-restraint that diminish self-control on subsequent tasks (Strayhorn, 2002). Nevertheless, self-regulation can also be strengthened by practice and training, similar to muscular exercise (Muraven et al., 1999). Animal studies (Chelonis et al., 1994; Chelonis & Logue, 1997) have revealed that when the effort required to exercise restraint is too great, there is a cessation of effort and even complete failure to respond. Studies with humans have revealed that in the face of anticipated challenges or difficulties, individuals tend to conserve their self-regulatory resource so that they may persevere less in the face of one challenge when another is looming (Baumeister & Heatherton, 1996; Strayhorn, 2002; Vohs et al., 2009). Muraven et al. (1998) suggest that the extent to which self-regulatory resources are drawn on determines subsequent decline and diminution of self-regulation in future undertakings. The overall inference from these and similar studies

is that when individuals attempt to embark on too many self-regulatory undertakings simultaneously, they are less likely to succeed.

Although the relevant literature has reported some advances in how to fuel self-regulatory resources, the research is still limited to a few examinations of potential variables. For example, glucose consumption (Vohs et al., 2009), as well as rest and sleep (Tice et al., 2007), have been shown to enhance self-regulation, but few studies have examined the role of psychological and sociological indicators. In laboratory settings, even brief experiences of positive affects could help individuals reassert their volitional abilities and restore their self-regulatory resources (Tice et al., 2007). However, it is likely that environmental and psychosocial variables play a significant role in replenishing the depleted mental resources that facilitate goal pursuit. In addition to psychological factors such as intelligence and temperament, social circumstances of the family, including parental income and education, neighborhood characteristics, and peer relations, play a major role in the educational outcome of adolescents. If these variables present as risk rather than protective factors, they likely deplete the self-regulatory resources that support goal pursuit. Self-regulation is a skill whose appropriate and extensive development would greatly facilitate the prevention of many mental disorders, especially those that involve impulse control.

In his work on the relationship between achievement and the pursuit of personal goals, Little (2007) has demonstrated that personal flourishing is advanced when individuals engage in meaningful, personally salient activities. Disciplined engagement in daily responsibilities and the systematic pursuit of lifelong aspirations are two interrelated activities that enhance human flourishing and impact on well-being across the lifespan. Activities that promote social goals and facilitate meaningful projects are those that contribute to and enhance personal well-being. These activities and pursuits affect individuals' positive development and are furthered by social and cultural expectations as well as interpersonal relationships. The environments that can improve well-being are consistent with supportive interactions and a sense of control that boosts individuals' accurate sense of both their resources and constraints (Little, 2007). The pursuit of personal projects by adolescents and its relationship to their positive development has been a focus of numerous studies. These projects have suggested that programs that contribute the most to adolescents' well-being

and positive development are social in nature, involve others, and allow for self-expression (Larson, 2000; Little, 2007). Examples include community projects and volunteer work, participation in organizations, and team sports. Volunteerism, in particular, has been shown to enhance and improve personal flourishing. Whether the intention is self-serving or altruistic—that is, primarily to enhance one's own skills or to satisfy one's interests and needs, or primarily to benefit others—the end result is that of gaining social capital (the ability to impact the well-being of others) and individual flourishing (Salmela-Aro & Little, 2007). Such activities provide the contexts in which the senses of agency and initiative develop (Larson, 2000). Individual identity is further developed through activities that involve awareness of being needed and of having something valuable to offer.

It has also been suggested that positive development and human flourishing are achieved through three distinct means: personal well-being, the ability to pursue personally meaningful individual or social projects, and social capital (Salmela-Aro & Little, 2007). Such projects are not private undertakings, but rather interpersonal phenomena that depend on and involve other individuals and different aspects of the community in a variety of ways for their achievement or failure. Environmental and contextual factors play a strong role in the three components of human flourishing that lead to thriving individuals within prosperous communities.

Well-being and positive development are promoted through concordance across individual goals and the social environment when socially motivated goals are the medium that begets a positive sense of identity and continuity. An example of the absence of concordance is found among suicidal adolescents, who, upon questioning, cannot provide reasons why theirs or even others' lives should continue (Ball & Chandler, 1989). Studies of suicide rates among Canadian Aboriginal youth have shown that within First Nation communities that were able to assert local control over important facets of community life, maintain access to traditional territories, and achieve self-determination, the rates of adolescent suicide were significantly lower than in other First Nation communities where control over community life and the pursuit of sustainable self-directed projects were not yet achieved (Chandler et al., 2003). The lower rates of suicide are suggestive of developed autonomy, active involvement in the promotion of one's cultural heritage, and cultural continuity. These studies demonstrate the relationship

between autonomy and positive development and how the promotion of one begets the other. Hence, important factors in the success of any prevention effort are the promotion of positive development, the endorsement of autonomy, and the establishment of environments and organizations to support activities that enhance self-determination and impetus. With regard to ICDs in particular, environments that teach and promote self-regulation and fuel its reserves, which in turn creates the discipline required to resist various impulses and support positive development, become the necessary backdrops of prevention programs.

To accomplish prevention and health promotion, a system of interrelated techniques must be employed. In general terms, this system consists of separate processes that involve different levels of the community. Gullotta (2005) has developed a four-technique model for prevention that summarizes the various aspects of prevention work. Gullotta has categorized these aspects under the general headings of education, social competency, mutual self-help or *natural caregiving*, and community organization and systems interventions.

Within Gullotta's four-technique model (2005) education consists of public information and can take a number of forms, ranging from print to radio, television, films, stories, and other artistic exhibits to Internet or any form of mass or group communication. Education can happen in the classroom, through community events, billboards, and various other forms of advertising. Although essential, this method of prevention alone is rarely effective. Examples from tobacco research have shown a discontinuity between knowledge and motivation. For instance, printed messages on cigarette boxes do increase smokers' knowledge of the hazards of smoking but do little to increase the motivation to stop smoking (Farrelly et al., 2003). A 3-year antismoking media campaign (1967–1970), considered the first groundbreaking antitobacco strategy, was most effective during its first year by providing health information related to smoking, yet the per capita smoking reduction was no more than 5% (Schneider & Murphy, 1981). The health information was simply serving as counter-marketing against a strong advertising-marketing campaign by the tobacco industry that emphasized the benefits of smoking. This example demonstrates the need not only for a multipronged approach to health education, but also for the synchronization of efforts by multiple players.

Building social competency is a second technique in prevention measures within the four-technique

framework. Similar to PYD, this approach is based on the premise that socially competent individuals are more resilient and better able to counter or tolerate the stressful events that are inevitable in life. The promotion of social competency engenders group membership, a sense of belonging and solidarity, and making meaningful contributions to one's group (Gullotta, 2005). Social competency entails positive socialization with family members and peers where these groups serve as a critical socialization backdrop and have a significant role in predicting the social adjustment of children and adolescents (Criss et al., 2009). Social competency also entails internalization of appropriate social skills, self-esteem, and self-mastery, including the ability to regulate emotions and take an interest in the larger social milieu (Gullotta, 2005). In essence, nurturing self-competency involves the development of life skills and a positive social structure in which the family and the peer group play a central role.

Mutual self-help, or natural caregiving, is the third tool of prevention (Gullotta, 2005). This measure of mental disorder prevention involves the formation of groups that come together to provide mutual service. A relevant example is Gamblers Anonymous and its adapted 12-step process of mutual aid and fellowship, which offers a successful form of peer support that to some extent replaces professional intervention and treatment. Others who provide mutual self-help are individuals from community organizations, teachers, members of the police force, or the clergy, who are in a position to offer assistance and advice without necessarily being trained as mental health professionals (Gullotta, 1987).

The fourth technique involved in prevention under Gullotta's framework (2005) is the involvement of community organizations concerned with the promotion of social capital. Examples include neighborhood watch groups, sports leagues, or groups concerned with residential zoning or child safety. These groups are normally made up of individuals with a shared concern who come together to express apprehensions and to cultivate solutions for their community problems. These community organizations are in a strong position to assess elements of dysfunction and to address them by raising awareness and by seeking action from legislative or judicial bodies (Gullotta, 2005).

In light of the above, it is evident that the role of the environment and public policy in promoting positive development is to provide the means

and the mechanisms by which citizens can attain agency and the required intrinsic motivation for self-determination. This overarching objective is a comprehensive and broad undertaking that forms the basis of universal prevention for a variety of mental health disorders. Children and adolescents growing up in an environment that fosters positive development and promotes agency will likely develop into thriving adults with an increased capacity for resilience and improved coping mechanisms to manage life stressors and adversity.

Management of Prevention Initiatives

For prevention programs to be successful, there must be a coordination of efforts by multiple parties, each with the aim of improving the overall attributes of the environments in which children and youth live and develop. Prevention of mental disorders requires knowledge of how the pathological behaviors begin and progress. Longitudinal studies, though relatively rare and difficult to conduct, have proven helpful in ascertaining empirically based prevention efforts. Many prevention programs are not evaluated on a long-term basis using the Centre for Substance Abuse Prevention (CSAP) in Washington framework due to a lack of funding. Unfortunately, lack of program effectiveness has sometimes led to a moratorium on funding prevention research (Sloboda et al., 2009). Although ineffective prevention programs may have been misguided, they have been helpful in refining future prevention work and in teaching lessons in efficacy. For the most part, prevention work in one domain can inform efforts in several others, the same way that ineffective programs can update and improve each other.

In the 1980s, the American Psychological Association's (APA) prevention task force launched an inquiry into existing prevention programs. Their investigation found that only 14 out of 300 prevention programs showed any evidence of long-term effectiveness. Since then, the field of research-based prevention has progressed and developed frameworks to guide prevention practices and policies (Weissberg et al., 2003). Both past successful and failed prevention efforts have taught the research community a number of important lessons, including, for example, the necessity of making careful needs assessments, knowing the available resources and key organizations, understanding the affected groups, and appreciating the existence and extent of comorbid disorders.

The CSAP has developed effective evidence-based models of prevention for the deterrence or

delay of disability from chronic diseases. This model encompasses many disorders, including substance abuse and mental health problems, and aims at promoting overall mental health. The CSAP prevention framework focuses on reducing risk factors associated with a particular disorder while simultaneously increasing protective factors. The U.S. National Registry of approved prevention programs can be found at www.nrepp.samhsa.gov. This government body requires that communities targeted for prevention efforts engage in five distinct yet reiterative phases of program development and operation. The first phase is that of needs assessment based upon epidemiological or other empirical data. This is followed by capacity building, which consists of developing the resources needed to engage in and assist prevention programs. The third phase incorporates specific strategies, followed by the implementation phase, and ending with an evaluation phase consisting of ongoing monitoring and surveillance of the project's effectiveness and outcome.

In spite of the fact that ICDs are universal problems, communities vary in the specific challenges they face and the groups affected by the problems. Consequently, the indicators that motivate communities to initiate and carry out prevention efforts tend to vary. Environmental factors may dictate an increase in the number of individuals experiencing a particular disorder. The extent to which a community normalizes (e.g., a change of vocabulary in industry and government discourse from *gambling* to *gaming*) and glamorizes (e.g., media advertising) gambling activity, and expands gambling venues, may be associated with gambling problems developed in various subpopulations. For example, a high density of lottery outlets in poor neighborhoods has been found to be significantly correlated with a higher prevalence of problem gambling among low-income individuals; similarly, amounts wagered in gambling venues are highest among those in low socioeconomic status groups and positively correlated with a given community's gambling expansions (Welte et al., 2004, 2006). Communities in which casinos operate tend to have a higher proportion of residents with gambling problems: a casino within 50 miles of one's home is associated with doubled levels of gambling-related problems and pathological gambling (Gerstein et al., 1999). Adolescent gambling problems in one community may be due to easy availability and accessibility of gambling venues or to lack of policy or compliance, but may be due to cultural norms and acceptance of gambling behavior in another.

Appropriate data must be collected to allow researchers to draw conclusions concerning the types of existing problems. It is often these collected data and conclusions that help to engage key organizations. The presentation of factual data regarding problems within a community can also help to situate a particular region with respect to others for comparison and allow priorities to be set. For example, adolescents and youth are often the primary target groups for prevention efforts given the potential for setbacks and future hindrance. In addition, assessment results may clarify the type of prevention work required. It may be that tertiary prevention is ineffective for a given community and that secondary or primary intervention allows the best use of available resources. Furthermore, a needs assessment can determine whether a community is equipped to carry out a prevention effort. At this stage, competent and willing organizations able to address the problem should be identified and their resources taken into consideration. Previous prevention programs have used a variety of community resources and organizations, including policy makers, schools, health service agencies (particularly those geared to serving adolescents and youth), parent groups, and law enforcement organizations.

The engagement of partners and allied organizations, gaining support for the strategy from the community through training for those involved in carrying out initiatives, maximization of participation from target populations through the use of incentives, reducing barriers by allowing for convenient transportation and other means that simplify and encourage involvement, and keeping control of the course and methods used through careful monitoring have been suggested as effective management strategies by the Substance Abuse and Mental Health Services Administration (SAMHSA). Other suggestions include combining multiple prevention strategies in order to improve results and using the media to ensure that preventive and educational messages reach a larger target audience. The SAMHSA campaigns in universal prevention initiatives point out the importance of evaluation and outcome measurement as well as a continuous process of assessment, design, implementation, and evaluation.

Conclusions

Prevention researchers have been advocating for prevention programs to move away from single-disorder initiatives and instead to target multiple risk behaviors given the many commonalities among the risk and protective factors across adolescent risky

behaviors (Battistich et al., 1996; Jessor, 1999). Furthermore, it is the interaction of protective and risk factors with the individual and his or her environment that ultimately determines the outcome of engaging in risky but possibly socially acceptable activities. Hence, the adoption of prevention programs that target multiple and general risk behaviors among adolescents and aim to enhance the positive development of children and adolescents by promoting protective factors and increasing resilience is likely a more advantageous approach to prevention of ICDs. Risk prevention must integrate the fostering of protective factors; however, protective factors alone cannot yield resilience since they only moderate or mitigate the effects of stressors. Hence, the promotion of resilience is likely the paramount overarching goal for prevention of ICDs.

Undoubtedly, prevention is the most important form of public health promotion. The need for intervention will continue, and professional treatment will remain essential. However, preventive efforts can minimize the occurrence of mental disorders, and there is much that can be done in this area to reduce the development of ICDs among at-risk groups. In addition, prevention is ultimately much less costly and surpasses treatment in terms of its desirability and psychosocial costs. It has the potential to reduce a great deal of human suffering. Prevention work can improve the potential and aptitude of children and adolescents, alleviate the burden of disease, and enhance the service capacity of health services and service providers. The best prevention methods use a community and collaborative approach and aim to prevent all mental disorders without focusing on one specific problem or pathology. Prevention can take the form of universal education before a problem begins, or it may attempt to focus on at-risk groups, or it may focus on minimizing harm for those who are already affected by subclinical symptoms of disease. The field of ICD prevention is in its infancy. More research, funding, and work needs to be done. While this is a daunting task, the rewards will be immeasurable.

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Assessment Instruments for Impulsivity and Impulse Control Disorders

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Abstract

This chapter provides a review of measures used to assess the impulse control disorders (ICDs) included in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., DSM-IV) and those proposed for DSM-V, as well as measures used to assess the related psychological construct of impulsivity implicated in the development and maintenance of ICDs. We first present instruments used to assess ICDs including diagnostic interviews, as well as self-report and clinician-rated measures of symptom severity. Second, we present self-report and behavioral measures of impulsivity. The review includes theoretical rationales, procedural details, and empirical evidence, including detailed psychometric data for each measure to provide a comprehensive guide to the assessment of ICDs and the construct of impulsivity.

Keywords: impulsivity, impulse control, assessment, psychometrics, behavioral task

The aim of this chapter is to describe and review the psychometric properties of measures used to assess the impulse control disorders (ICDs) included in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., DSM-IV; APA, 2000) and those proposed for DSM-V, as well as measures used to assess the related psychological construct of impulsivity. The first part of this chapter focuses on the instruments used to assess ICDs. This review includes diagnostic interviews, as well as self-report and clinician-rated measures of symptom severity. This review is not exhaustive, given the large number of measures for some disorders and the absence of published measures for others. Rather, our aim is to present a representative sample of reliable, valid, and commonly used measures for each of the ICDs. The second part of the chapter focuses on the measurement of impulsivity, a construct implicated in the development and maintenance of ICDs as well as other forms of psychopathology. In this section, we describe and present widely used behavioral measures of impulsivity with psychometric data

for each measure; it is notable that psychometric data for behavioral measures are less consistently available than for self-report measures. It is our hope that this chapter will provide an empirically and clinically useful review of measures of impulse control disorders and impulsivity.

Assessment of ICDs

Impulse control disorders are characterized by repetitive engagement in behavior (1) despite negative consequences; (2) perceived lack of control over the behavior; (3) a strong urge to perform the behavior; and (4) enjoyment or a sense of relief when the behavior is performed (Grant & Potenza, 2004). Impulse control disorders represent a compelling clinical challenge, and our understanding of these disorders and our effectiveness in treating them depend on the ability to conduct accurate and thorough assessments. A variety of instruments and assessment modalities have been developed to evaluate ICDs. The purpose of the structured clinical interviews is to provide a diagnosis of ICDs,

and they contain questions related to the symptom domains outlined above. By contrast, the functions of self-report measures are more varied, including screening for psychopathology; assessing symptom severity, distress, or functional impairment; and identifying disorder subtypes. Accordingly, some self-report measures assess symptoms directly, while others assess correlates of problem severity. In this section, each measure will be presented and its psychometric properties will be reviewed to the extent that this information is available. Table 38.1 contains basic properties and descriptive data on ICDs for some of the measures included in this section (this list includes only measures for which descriptive data were available). We will begin with a diagnostic interview developed to screen across ICDs; the remainder of this section will be organized by disorder. Intermittent explosive disorder (IED) and pyromania are not included because of the

unavailability of clinical measures specific to these disorders.

General Assessment of ICDs

MINNESOTA IMPULSIVE DISORDERS

INTERVIEW (MIDI)

The MIDI is a 36-item semistructured interview developed to screen and diagnose ICDs, including the disorders included in the DSM-IV (i.e., pyromania, kleptomania, trichotillomania, intermittent explosive disorder, pathological gambling) as well as those proposed for inclusion in the DSM-V (i.e., pathological skin picking, compulsive buying, and compulsive sexual behavior; Christenson et al., 1994). The MIDI is the only diagnostic interview that provides a comprehensive assessment of ICDs. Each module consists of a preliminary screening question and follow-up questions that correspond to the DSM-IV diagnostic criteria for each disorder.

Table 38.1 Means, Standard Deviations and Internal Consistencies for Self-Report Symptom Assessments for ICDs

	# of Items	Range	Clinical M (SD)	Control M (SD)	Internal Consistency
Gambling Symptom Assessment Scale ¹	12	0–48	23.9 (8.2)		.87
Massachusetts General Hospital Hair Pulling Scale ²	7	0–28	17.3 (5.1)		.85
MGH-HPS: Severity	4	0–16	9.5 (3.4)		.83
MGH-HPS: Control	3	0–12	7.7 (2.5)		.81
Milwaukee Inventory for Subtypes of Trichotillomania—Adult Version					
MIST-A: Automatic ³	10	0–90	25.7 (9.0)		.73
MIST-A: Focused ³	5	0–45	45.4 (16.2)		.77
Kleptomania Symptom Assessment Scale ⁴	11	0–44			
Skin Picking Scale ⁵	6	0–24	12.8 (3.8)	2.8 (2.6)	.80
Skin Picking Impact Scale ⁶	10	0–50	27.5 (16.6)	1.2 (3.5)	.88–.93
Compulsive Sexual Behavior Inventory ⁷					
CBSI: Control	13	13–65	31.7 (9.8)	57.2 (3.4)	.96
CBSI: Abuse	8	8–40	37.7 (2.8)	39.6 (.7)	.91
CBSI: Violence	7	7–35	33.7 (2.0)	34.7 (.8)	.88

¹Kim et al., 2009).

²Keuthen et al. (1995).

³Flessner et al. (2008).

⁴Grant & Kim (2002).

⁵Keuthen et al. (2001a).

⁶Keuthen et al. (2001b).

⁷Coleman et al. (2001).

If the respondent endorses the screening question, the interviewer assesses (1) urges to perform the behavior, (2) efforts to resist the urges, (3) feelings of tension prior to performing the behavior, (4) feelings of release after performing the behavior, and (5) distress and impairment associated with the behavior. Most modules also include disorder-specific questions to assess clinically relevant details (e.g., the purpose of theft in kleptomania, the location of hair pulled in trichotillomania). The modules include a combination of closed-ended questions (e.g., yes/no) and open-ended questions (e.g., the function or impact of the behavior). The MIDI is widely used in clinical and research settings (Black & Moyer, 1998; Grant et al., 2006a; Isaias et al., 2007; Lejoyeux et al., 2002; Weintraub et al., 2006). It requires 20 minutes to administer and does not require the interviewer to have extensive training or experience with ICDs.

Reliability. Neither the internal consistency of the items nor the interrater reliability of diagnoses made on the basis of this measure has been published.

Validity. Grant and colleagues examined the validity of the MIDI in a sample of psychiatric inpatients with a range of diagnoses (Grant et al., 2005). Probable cases were identified by the MIDI and followed up with disorder-specific diagnostic interviews. Based on the concordance of diagnoses made as a result of these interviews, the MIDI demonstrated good to excellent sensitivity and specificity for each of the disorders assessed: pathological gambling (sensitivity: 1.0, specificity: .98), kleptomania (sensitivity: .90, specificity: .93), IED (sensitivity: 1.00, specificity: .97), pyromania (sensitivity: 1.00, specificity: 1.00), trichotillomania (sensitivity: 1.00, specificity: .99), compulsive sexual behavior (sensitivity: .80, specificity: .97; Grant et al., 2005). Compulsive skin picking and compulsive shopping were not assessed in this study, although they are included on the MIDI. Despite promising data, little other psychometric data are available; accordingly, continued research is warranted to establish the psychometric properties of this instrument in psychiatric and nonclinical samples.

Pathological Gambling

THE STRUCTURED CLINICAL INTERVIEW FOR PATHOLOGICAL GAMBLING (SCI-PG)

The SCI-PG is a semistructured interview designed to provide a diagnosis of pathological gambling (Grant et al., 2004). The format and scoring of the SCI-PG were developed to be compatible with the SCID-IV (First et al., 1995). The SCI-PG

consists of 11 items corresponding to each of the DSM-IV diagnostic criteria for pathological gambling (10 inclusion criteria and 1 exclusion criterion). Symptom presence is scored on a scale from 1 to 3, where 1 reflects the absence of a symptom, 2 reflects a subthreshold symptom, and 3 indicates a clinically significant symptom. Five or more symptoms (out of 11) must be scored a 3 to make a diagnosis of pathological gambling. The SCI-PG requires approximately 15 minutes to administer, and the authors recommend that the interviewer be familiar with problem gambling because the respondent's answers may require clarification with appropriate follow-up questions (Grant et al., 2004).

Reliability. The reliability of the SCI-PG was evaluated in a sample of adult outpatients seeking pharmacological treatment for pathological gambling (Grant et al., 2004). The test-retest reliability (stability) of diagnoses was examined over a period of less than a week. Overall, test-retest stability of the diagnosis was excellent for the presence versus absence of pathological gambling ($\kappa = 1.00$); the test-retest reliability for the number of symptoms endorsed was also excellent ($r = .97$). Further, the individual items demonstrated good to excellent test-retest stability ($r = .87\text{--}1.00$), with the exception of the question assessing preoccupation with gambling ($r = .65$), which demonstrated marginal stability. Interrater reliability was evaluated in a randomly selected subsample of participants. To determine interrater reliability, both the interviewer and an observer scored the same assessment. The SCI-PG demonstrated excellent interrater reliability overall ($\kappa = 1.00$) in establishing the presence versus absence of the diagnosis. However, several of the questions, specifically those pertaining to the legal, interpersonal, and professional consequences of gambling, showed only marginal interrater reliability ($\kappa = .55\text{--}.66$). These findings highlight the need for the interviewers to have a shared operational definition of functional impairment.

Validity. The diagnostic of the SCI-PG (dichotomously scored) was moderately to strongly correlated with other measures of gambling symptom severity, including a clinician-rated interview ($r = .38$) and a self-report measure ($r = .78$). The SCI-PG-derived diagnosis was not significantly correlated with measures of anxiety ($r = .23$) or depression ($r = .19$), providing evidence for the divergent validity. Classification accuracy was examined in a separate sample of outpatients seeking treatment for ICDs at a specialty clinic. To establish classification accuracy, the SCI-PG diagnosis was compared to

diagnoses made by experts on the basis of longitudinal clinical observation. The SCI-PG demonstrated high sensitivity (.88) and specificity (1.00), as well as excellent positive predictive value (1.00) and adequate negative predictive value (.66). Although the psychometric properties of the SCI-PG are well characterized in clinical treatment-seeking samples, future research should evaluate its reliability and validity in community samples as well as general psychiatric samples. Additionally, research is needed to examine the sensitivity of the SCI-PG to symptom change over the course of treatment.

SOUTH OAKS GAMBLING SCREEN (SOGS)

Based on the DSM-III diagnostic criteria, the SOGS was developed to identify probable cases of pathological gambling (Lesieur & Blume, 1987). The SOGS is the most widely used self-report assessment instrument for pathological gambling (Petry & Amentano, 1999). It consists of 20 items that assess patterns of gambling, family and occupational disruption, use of deception to hide gambling losses, financial problems (e.g., defaulting on debts, borrowing from others to repay debts), and engagement in criminal activity to support gambling. Most items are scored as yes/no, although two require the participant to report the relative frequency of certain behaviors. The measure is scored by summing the affirmative responses. A score of 5 or more suggests probable pathological gambling.

Reliability. Stinchfield (2002) conducted principal components analyses to examine the factor structure of the SOGS in the general population and among problem gamblers. The analysis indicated a single primary factor for each group, which accounted for 21% of the variance in the general population sample and 31% of the variance in a sample of pathological gamblers. However, when these data were reanalyzed, Strong and colleagues (2004a) identified a small second factor that consisted of subjective self-evaluations (e.g., "Do you feel you have ever had a problem with gambling?"; Strong et al., 2004a). This factor appeared to be related to problem severity among pathological gamblers but not in nonclinical groups (Strong et al., 2004b). Strong et al. suggested that omission of these five subjective items could improve the reliability of the SOGS in nonclinical groups, but this modification has not been adopted widely in analogue sample studies.

Lesieur and Blume (1987) examined the internal consistency of the SOGS in a combined sample of pathological gamblers, students, and hospital

employees. The measure demonstrated excellent internal validity (Cronbach's $\alpha = .97$). In a subsequent sample of pathological gamblers and controls, the measure showed marginal internal consistency in a combined sample (Cronbach's $\alpha = .69$) but good internal consistency in the subsample of pathological gamblers (Cronbach's $\alpha = .86$). Test-retest reliability was examined over a period of 30 or more days in a separate sample of inpatients and outpatients in treatment for substance use disorders (Lesieur & Blume, 1987). The SOGS was scored dichotomously (i.e., presence-absence of diagnosis). Overall, test-retest reliability was adequate ($r = .71$), with greater reliability for outpatients ($r = 1.0$) than for inpatients ($r = .61$). The authors suggested that this discrepancy reflected the inpatients' awareness that their responses would be used as part of treatment planning (Lesieur & Blume 1987).

Validity. Construct validity was evidenced by the high rates of probable pathological gamblers identified in the sample recruited from Gamblers Anonymous (96.7%) and the low rates of probable pathological gamblers in the student and hospital employee samples (3.9% and 0.7%, respectively; Lesieur & Blume, 1987). Evidence for the concurrent validity of the measure was provided by correlations of the SOGS-based diagnosis with other indicators of problem gambling. For example, Lesieur and Blume (1987) reported high correlations between the SOGS score and DSM-III diagnostic criteria for pathological gambling. Similarly, Stinchfield (2002) found that the SOGS total score was significantly correlated with the number of DSM-IV diagnostic criteria in the general population ($r = .77$) and in the sample of pathological gamblers ($r = .83$). Further, the largest amount of money spent gambling in 1 day was significantly associated with the SOGS score in the general population ($r = .32$) and among pathological gamblers ($r = .35$).

Initial classification accuracy analyses suggest a score of 5 as the cutoff for probable pathological gambling (Lesieur & Blume, 1987). This cutoff correctly classified 98.1% of pathological gamblers, 96.3% of students, and 99.3% of hospital employees based on the DSM-III diagnostic criteria (Lesieur & Blume, 1987). Similarly, Stinchfield (2002) found that the cutoff of 5 demonstrated high positive predictive value (.96) and high negative predictive value (.90) in the sample of pathological gamblers when evaluated against the DSM-IV diagnostic criteria. On the other hand, in Stinchfield's (2002) general population sample, this cutoff

demonstrated marginal positive predictive value (.67) and poor negative predictive value (.50). Taking all findings into account, Stinchfield reported that the cutoff of 5 remains the best indicator of probable pathological gambling in combined samples, with an overall correct classification rate of 94% in the combined sample.

In summary, the SOGS is a self-report measure that can be used to identify probable cases of pathological gambling. Evidence largely supports its reliability and validity, but the measure is not particularly sensitive to change over time and its use to detect symptom improvement or exacerbation (i.e., in a treatment outcome study) is questionable. Furthermore, scores of 5 or 6 in the general population should be interpreted with caution, as they are associated with only a 50% chance of correctly classifying the individual.

GAMBLING SYMPTOM ASSESSMENT SCALE (G-SAS)

The G-SAS is a self-report measure of pathological gambling symptom severity that was developed to be sensitive to changes in symptoms over time (Kim et al., 2009). It is modeled after the Yale-Brown Obsessive Compulsive Scale for Pathological Gambling (PG-YBOCS; Hollander et al., 2005). The G-SAS does not provide a diagnosis, but instead assesses the average intensity, frequency, and/or duration of symptoms over the past 7-day period. It includes items to assess urges, thoughts, and behaviors as well as anticipatory tension and excitement, mental distress, and functional impairment associated with gambling. The G-SAS consists of 12 items, scored on a scale from 0 (minimal symptom presence) to 4 (maximum symptom presence, although the qualitative values vary for each item). Total scores range from 0 to 48, with higher scores reflecting greater symptom severity. It is notable that the version of the G-SAS described here deviates from the original measure (Grant & Kim, 2001), which consisted of 10 items scored from 0 to 8. To our knowledge, the earlier version did not undergo extensive psychometric evaluation; however, it has been in use for nearly a decade. Readers should be aware of this difference and use caution in interpreting G-SAS scores across studies.

Reliability. Kim and colleagues (2009) evaluated the internal consistency and test-retest reliability of the G-SAS in a sample of outpatients participating in a pharmacological study of treatment for pathological gambling (all participants were in the placebo condition). The G-SAS demonstrated good internal

consistency (Cronbach's $\alpha = .87$) at the baseline assessment. Participants completed the G-SAS 7 days apart during the first and second weeks of the treatment study. The mean G-SAS score demonstrated poor test-retest reliability ($\rho = .56$) in the full sample. However, when outliers were removed, the test-retest reliability of the mean score was stronger ($\rho = .79$). Kim and colleagues also examined the test-retest stability for each item. Seven items demonstrated moderate test-retest reliability over 1 week ($\kappa = .48\text{--}.61$), whereas five items showed only marginal test-retest reliability ($\kappa = .34\text{--}.39$). The lowest reliability coefficients were observed in items assessing urges, control over gambling-related thoughts, and distress associated with gambling. The low test-retest reliability may reflect the fact that, as a patient-rated scale, the G-SAS score can be influenced by idiosyncratic response styles. This possibility could be addressed by ensuring that participants understand the scoring system. On the other hand, the lower reliability may be due to perceived symptom improvement (even in the placebo group) early in treatment. Future research should examine test-retest reliability in an untreated sample.

Validity. Validity analyses were conducted in a sample of outpatients seeking pharmacological treatment for pathological gambling. The G-SAS total scores were moderately correlated with a clinician-rated scale of symptom severity ($\rho = .51$) at baseline, suggesting good concurrent validity. Moreover, in the sample that received the active drug ($n = 125$), the change score on the G-SAS was significantly correlated with the change score on the physician's rating of global improvement ($\rho = .69$) and with the participant's rating of global improvement ($\rho = .71$). Similarly, in the active drug group, the change score on the G-SAS was significantly associated with the change score on the clinician-rated measure ($\rho = .81$). Taken together, the findings suggest the validity of the G-SAS as a measure of change in symptom severity over the course of treatment. However, steps should be taken to improve the test-retest reliability of the G-SAS to accommodate its use as a primary outcome measure. Considering cutoffs to indicate symptom severity, Kim and colleagues (2009) have proposed clinical cutoffs. Specifically, a score of 8–20 suggests minimal symptoms, 21–30 reflects moderate symptoms, 31–40 indicates severe symptoms, and 41–48 reflects extreme symptoms. However, the rationale and empirical support for this classification are not clearly outlined, and the classification accuracy of

this system does not appear to be available in the literature.

Trichotillomania

TRICHOTILLOMANIA DIAGNOSTIC INTERVIEW (TDI)

The TDI is a clinician-administered semistructured interview developed to provide a diagnosis of trichotillomania based on the DSM-III-R criteria for the disorder, with modifications to accommodate revisions in the DSM-IV and DSM-IV-TR (Rothbaum & Ninan, 1994). Specifically, the modifications include the addition of a question to assess mounting tension before hair pulling and a question to assess clinically significant distress or impairment as a result of excessive hair pulling (Franklin & Tolin, 2007). Although the TDI is widely used to diagnose trichotillomania, no psychometric information for this interview is available (Franklin & Tolin, 2007).

MASSACHUSETTS GENERAL HOSPITAL HAIR PULLING SCALE (MGH-HPS)

The MGH-HPS is a commonly used self-report measure of trichotillomania symptom severity (Keuthen et al., 1995). It is a 7-item scale modeled after the Yale-Brown Obsessive Compulsive Scale (Y-BOCS; Goodman et al., 1989). The scale consists of seven items, which assess the frequency, intensity, and control over urges, as well as the frequency, resistance to, and control over actual hair pulling. The final item assesses distress related to hair pulling (Diefenbach et al., 2006; Keuthen et al., 1995, 1998). Participants rate the severity of their symptoms on a scale from 0 (no symptoms) to 4 (extreme symptoms). Total scores range from 0 to 28, with higher scores indicating greater symptom severity.

Reliability. The factor structure of the MGH-HPS was evaluated in a large Internet-based sample of individuals seeking support for trichotillomania (Keuthen et al., 2007). Exploratory factor analyses revealed two factors: Factor 1 was labeled *Severity* and consisted of urge frequency and severity, hair pulling frequency, and distress. This factor had an eigenvalue of 3.73, accounted for 53.2% of the variance in the total scale score, and demonstrated adequate internal consistency (Cronbach's $\alpha = .83$). Factor 2 was termed *Resistance and Control* and consisted of control over hair pulling, resistance to hair pulling, and control over urges. This factor had an eigenvalue of 1.26, accounted for 17.9% of the variance in the total scale score, and also demonstrated adequate internal consistency (Cronbach's

$\alpha = .81$). The authors speculated that these factors may reflect different pathways to symptom improvement, noting that medication may reduce symptom severity, whereas motivational or behavioral treatments may enhance resistance and control. Although this suggestion is intriguing, it does not yet have empirical support. Internal consistency has been replicated in a sample of adults seeking treatment for trichotillomania. The overall measure demonstrated adequate internal consistency (Cronbach's $\alpha = .80$) in the sample of treatment-seeking individuals with trichotillomania. Keuthen and colleagues reported similar internal consistency for the overall measure (Cronbach's $\alpha = .84$), and for each subscale (Keuthen et al., 2005).

Validity. The validity of the MGH-HPS was examined in a study of adults seeking treatment for trichotillomania. These participants completed the MGH-HPS as well as three clinician-rated measures of trichotillomania symptom severity, alopecia and global symptom severity, and self-report measures of depression and anxiety. The MGH-HPS total score correlated moderately with two clinician-rated measures of trichotillomania ($r = .52-.55$), providing evidence of convergent validity. However, the MGH-HPS total score was not significantly associated with clinicians' ratings of alopecia ($r = .10$), impairment due to trichotillomania ($r = .19$), or clinical global impression ($r = .13$), suggesting the divergent validity of the MGH-HPS. Further, the MGH-HPS was not significantly associated with depressive symptoms ($r = .26$), but was moderately, though not significantly, associated with anxiety ($r = .56$). Thus, the MGH-HPS appears to assess the severity of hair-pulling behavior rather than the extent of impairment associated with hair pulling.

The validity of the two-factor structure of the MGH-HPS was examined in an Internet-based sample of adults seeking support for trichotillomania. All participants completed the MGH-HPS and self-reported depression and anxiety, total disability, time spent resisting hair pulling, and time and money spent hiding hair loss. The total disability score was significantly positively associated with Severity ($r = .23$), but not was not associated with Resistance and Control ($r = .05$). Similarly, time spent resisting urges to pull hair was significantly and positively associated with Severity ($r = .22$) but negatively and quite modestly associated with Resistance and Control ($r = -.10$). Finally, the two factors were significantly correlated with one another ($r = .50$) and with the total score (Factor 1: $r = .91$; Factor 2: $r = .82$), suggesting internal validity.

MILWAUKEE INVENTORY FOR SUBTYPES

OF TRICHTOILLOMANIA—ADULT

VERSION (MIST-A)

The MIST-A is a self-report measure used to identify subtypes of trichotillomania, designed as a supplement to diagnostic and symptom severity instruments (Flessner et al., 2008). It consists of 15 items that assess the antecedents and consequences of hair pulling. The measure consists of two orthogonal subscales: the Automatic subscale consists of 5 items (e.g., “I am usually not aware of pulling my hair during a pulling episode”) and the Focused subscale consists of 10 items (e.g., “I pull my hair to control how I feel”). The measure is rated on a scale from 0 (“not true of any of my hair pulling”) to 9 (“true for all of my hair pulling”). Because the subscales are thought to reflect distinct functions, the total score should not be interpreted (Flessner et al., 2008). Subscale scores range from 0 to 45 for Automatic and from 0 to 90 for Focused, with higher scores indicating more frequent hair pulling of that type. In one study of chronic hair pullers, approximately 47% of research participants reported primarily automatic hair pulling and 34% reported predominantly focused hair pulling, while the remaining 19% reported a combination of the two (du Toit et al., 2001). Franklin and colleagues (2006) suggest that different subtypes may benefit from different interventions, highlighting the clinical relevance of this distinction.

Reliability. Exploratory and confirmatory factor analyses of the MIST-A have been conducted using an Internet-based sample of adults seeking support for trichotillomania (Flessner et al., 2008). Exploratory factor analyses conducted on 24 items revealed two orthogonal factors. The five-item Automatic factor had an eigenvalue of 3.13 and accounted for 13.0% of the variance in the total scale score. The 10-item Focused factor had an eigenvalue of 4.11 and accounted for 17.1% of the variance in the total scale score. Nine items did not load strongly on either factor and were eliminated; many of these items related to hair-pulling urges and resistance to urges. Confirmatory factor analysis conducted on the remaining 15 items revealed a fair fit to the data. The Automatic subscale demonstrated acceptable internal consistency (Cronbach's $\alpha = .73\text{--}.77$), as did the Focused subscale (Cronbach's $\alpha = .77\text{--}.78$). Future research is needed to support the factor structure of this measure and to investigate the test-retest stability of these subtypes.

Validity. The validity of the MIST-A was assessed in an Internet-based sample of adults seeking support for trichotillomania. Consistent with expectations,

the Automatic subscale was significantly negatively correlated with the proportion of time participants reported being aware of their hair pulling ($r = -.46$), providing support for the concurrent validity of this subscale. As evidence of its divergent validity, the Automatic subscale was not significantly associated with hair pulling to establish symmetry or experience a specific bodily sensation, nor was it associated with physical or mental anxiety prior to hair pulling ($r < .01$). Further, the Automatic subscale was only weakly associated with stress ($r = .15$) and anxiety ($r = .12$) and was unrelated to depression ($r = .05$). On the other hand, the Focused subscale was significantly associated with the frequency of hair-pulling episodes accompanied by a sense of physical and mental anxiety ($r = .25$ and $.32$, respectively). The Focused subscale was associated with the proportion of pulling episodes initiated with the goal of establishing symmetry ($r = .20$) and the proportion of pulling episodes initiated to experience a specific bodily sensation ($r = .35$). In addition, focused pulling was associated with greater depression ($r = .32$), anxiety ($r = .32$), and stress ($r = .36$), indicating an association between this form of hair pulling and emotional distress. Finally, the subscales were not correlated with one another ($r = .01$), supporting the independence of these subtypes. There is considerable evidence for the reliability and validity of the MIST-A. To supplement this evidence, future psychometric development should include establishing the reliability and validity of this measure in clinical samples. Further development will provide insight into the clinical utility of the measure and provide support for the validity of the subtypes.

Kleptomania

STRUCTURED CLINICAL INTERVIEW FOR KLEPTOMANIA (SCI-K)

The SCI-K is a semistructured interview developed to provide a standardized diagnosis of kleptomania based on the DSM-IV diagnostic criteria (Grant et al., 2006a). It was developed to be consistent with the SCID-IV in terms of format and scoring. The SCI-K consists of nine probes and related follow-up questions, which correspond to the DSM-IV diagnostic criteria for kleptomania. The first five probes query (1) recurrent impulses to steal, (2) recurrent failure to resist impulses, (3) stealing items not needed for personal or financial reasons, (4) increasing tension before stealing, (5) and relief or pleasure after stealing. The final four probes assess rule-out criteria: (6) stealing is not committed to express

anger or take revenge, (7) stealing is not committed in response to delusions or hallucinations, (8) stealing is not better accounted for by a manic episode, and (9) stealing is not better accounted for by antisocial personality disorder (Grant et al., 2006a). Questions are scored 1 (absent), 2 (subthreshold), or 3 (true or threshold). The interview requires approximately 20 minutes to administer. To make a diagnosis of trichotillomania, the first five questions must be scored 3.

Reliability. Internal consistency of the SCI-K was examined in a sample of adults seeking pharmacotherapy or psychotherapy for kleptomania (Grant et al., 2006a). Overall, the measure demonstrated excellent internal consistency (Cronbach's $\alpha = .96$). The internal consistency of the five inclusionary items was excellent (Cronbach's $\alpha = .93$), as was the internal consistency of the four exclusionary items (Cronbach's $\alpha = .98$). The SCI-K was administered at two time points 2–3 weeks apart prior to beginning treatment. The reliability of the diagnosis (kleptomania vs. nonkleptomania) was extremely high ($\Phi = .96$). Interrater reliability was based on a single interview, rated by the interviewer and an observer. The SCI-K demonstrated fair interrater reliability ($\Phi = .72$) for the diagnosis of kleptomania. Grant and colleagues (2006a) noted that there is disagreement over the validity of this diagnostic criterion, suggesting that the psychometric problems associated with this item may reflect as a weakness in the conceptualization of kleptomania rather than a particular limitation of the SCI-K.

Validity. Concurrent and divergent validity were assessed in a randomly selected subsample of the full patient sample. The SCI-K-based diagnoses were highly and significantly correlated with the number of symptoms endorsed on self-report measure of kleptomania ($\Phi = .77$), providing evidence of good concurrent validity. The SCI-K diagnosis was not significantly associated with clinician-rated depression severity ($r = -.02$), indicating good divergent validity. As noted previously, classification accuracy was examined in a separate sample of outpatients in treatment at an ICD specialty clinic ($n = 46$; 52.2% female). Clinical judgment based on the DSM-IV diagnostic criteria for kleptomania served as the standard against which the SCI-K was evaluated (Grant et al., 2006a). The SCI-K showed good sensitivity (.90) and specificity (.94). It correctly classified 81.8% of those with kleptomania (i.e., adequate positive predictive value) and 97.1% of those without kleptomania (i.e., good negative predictive value).

The preliminary psychometric data for the SCI-K are limited but encouraging. Findings suggest strong internal consistency, good concurrent and divergent validity, and adequate classification accuracy. However, it should be noted that the reliability and validity of the standards against which the SCI-K was judged (e.g., self-report measure based on diagnostic criteria, clinical impressions) have not been established. Continued research is necessary to support the reliability and validity of this measure in patient and nonpatient samples.

KLEPTOMANIA SYMPTOM ASSESSMENT SCALE (K-SAS)

The K-SAS is a self-report measure of kleptomania symptom severity developed to be sensitive to changes over time in symptom severity (Grant & Kim, 2002; Grant et al., 2005). It consists of 11 items, which assess severity, duration, and control over urges to steal (items 1–4), frequency, duration, and control over thoughts about stealing (items 5–7), excitement prior to and pleasure during theft (items 8 and 9), and distress and functional impairment caused by stealing (items 10 and 11). Each item is rated on a scale from 0 (no symptoms) to 4 (severe symptoms), though the qualitative values vary across questions. Possible scores range from 0 to 44, with higher scores indicating greater kleptomania symptom severity.

Reliability. The K-SAS demonstrated good internal consistency (Cronbach's $\alpha = .90$) in a sample of outpatients seeking treatment for kleptomania (Grant & Kim, 2002). Participants completed the K-SAS at baseline and again 7 days later, following a week-long placebo lead-in. Test-retest reliability was poor ($r = .57$), which may be due to the nature of the study (i.e., treatment).

Validity. As evidence of the construct validity of the K-SAS, participants receiving pharmacological treatment for kleptomania demonstrated significant decreases in K-SAS scores from baseline to the end of treatment (Grant & Kim, 2002). Over the course of the pharmacological treatment study, symptoms were assessed on a weekly basis using the K-SAS and clinician-rated measures (Global Assessment of Functioning, Clinical Global Impression). The K-SAS showed good concurrent validity with physician-rated Clinical Global Impression ($r = .63\text{--}.87$ over 10 weeks) and Global Assessment of Functioning ($r = -.88$ to $-.62$). In another study of outpatients with kleptomania, the K-SAS was significantly associated with deficits in executive function ($r = -.69$), such that greater symptom severity was associated

with fewer correct responses on the Wisconsin Card Sorting Test (Grant et al., 2007). Taken together, there is limited evidence for the validity of the K-SAS as a measure of symptom severity specifically, and further psychometric evaluation is warranted.

Pathologic Skin Picking

SKIN PICKING SCALE (SPS)

The SPS is a brief self-report instrument that aids in the identification of compulsive skin picking (Keuthen et al., 2001b). The six-item scale is modeled after the Y-BOCS and assesses the frequency and intensity of urges, time spent picking, and interference due to picking, as well as associated distress and impairment. Respondents rate each item on a 5-point scale from 0 (none) to 4 (extreme), with total scores ranging from 0 to 24. Higher scores suggest more severe compulsive skin picking.

Reliability. The psychometric properties of this measure were examined in a clinical sample of self-injurious skin pickers (i.e., patients who experienced significant tissue damage and distress or functional impairment due to their skin picking) and in a college sample of non-self-injurious skin pickers. The SPS demonstrated adequate internal consistency in the clinical sample ($\text{Cronbach's } \alpha = .80$). Internal consistency was not reported for the nonclinical group.

Validity. As evidence of construct validity, higher scores are associated with tension prior to picking and relief after picking, as well as objective measures of skin picking such as duration, distress, and impairment related to skin picking. Self-injurious skin pickers scored significantly higher on the measure than non-self-injurious skin pickers. Sensitivity and specificity analyses identified a score of 7 as the optimal cutoff to identify probable cases of compulsive skin picking. This cutoff correctly classified 96.4% of the self-injurious skin pickers and 92.2% of the non-self-injurious skin pickers. These results are promising, but continued psychometric development should focus on the test-retest reliability of this measure, its factor structure, and its ability to detect changes in symptoms over time.

SKIN PICKING IMPACT SCALE (SPIS)

Keuthen and colleagues (2001a) developed the SPIS, a measure of the psychosocial consequences associated with compulsive skin picking. The SPIS was designed to be used as part of a larger functional assessment of skin-picking behavior, acknowledging the need to conduct independent evaluations of symptom severity and degree of impairment.

The measure assesses emotional (e.g., “I feel embarrassed because of my skin picking”), social (e.g., “I don’t like people looking at me because of my skin picking”), and behavioral (e.g., “It takes me longer to go out because of my skin picking”) consequences of skin-picking behavior. The 10-item measure is scored using a scale from 0 (none) to 5 (severe), with possible scores ranging from 0 to 50. Higher scores indicate greater psychosocial impairment.

Reliability. The measure demonstrated excellent internal consistency in a sample of self-injurious skin pickers ($\text{Cronbach's } \alpha = .93$) and good internal consistency in a student sample of non-self-injurious skin pickers ($\text{Cronbach's } \alpha = .88$).

Validity. As expected, the clinical sample scored significantly higher than the student sample on the SPIS. All items were significantly correlated with the total score ($r = .60\text{--}.80$), suggesting that the SPIS was assessing a common construct. Participants completed self-reports of symptoms of depression and anxiety and characteristics of their skin-picking behavior in addition to the SPIS. Among self-injurious skin pickers, the SPIS total score was positively correlated with anxiety symptoms ($r = .52$) and depressive symptoms ($r = .42$), as well as with duration of daily skin picking ($r = .42$), satisfaction during picking ($r = .36$), and self-reported shame after picking ($r = .51$). In the non-self-injurious sample, the SPIS score was not significantly correlated with any index of psychological distress (depression: $r = -.04$, anxiety: $r = -.02$, shame: $r = .18$), nor was it associated with the daily duration of skin-picking episodes ($r = .01$). These findings provide support for the validity of the SPIS as a measure of distress and impairment among self-injurious skin pickers. Sensitivity and specificity analyses were conducted to determine the optimal cutoff to differentiate compulsive from normal skin picking. A cutoff of 7 correctly classified 83.9% of participants with self-injurious skin picking and 96.2% of participants with non-self-injurious skin picking.

Compulsive Buying

COMPULSIVE BUYING SCALE (CBS)

The CBS is a widely used self-report measure used to identify probable cases of compulsive buying (Faber & O’Guinn, 1992). It consists of seven items that address three aspects of problematic consumer behavior identified by Faber et al. (1987): (1) excessive spending (e.g., “If I have any money left at the end of the pay period, I just have to spend it”); (2) strong urges to shop (e.g., “Felt anxious or

nervous on the days I didn't go shopping"); and (3) a feeling of guilt after buying (e.g., "Felt others would be horrified if they knew of my spending habits"). Several items were developed to capture symptoms unique to compulsive buying, while others reflect symptoms common to all ICDs (e.g., difficulty controlling urges, tension prior to engaging in the behavior). The CBS is scored on a scale from 1 (strongly agree, very often) to 5 (strongly disagree or never), and total scores are calculated based on an equation with each item weighted by its difficulty. More negative scores reflect greater problem severity. A scaled score below -1.34 is classified as indicating a compulsive buyer.

Reliability. Reliability of the CBS was examined in a sample of self-referred treatment-seeking (but still untreated) individuals with compulsive shopping and a comparison sample recruited from the general population (Faber & O'Guinn, 1992). Factor analysis indicated a single factor (eigenvalue 4.45), which accounted for 64% of the variance in the CBS score. All items loaded strongly on the factor (the mean factor coefficient was .79). The CBS demonstrated excellent internal consistency in the clinical sample (Cronbach's α = .95; Faber & O'Guinn, 1992).

Validity. The sample recruited for validity analyses included probable compulsive buyers identified by the CBS in the general population screening, self-referred compulsive buyers, and general consumers (Faber & O'Guinn, 1992). Compulsive buyers differed significantly from general consumers on all psychological variables, including lower self-esteem, greater obsessive-compulsive symptomatology, and greater emotional lift and remorse following buying (Faber & O'Guinn, 1992). Concerning the financial outcomes, compulsive buyers had more debt and carried a monthly balance on more credit cards than did general consumers. Additionally, the CBS demonstrated good classification accuracy, correctly classifying 87.5% of participants with and without compulsive shopping. The CBS appears to be a reliable and valid instrument to screen for cases of compulsive buying in the general population. The test-retest stability of this measure was not evaluated in the initial validation study due to the anonymous nature of this questionnaire-based study, and it should be explored in future research. Moreover, in light of recent trends in consumer spending and household debt (see Bernthal et al., 2005; Cynamon & Fazzari, 2008), it may be worthwhile to confirm the validity of the clinical cutoff identified by Faber and O'Guinn in 1992.

Compulsive Sexual Behavior

COMPULSIVE SEXUAL BEHAVIOR INVENTORY (CSBI)

The CSBI is a self-report measure of compulsive sexual behavior that assesses the severity of this behavior (Coleman et al., 2001). It assesses hypersexuality, dyscontrol of sexual impulses and behavior, and theoretically relevant risk factors for compulsive sexual behavior. The CSBI is a 28-item instrument that consists of three subscales: Control, Abuse, and Violence. The scale is scored from 1 (very frequently) to 5 (never), with total scores ranging from 28 to 140. Lower scores indicate greater sexual compulsivity. Exploratory factor analysis revealed the three-factor solution (Control, Abuse, and Violence), which accounted for 58% of the variance in symptom severity in a combined sample of men with nonparaphilic sexual behavior, men with pedophilia, and healthy controls (Coleman et al., 2001). The Control subscale accounted for 42% of the variance (eigenvalue = 16.66), the Abuse subscale accounted for 10.1% of the variance (eigenvalue = 4.26), and the Violence subscale accounted for 5.9% of the variance (eigenvalue = 2.46). The CSBI demonstrated good to excellent internal consistency for each subscale (Control: Cronbach's α = .96, Abuse: Cronbach's α = .91, Violence: Cronbach's α = .88). The CSBI was administered twice to an Internet-based sample of men who have sex with men (Miner et al., 2007). The measure showed good test-retest reliability over 7–10 days (r = .86).

Validity. In the study of Coleman et al. (2001), pedophilic participants scored significantly lower on the Control subscale than participants with nonparaphilic compulsive sexual behavior or controls, suggesting greater difficulty with control of sexual thoughts and behaviors and providing evidence of construct validity. As further evidence, individuals designated as high in sexual compulsivity based on a median split reported more lifetime sexual partners, more sexual partners in the past 3 months, and more unprotected sexual encounters in the past 3 months (Miner et al., 2007). Pedophilic participants also scored lower on the Violence subscale than controls, suggesting greater engagement in problematic sexual behavior among the pedophilic group, whereas sexual compulsives did not differ significantly from either group on this subscale. No group differences were found for the Abuse subscale (Coleman et al., 2001).

The CSBI demonstrated satisfactory reliability and validity among male participants with a history of sexually compulsive behavior (Coleman et al., 2001) and men who have sex with men (Miner

et al., 2007). Continued research, using more varied samples that include female participants, is needed to identify the optimal clinical cutoff to differentiate individuals with and without compulsive sexual behavior. The concurrent and divergent validity of the measure should also be explored. Finally, continued psychometric evaluation is necessary to determine whether the factor structure, reliability, and validity of the CSBI apply to females with sexually compulsive behavior.

Assessment of Impulsivity

In the previous sections, we reviewed the assessment instruments for the ICDs included in the DSM-IV and those proposed for the DSM-V. In the remainder of this chapter, we will shift our focus to the assessment of the construct of impulsivity, a trait implicated in problematic behaviors that characterize each of the ICDs and other forms of psychopathology. *Impulsivity* is defined as an individual's tendency to behave impulsively, that is, rapidly, without forethought, and without regard for the consequences (Moeller et al., 2001) across a range of situations. Impulsivity is a broad multidimensional construct, and there is no single agreed-upon definition (Evenden, 1999). Indeed, the dimensions of impulsivity vary considerably as a function of discipline, theory, and measurement approach. In this section, we will describe the psychometric properties of three widely used self-report measures of impulsivity, followed by a review of behavioral instruments used to assess impulsivity.

Self-Report Measures

Self-report instruments have long been the dominant approach to the measurement of impulsivity. These measures rely on individuals to provide a subjective account of how they usually act, think, and feel across a range of situations. Due to space constraints, we have chosen to present an in-depth review of three of the most widely used measures of impulsivity, acknowledging that this brief list omits many influential and widely cited measures, including the EASI II Impulsivity Scales (Buss & Plomin, 1975), Dickman's Functional and Dysfunctional Impulsivity Scales (Dickman, 1990), the Impulsive Sensation Seeking scale of the Zuckerman-Kuhlman Personality Questionnaire (Zuckerman et al., 1993), the Impulsiveness subscale of the Temperament and Character Inventory (Cloninger et al., 1991), and the Control subscale of the Multidimensional Personality Questionnaire (Tellegen, 1982). We refer the reader to Schmidt (2003) for a thorough review of the

psychometric properties of self-report measures of impulsivity. Table 38.2 contains a summary of normative data for each of the measures presented here.

BARRATT IMPULSIVENESS SCALE (BIS-11)

The BIS-11 is the most widely used measure of trait impulsiveness, defined as an individual's tendency to think and behave impulsively across a range of situations (Patton et al., 1995). The BIS was developed to be orthogonal to anxiety-related traits, to distinguish multiple dimensions of impulsivity, and to differentiate this trait from related but distinct constructs, such as sensation seeking and risk taking (Stanford et al., 2009). Originally published in 1959 (Barratt, 1959), the BIS is currently in its 11th revision (BIS-11).

The BIS-11 is a 30-item measure scored on a scale from 1 (rarely/never) to 4 (almost always/always). Total scores range from 0 to 120, with higher scores indicating greater impulsivity. The BIS-11 consists of six first-order factors (attention, motor, self-control, cognitive complexity, perseverance, and cognitive instability) and three second-order factors (motor, non-planning, and attentional; Stanford et al., 2009). Motor impulsiveness, comprised of the first-order factors motor and perseverance, consists of 11 items and assesses the tendency to act quickly or on the spur of the moment and to give up easily (e.g., "I act 'on impulse'"). Nonplanning impulsiveness, comprised of the first-order factors of self-control and cognitive complexity, consists of 11 items and assesses the tendency to act without consideration for the future and a preference for challenging cognitive tasks (e.g., "I am a careful thinker" is reverse scored). Attentional impulsiveness, comprised of the first-order factors attention and cognitive instability, consists of eight items and assesses the ability to focus attention and concentrate (e.g., "I squirm at plays or lectures.").

Reliability An exploratory principal components analysis conducted on the BIS-10 revealed six first-order factors (attention, motor impulsiveness, self-control, cognitive complexity, perseverance, and cognitive stability) and three second-order factors (attentional, motor, non-planning). Notably, the statistically derived factor structure of the BIS-11 (motor, attentional, non-planning) was found to differ somewhat from the theory-based structure of the BIS-10 (motor, cognitive, non-planning; Barratt, 1985; Patton et al., 1995). Specifically, a number of items related to rapid or hasty decision making, which were initially conceptualized as part of the cognitive impulsiveness factor in the BIS-10, loaded

Table 38.2 Means, Standard Deviations and Internal Consistencies for Commonly Used Self-Report Measures of Impulsivity

	# of Items	Range	Male M (SD)	Female M (SD)	Total M (SD)	Internal Consistency
BIS-11 Total ¹	30	0–120	62.8 (9.2)	62.1 (10.6)	62.3 (10.3)	.83
Second-Order Subscales						
BIS-11 Attentional	8	0–32	16.8 (3.9)	16.7 (4.1)	16.7 (4.1)	.74
BIS-11 Motor	11	0–44	22.4 (3.4)	21.8 (4.1)	22.0 (4.0)	.59
BIS-11 Non-Planning	11	0–44	23.6 (4.5)	23.6 (5.0)	23.6 (4.9)	.72
First-Order Subscales						
BIS-11 Attention	5	0–20	10.3 (2.8)	10.4 (2.9)	10.4 (2.9)	.72
BIS-11 Motor	7	0–28	15.2 (2.8)	15.0 (3.4)	15.0 (3.2)	.64
BIS-11 Self-Control	6	0–24	12.4 (3.1)	12.0 (3.3)	12.1 (3.3)	.72
BIS-11 Cognitive Complexity	5	0–20	11.3 (2.4)	11.6 (2.6)	11.5 (2.6)	.48
BIS-11 Perseverance	4	0–15	7.2 (1.8)	6.8 (1.7)	6.9 (1.8)	.27
BIS-11 Cognitive Instability	3	0–12	6.4 (1.8)	6.3 (1.9)	6.4 (1.9)	.55
UPPS Urgency ²	12	0–48			30.7 (9.4)	.91
UPPS (lack of) Perseverance	10	0–40			31.8 (6.6)	.87
UPPS (lack of) Premeditation	11	0–44			36.7 (7.7)	.81
UPPS Sensation Seeking	12	0–48			38.1 (11.1)	.91
I-7 Impulsiveness ³	19	0–19	8.8 (4.3)	8.2 (4.4)		.84
I-7 Venturesomeness	16	0–16	10.6 (3.2)	8.3 (3.8)		.85
I-7 Empathy	19	0–19	11.2 (3.5)	14.3 (3.1)		.69

¹Stanford et al. (2009).

²Anestis et al. (2007).

³Eysenck et al. (1985).

on the non-planning factor in subsequent principal components analyses (Patton et al., 1995). The revised cognitive factor was renamed *attentional impulsiveness* to reflect the remaining items (e.g., racing thoughts, difficulty concentrating; Stanford et al., 2009). Despite consistent support for the modified factor structure, many sources continue to refer to the Attentional subscale as *Cognitive impulsiveness*. The second-order factors (i.e., attentional, motor and non-planning) have been found to correlate modestly with one another ($r = .39\text{--}.51$) and with the total score ($r = .76\text{--}.89$), suggesting that despite the existence of well-defined factors, the BIS-11 reflects a single underlying construct (Stanford et al., 2009).

In a recent large-scale study of the BIS-11, the overall scale demonstrated good internal consistency

(Cronbach's $\alpha = .83$). The internal consistencies for each of the first- and second-order subscales were poor to fair (see Table 38.2). This likely reflects the small number of items in each first- and second-order subscale. For the scale total, test-retest reliability was satisfactory ($\rho = .83$) over a 1-month test-retest interval. The test-retest reliability of each of the second-order factors was fair (attentional: $\rho = .61$, motor: $\rho = .67$, non-planning: $\rho = .72$), while the test-retest reliabilities for the first-order subscales ranged from poor to fair ($\rho = .23\text{--}.72$; Stanford et al., 2009).

Validity. A common use of the BIS-11 is to examine whether purportedly impulsive groups (i.e., offenders, patients with disinhibitory psychopathology) report greater impulsivity than healthy controls. The BIS-11 total scores have been found

to be higher among cocaine-dependent adults and ecstasy users relative to controls (Bond et al., 2004; Lane et al., 2007; Pleskac et al., 2008) and have been found to be higher among early-onset alcoholics relative to late-onset alcoholics (Dom et al., 2006). Similarly, BIS-11 scores were elevated among individuals with borderline personality disorder (Ferraz et al., 2009) and antisocial personality disorder (Dolan & Fullam, 2004; Warren & South, 2006) relative to healthy controls. In forensic samples, violent offenders scored higher than non-violent offenders (Smith et al., 2006). Group differences have also been found for specific subscales: for example, suicide attempters have been found to score higher than controls on the Motor (Dougherty et al., 2004) and Attentional subscales (Quednow et al., 2006), while Non-Planning has been shown to be elevated in both unipolar and bipolar depression (Peluso et al., 2007). Taken together, findings provide strong support for the construct validity of the BIS-11.

The BIS-11 total score has been found to be significantly associated with other self-report measures of impulsivity ($r = .63$) and disinhibition ($r = .36$), providing evidence of convergent validity (Stanford et al., 2009). Nonsignificant correlations with similar but theoretically unique personality traits (e.g., reward responsiveness: $r = -.04$; thrill seeking: $r = .11$) support the divergent validity of the measure. In addition, the BIS-11 has been found to be associated with indices of impulsive behavior. For example, among abstinent drug users in residential treatment, higher BIS-11 scores were associated with greater crack cocaine use (total: $r = .28$, motor: $r = .18$, attentional: $r = .24$, non-planning: $r = .28$; Lejuez et al., 2007). Similarly, the BIS total score was significantly associated with the frequency of clinically significant impulsive behavior ($r = .51$) and with distress ($r = .46$) and impairment ($r = .55$) resulting from impulsive behavior (Schmidt et al., 2004).

In their recent review, Stanford and colleagues (2009) made a number of recommendations with regard to scoring and interpretation of the BIS-11. First, they suggest that total scores between 52 and 71 should be considered within the normal range of impulsivity. Scores of 72 or greater (i.e., one or more standard deviations above the mean of the validation sample) can be used to designate high trait impulsivity, while scores below 52 (i.e., one or more standard deviations below the mean) may reflect either extreme inhibition or dishonesty (Helfritz & Stanford, 2006; Knyazev & Slobodskaya, 2006). Notably, the cutoff of 74 represents a slight revision to the cutoff of

72 suggested by Patton and colleagues (1995). To our knowledge, sensitivity and specificity analyses have not been conducted to establish the validity of this cutoff. However, there is evidence that young adults designated as high in impulsivity using this criterion were more than twice as likely to engage in risky behavior (e.g., deliberate self-harm, shoplifting) than individuals who scored in the normal range (Stanford et al., 2009), providing preliminary evidence for the construct validity of this cutoff.

Bearing these recommendations in mind, Stanford and colleagues (2009) suggest that the BIS-11 total score should be neither overinterpreted nor reported alone, given the multidimensional nature of this construct. Rather, in assessment contexts, each subscale provides unique information about the facets of impulsivity that characterize groups or individuals, which should be considered in conjunction with the total score. For example, different psychiatric groups may demonstrate elevations on certain subscales but not others (e.g., Tamam et al., 2008). However, despite the potential clinical utility of these subtraits, continued research is warranted to evaluate and improve the psychometric properties of the first- and second-order factors (Stanford et al., 2009).

EYSENCK IMPULSIVENESS SCALE (I-7)

The I-7 is a 54-item measure of impulsivity derived from Eysenck and colleagues' tridimensional model of personality (Eysenck et al., 1985). Within this framework, personality is defined by the biologically influenced traits neuroticism, extraversion, and psychotism. Trait impulsivity is thought to consist of two broad underlying factors: impulsiveness, which is related to psychotism, and venturesomeness, which is related to extraversion (Eysenck et al., 1985; Whiteside & Lynam, 2001). Accordingly, two of the three I-7 subscales are Impulsiveness (19 items, e.g., "Do you generally do or say things without stopping to think?") and Venturesomeness (16 items, e.g., "Do you sometimes like doing things that are a bit frightening?"). A third subscale, Empathy (19 items, e.g., "Do you become more irritated than sympathetic when you see someone cry?"), was originally included to avoid monotony (Luengo et al., 1991) and is not thought to comprise a dimension of impulsivity. Low correlations of Impulsiveness and Empathy and Venturesomeness and Empathy support this assertion (Eysenck et al., 1985; Miller et al., 2004). For all items, the participant responds either yes or no to each item; affirmative responses are scored as 1 and negative responses

are scored as 0 (12 items are reverse scored). Scores range from 0 to 54, with higher scores indicating greater impulsivity.

Reliability. Principal components analyses conducted in a sample of college students indicated 19 factors with eigenvalues greater than 1, which together accounted for 64.4% of the total variance (Luengo et al., 1991). However, when the model was limited to only three factors, results supported the original factor structure. Impulsiveness had an eigenvalue of 5.89 and accounted for 10.9% of the variance, Venturesomeness had an eigenvalue of 4.76 and accounted for 8.8% of the variance, and Empathy had an eigenvalue of 2.73 and accounted for 5.1% of the variance. The three-factor solution explained 24.8% of the total variance. While these findings have been interpreted as support for the factor structure of the I-7, others have noted that that this leaves a troubling amount of unexplained variance (Merenda, 1997; Tinsley & Tinsley, 1987). The factors were modestly correlated (Impulsiveness and Venturesomeness: $r = .18$, Impulsiveness and Empathy: $r = .15$, Venturesomeness and Empathy: $r = -.20$), indicating their general independence.

The I-7 demonstrated good internal consistency for Impulsiveness (Cronbach's $\alpha = .83$ for females, $.84$ for males) and Venturesomeness (Cronbach's $\alpha = .84$ for females, $.85$ for males). The Empathy subscale demonstrated fair internal consistency (Cronbach's $\alpha = .69$). Across studies, findings indicate good internal consistency of the Impulsiveness and Venturesomeness subscales (Luengo et al., 1991; Miller et al., 2004). Test-retest reliability was found to be fair to good for all subscales ($r = .76$ for Impulsiveness, $r = .80$ for Venturesomeness, and $r = .63$ for Empathy) over a 1-year test-retest interval (Luengo et al., 1991).

Validity. Modest correlations between Impulsiveness and Venturesomeness ($r = .24$ for males, $r = .11$ for females) provide some support for the independence of these constructs. Consistent with the Eysenck's conceptualization of the dimensions of impulsivity, Venturesomeness was associated with extraversion ($r = .39$ for males, $r = .22$ for females) and Impulsiveness was associated with psychotism ($r = .46$ for males, $r = .45$ for female). As further evidence of construct validity, participants designated high in impulsivity on the basis of their I-7 score (based on a median split) were found to score significantly higher on all subscales of the BIS-11, and reported greater hostility, anger, and physical and verbal aggression than the low-impulsivity group (Marsh et al., 2002). The I-7 also was found

to be associated with early substance use among adolescents and has been found to mediate the relationship between crack cocaine use (vs. heroin) and risky sexual behavior (Lejuez et al., 2005b).

The I-7 subscales consistently demonstrate strong convergent validity with other measures of impulsivity. For example, Miller and colleague (2004) examined the associations among subscales of four measures of impulsivity and disinhibition. They found that I-7 Impulsiveness was significantly associated with Motor, Non-Planning, and Cognitive Impulsivity on the BIS ($r = .52\text{--}.58$) and Dysfunctional Impulsivity on the Dickman Impulsivity Inventory ($r = .78$). Venturesomeness demonstrated a different pattern of correlations, and was associated with Functional Impulsivity ($r = .43$) and the Fun subscale of the BIS/BAS ($r = .47$). Evidence of divergent validity was provided by nonsignificant correlations between Impulsiveness and Empathy ($r = .03$ for males, $r = .06$ for females) and Venturesomeness and Empathy ($r = .01$ for males, $r = -.04$ for females; Aluja & Blanch, 2007).

UPPS IMPULSIVE BEHAVIOR SCALE (UPPS)

The UPPS Impulsive Behavior Scale is a more recently developed and widely adopted measure that addresses conceptual heterogeneity in theories of impulsivity and models of personality by identifying personality processes common across theories and assessment instruments (Whiteside & Lynam, 2001). In contrast to the BIS-11, the UPPS is not considered a measure of trait impulsivity; rather, the scales reflect distinct personality traits that lead to impulsive-type (i.e., disinhibited) behavior (Lynam & Miller, 2004; Whiteside & Lynam, 2001).

The UPPS is a 45-item measure¹ scored on a scale from 1 (strongly agree) to 4 (strongly disagree), with higher scores indicating greater impulsivity. It yields four subscale scores, but the total score is not interpretable. A series of factor analyses revealed four factors (urgency, lack of premeditation, lack of perseverance, and sensation seeking) thought to reflect distinct pathways to impulsive-type behavior. The Urgency subscale consists of 12 items and assesses the tendency to engage in undercontrolled behavior during periods of emotional distress² (e.g., "When I feel upset, I will often say things that I later regret"). The (lack of) Premeditation subscale consists of 11 items and assesses the tendency to act without considering the potential consequences of one's actions (e.g., "My thinking is usually careful and purposeful" is reverse scored.) The (lack of)

Perseverance consists of 10 items and assesses an individual's ability to persist on a task that is difficult or boring ("There are so many little jobs to be done that sometimes I just ignore them all"). Sensation Seeking consists of 12 items and assesses the tendency to pursue novel experiences and the tendency to seek and enjoy activities that are exciting ("I'll try anything once").

Reliability. As described above, exploratory factor analysis conducted on multiple self-report measures of impulsivity revealed four factors: Urgency, (lack of) Perseverance, (lack of) Premeditation, and Sensation Seeking, which together accounted for 66% of the variance in the total score (Whiteside & Lynam, 2001). In subsequent exploratory and confirmatory factor analyses, Magid and Colder (2007) found support for this factor structure, with eigenvalues ranging from 6.94 to 2.25. Sensation seeking was found to explain 54% of the variance, (lack of) Premeditation and Urgency each explained 52% of the variance, and (lack of) Perseverance accounted for 43% of the variance. The magnitude of correlations among the factors varies considerably across samples (e.g., Magid & Colder, 2007; Whiteside & Lynam, 2001; Whiteside et al., 2005), but the direction of the relationships is fairly consistent. In general, correlations of Sensation Seeking and (lack of) Perseverance have been the lowest ($r = .00\text{--}.03$) and correlations of (lack of) Premeditation with (lack of) Perseverance has been the highest ($r = .38\text{--}.65$; Magid & Colder, 2007; Whiteside & Lynam, 2001). The subscales demonstrated good to excellent internal consistency (Cronbach's $\alpha = .82\text{--}.91$; Whiteside & Lynam, 2001), which have been replicated in subsequent studies (e.g., Anestis et al., 2007). The subscales were moderately to strongly correlated across two assessments 3 to 4 weeks apart (Urgency: $r = .73$; Premeditation: $r = .73$, Perseverance: $r = .64$; Sensation Seeking: $r = .86$), suggesting adequate to good test-retest reliability (Anestis et al., 2007).

Validity. As evidence of the construct validity of this measure, the UPPS subscales have been found to differentiate among individuals with and without various forms of disinhibitory psychopathology (e.g., bulimia nervosa, substance use and borderline personality disorder, antisocial personality disorder; Anestis et al., 2007; Fischer et al., 2003; Magid & Colder, 2007; Whiteside & Lynam, 2003), as well as among individuals with and without lifetime histories of aggression (Ray et al., 2009; Whiteside et al., 2005). Preliminary evidence of convergent and divergent validity comes from the associations

of the UPPS subscales with distinct facets of personality, as measured by the NEO Personality Inventory-Revised (Costa & McCrae, 1992). For instance, Whiteside and Lynam (2001) found that Urgency loaded on a factor with neuroticism, Sensation Seeking loaded on extraversion, and both (lack of) Perseverance and (lack of) Premeditation loaded negatively on a factor with conscientiousness. Further support comes from studies examining the relationship of the UPPS subscales to clinically relevant variables. For example, Anestis and colleagues (2007) found that Urgency was positively correlated with depression ($r = .28$), anxiety ($r = .35$), bulimic symptoms ($r = .52$), and interpersonal difficulties (e.g., reassurance seeking; $r = .36$). In contrast, (lack of) Perseverance was associated with bulimic symptoms ($r = .37$) but none of the other psychopathology variables. Neither (lack of) Premeditation nor Sensation Seeking was associated with any of the clinical variables, suggesting the particular relevance of Urgency to certain forms of psychopathology. In a study examining the relationships of the UPPS subscales to eating disorder symptoms, Mobbs and colleagues (2008) found that Urgency was uniquely associated with concern for dieting ($r = .22$), while only (lack of) Perseverance was significantly associated with weight fluctuation ($r = .25$). None of the other subscales were significantly associated with other problematic eating, suggesting the divergent validity of these measures. Further, in a study examining the relationship of impulsivity to alcohol use, Sensation Seeking was positively associated with an index of frequency and quantity of alcohol consumption ($r = .27$) and positively associated with alcohol-related problems ($r = .21$). Sensation Seeking was also associated with motivations for substance use, including a moderate association with enhancement motives ($r = .30$) and social motives ($r = .21$), while Sensation Seeking was not associated with conformity motives ($r = -.03$; Magid et al., 2007).

Behavioral Measures of Impulsivity

Despite various strengths and a large body of empirical support for self-report measures of impulsivity, a variety of limitations also have been documented. Most notably, there are multiple threats to the accurate reporting of one's impulsivity, including poor self-evaluation skills, inability to report accurately on personal characteristics, or unwillingness to disclose information that may be perceived negatively by others (cf. Lejuez et al., 2002). Considering these limitations, there is great value in

the use of behavioral measurement to complement self-report data. Behavioral measures of impulsivity include measures that directly assess specific behaviors of interest using standardized laboratory-based computerized tasks. Recent evidence indicates that these assessments can be organized into three broad behavioral domains: impulsive decision making, inattention, and disinhibition (de Wit, 2009; Reynolds et al., 2008). Measures of decision making generally involve the participant making choices between rewards that are delayed/immediate or probabilistic/certain. Comparatively, measures of inattention do not involve the participant making choices; rather, they evaluate the participant's ability to maintain alertness and receptivity for a particular set of stimuli or changes in stimuli over time. Finally, measures of disinhibition emphasize the ability to inhibit prepotent motor responses or unwanted behaviors. These three categories of measures are statistically independent (Reynolds et al., 2008), and each is uniquely sensitive to acute drug effects (see below) and drug-using behavior (e.g., cigarette smoking; Fields et al., 2009). Such findings illustrate the importance of a multidimensional conceptualization of these behavioral measures, similar to the factor structures identified with self-report measures of impulsivity.

Behavioral measures of impulsivity have several advantages over self-report measures for certain types of research questions. A strength of behavioral measures is their suitability for repeated use in treatment studies and within-subjects designs (Dougherty et al., 2005), following appropriate methodological and/or statistical correction for learning effects and test-retest stability (e.g., employing a reliable change index or using alternative forms; Halperin et al., 1991). These measures of impulsivity are sensitive to state-dependent change in behavior, including pharmacological, physiological, and environmental manipulation (Dougherty et al., 2008; Swann et al., 2005). For example, behavioral measures reveal that administration of dopamine antagonists, alcohol, and MDMA, uniquely affects particular dimensions of impulsivity (Dougherty et al., 2005, 2008; Ramaekers & Kuypers, 2006), as does the phase of illness in bipolar disorder (Swann et al., 2003). Self-report measures of impulsivity are relatively less sensitive to such state changes in behavior because these assessments evaluate self-perceptions of behavior (i.e., the participant's ratings of his or her own behaviors) rather than the behavior itself.

Other advantages of using behavioral measures of impulsivity include their appropriateness for use

with young children and adolescents, as well as the availability of animal models for many of the measures used with humans. Compared with self-report measures, behavioral procedures do not require the capacity for abstraction during the assessment beyond the task itself. Therefore, young children who may have trouble accurately completing a self-report measure can still complete most behavioral measures. For the same reason (i.e., lack of need for abstraction), many of the behavioral measures used with humans have animal counterpart procedures (see Richards et al., 1997). This ability to extend research to animal models allows researchers to explore more feasibly certain types of issues that may be difficult to address in human studies—for example, the specific neural mechanisms associated with a type of impulsive behavior or drug effects on that behavior.

There also are disadvantages to using laboratory behavioral measures of impulsivity. Chief among them is that the measures are labor-intensive and time-consuming to administer. Most tasks require up to 15 minutes to complete (with some being considerably longer), and multiple measures must be administered to assess distinct dimensions of impulsivity (Dougherty et al., 2005). Adding to this complication is the fact that these assessments are state sensitive (the downside of the advantage noted above), meaning that it is important that the testing environments be nondistracting and consistent across participants. Because of these considerations, many of the research studies that include these assessments have small participant samples that are recruited from a small geographic area near the research facility. Consequently, few psychometric data are available for most of the measures described in this section. Thus, we will begin with a review of commonly used measures and consider psychometric properties for each measure to the extent that this information is available.

In the following sections, we review specific behavioral measures and how each measure operates. This review is not intended to be exhaustive but only a description of some of the more commonly reported behavioral measures. We have organized these measures according to the three dimensions of impulsive behavior described above. However, some of these measures have not been formally evaluated to determine which dimension of behavior they would best represent; in these cases, we base our categorization on conceptual and methodological similarities. Further, we include measures of risk taking and decision making, related constructs

that bear some relevance to impulsivity. Although constructs reflecting risk taking, decision making, and impulsivity are separable and conceptually distinct (Bechara, 2003; Lejuez et al., 2005a), they can be meaningfully conceptualized together under a broader umbrella of disinhibition (Lejeuz et al., 2002; Patterson et al., 1987). Furthermore, although all of these measures can broadly be considered assessments of impulsivity (i.e., by virtue of associations with impulsive behaviors like addiction, pathological gambling, or other clinical conditions), each still includes unique features. This qualification even applies to measures within a single dimension. For example, the Balloon Analogue Risk Task (BART) and the Experiential Discounting Task (EDT) are both broadly considered here to be measures of decision making because each involves the participant making decisions; however, the BART is an assessment of risk-taking propensity and the EDT an assessment of discounting by delay (see Shashwath et al., 2009, for comparison of these two measures). Therefore, measures that are grouped together dimensionally should not be considered the same but rather similar (or sharing certain characteristics) when compared against measures in other dimensions.

IMPULSIVE DECISION MAKING

Kirby Delay Discounting Measure

The Kirby is a brief, widely used measure of delay discounting. The measure, which has both pencil-and-paper and computerized forms, consists of 27 fixed choices between a smaller, immediate reward and a larger, delayed reward (Kirby & Marakovic, 1996). The Kirby can be administered as either a hypothetical or real-reward measure. For the real-reward option, the participant is informed that completing the questionnaire will make him or her eligible for a prize corresponding to one of the choices on the questionnaire. Typically, one of the 27 choice questions is selected at random and the participant receives whatever he or she chose for that question. For both hypothetical and real-reward administrations of the Kirby, the participant is encouraged to make each choice as if he or she will actually receive the chosen reward.

The delays for this measure range from 7 to 186 days. The Kirby is scored in a manner that results in k values (an index of delay discounting) assigned to participants along a range of 10 discrete steps: .00016, .00025, .00063, .0016, .0039, .010, .0126, .065, .16, and .25. This measure provides three separate k values for each participant for large (\$85, \$80, and \$75),

medium (\$60, \$55, and \$50), and small (\$35, \$30, and \$25) delayed monetary amounts. Values of k are assigned according to choice patterns across the 27 items, with more choices for the smaller immediate amounts associated with larger k values. Therefore, larger k values indicate greater delay discounting of value for the delayed options, which is associated with heightened impulsivity (Ainslie, 1975).

Although the Kirby is a widely cited and commonly used instrument, there are few data specific to psychometric properties of this measure or the construct of delay discounting more specifically. There are many variations on the delay discounting procedure, most of which follow the basic structure of the Kirby and differ only in the value of rewards used or the length of delay intervals. Because psychometric properties are generally consistent across these measures, we will review the psychometric evidence for delay discounting measures more broadly.

Reliability. Three studies have examined the test-retest stability of delay discounting paradigms. One study employed a hypothetical standard amount of \$1000 (Simpson & Vuchinich, 2000). Over a 1-week test-retest period, there was poor stability for individual equivalence points for short delays (1 week: $r = .03$; 1 month: $r = .24$) but good to excellent stability for equivalence points at longer delays (6 months: $r = .83$; 25 years: $r = .95$). In addition, the test-retest stability of the k value was excellent ($r = .91$; Simpson & Vuchinich, 2000). However, test-retest stability for hypothetical rewards was lower for smokers compared to nonsmokers (nonsmokers: $r = .90$; smokers: $r = .76$), suggesting that hypothetical delay discounting measures may be less reliable among more impulsive individuals (Bickel et al., 1999, 2003). However, when rewards were real (rather than hypothetical), test-retest stability was not found to differ across smokers and nonsmokers over a 1-week period ($r = .76-.77$; Bickel & Johnson, 2003). Another study used hypothetical rewards over a 3-month test-retest period. Findings suggested poor stability for individual indifference points at short delay intervals (1 week: $r = .16$), but stability was found to improve at longer delays (5 years: $r = .76$; 25 years: $r = .67$; Ohmura et al., 2006). Log k values were moderately correlated at 3 months ($r = .61$), indicating marginal test-retest stability. Overall, findings suggest good test-retest stability for k when the test-retest period is brief, rewards are hypothetical, and the sample is relatively low in impulsivity. Test-retest stability for k is marginal to fair at longer test-retest periods and when rewards are real.

The test-retest reliability of individual indifference points is poor for brief delays and good for long delays, so conclusions should be drawn cautiously from these data.

Consistency, which refers to the proportion of choices that are consistent with the assigned discount rate (k), is another index of reliability for delay discounting procedures (Kirby et al., 1999). Consistency was found to be high among opioid-dependent inpatients (94%) and healthy controls (96%; Kirby et al., 1999). Although this index of reliability is somewhat unconventional, it provides some support for the consistency of delay discounting procedures and for the hyperbolic estimation of k (Kirby et al., 1999).

Validity. As evidence of construct validity, substance-using participants who were designated as high in impulsivity on the basis of their clinical diagnoses demonstrated greater delay discounting than controls. For example, heroin users, smokers, and binge drinkers were all found to have higher (i.e., more impulsive) discounting rates than healthy volunteers (Kirby et al., 1999; Madden et al., 1997; Reynolds & Schiffbauer, 2004; Vuchinich & Simpson, 1998). Further, other findings suggest that pathological gamblers with substance use disorders discount more steeply than pathological gamblers without substance use disorders (Petry, 2001), suggesting an additive effect. Nevertheless, it is important to consider studies indicating that discounting rates did not differ among groups with childhood conduct disorder, alcohol dependence, and alcohol dependence with childhood conduct disorder (Bobova et al., 2009; Johnson et al., 2007).

Associations with other measures of impulsivity and disinhibition support the concurrent validity of the discount rate, k . For example, log-transformed k was modestly but significantly associated with subscales of the Eysenck Impulsiveness Questionnaire (impulsiveness: $r = .27$; venturesomeness: $r = .19$, $p < .05$; empathy: $r = -.19$, $p < .05$) and the BIS-10 (nonplanning: $r = .25$; cognitive: $r = .19$) in a combined sample of opioid-dependent inpatients and healthy controls. Similar associations have been found in samples of young adults (Richards et al., 1999): log-transformed k was correlated with disinhibition ($r = .45$), as measured by the Sensation Seeking Scale, as well as impulsivity ($r = .35$) and extraversion ($r = .36$) on the Eysenck Personality Inventory. The discounting rate is also correlated with a range of problematic behaviors, including the severity of problems associated with alcohol ($r = .24$), marijuana ($r = .19$), other drug use ($r = .20$), and

childhood conduct disorder ($r = .31$; Bobova et al., 2009). Moreover, the discounting rate value was found to prospectively predict postpartum relapse to smoking among women who stopped smoking during pregnancy (Yoon et al., 2007), providing support for the predictive validity of the measure. Thus, the delay discounting rate appears to have good construct and concurrent validity with measures of impulsivity and extent of impairment related to disinhibitory psychopathology.

Experiential Discounting Task (EDT)

The EDT is a computerized behavioral task that assesses an individual's propensity to discount the value of a reinforcer as a function of delay (i.e., delay discounting; Reynolds & Schiffbauer, 2004). Until recently, delay discounting measures relied largely on hypothetical question-based paradigms such as the Kirby. Critics of the hypothetical measures have questioned whether hypothetical scenarios based on long delays and large sums of money are ecologically valid, particularly in the absence of choice-contingent consequences and learning (e.g., Critchfield & Kollins, 2001). In addition, questions have been raised about the state dependence of delay discounting, as it has for other aspects of impulsivity (e.g., Dougherty et al., 2005, 2009). The EDT was developed in response to these concerns.

Consistent with the Kirby and other delay discounting paradigms, the EDT requires the participant to decide between a reward that is immediate and certain but smaller and one that is delayed and uncertain but larger. Unlike self-report measures, the participant experiences the consequences of his or her choice (including delay, probability, and reward from a coin dispenser) before making his or her next choice (Reynolds & Schiffbauer, 2004). The EDT is a forced-choice paradigm wherein the value of the rewards and the length of the delay vary across trials and choice blocks based on the participant's responses. Therefore, a participant's choices can be used to examine his or her discounting of reward value as a function of increasing delay.

Of the two choice options, the delayed standard amount is a larger, delayed, and probabilistic reward value (i.e., 30 cents at a 35% chance) against which all other response options are weighed. The only aspect of the delayed standard option that varies across choice sessions is the delay interval (e.g., 0, 7, 14, or 28 seconds). Alternatively, the adjusting immediate option is always delivered immediately and is 100% certain. The value of this immediate option begins at 15 cents (i.e., half the value of the

delayed standard option), but it is adjusted by a set percentage during a session according to the participant's choices. If the participant chooses the delayed standard option, the value of the adjusting amount increases for the next choice trial, thus increasing its choice value and making it more attractive for the next choice. However, if the participant chooses the immediate adjusting option, the value of the adjusting amount decreases for the next choice trial, thus making the standard option comparatively more attractive for the next choice. Adjusting the value of the immediate option in this way allows the determination of an indifference point for a given delay to the standard option, which is defined as the reward value of the adjusting option (determined by the participants choices) at which the participant chooses each choice option equally often (i.e., 50% of the time). At the point of indifference, the participant's choice pattern holds the adjusting option constant across choices (due to the manner in which adjustments are made), indicating that each choice option is of equal subjective value for the participant at the specified delay of the standard option. Indifference points are determined for each of the four delay intervals to the standard option and plotted to form a discount function. For each participant, the pattern of discounting reflected across his or her indifference points is characterized with either a hyperbolic decay model (e.g., Reynolds, 2006; Reynolds & Schiffbauer, 2004; Voon et al., 2010) or an area under the curve (e.g., Melanko et al., 2009; Reynolds et al., 2008; Shiels et al., 2009) method.

Reliability. The reliability of the EDT is the subject of ongoing research; however, the similarities to reliable conventional measures of delay discounting suggest promise in this regard.

Validity. As evidence of its construct validity, delay discounting values from the EDT have been found to be steeper among adolescent smokers than nonsmokers low in psychopathy traits relative to controls (Melanko et al., 2009) and in adult smokers compared to nonsmoking controls (Reynolds, 2006). Furthermore, among adolescents attempting to quit smoking, those who relapsed demonstrated greater discounting on the EDT. Consistent with predictions, participants performed more impulsively on the EDT when they were sleep deprived (Reynolds & Schiffbauer, 2004) and were less impulsive on the EDT following methylphenidate administration in children diagnosed with attention deficit hyperactivity disorder (ADHD; Shiels et al., 2009). These findings provide support for the construct

validity of the EDT and demonstrate its sensitivity to state-dependent fluctuations in impulsivity.

In support of its concurrent validity, EDT demonstrated significant correlations with conventional (i.e., question-based) measures of delay discounting ($r = .52$) among adults (Reynolds, 2006). Notably, the association between these measures was attenuated or absent in adolescents ($r = .10\text{--}.26$; Melanko et al., 2009; Reynolds et al., 2008), suggesting developmental differences in the relationships between measures. It has been suggested that this decreased association in younger participants may be caused by measurement error for the question-based measure, which was one reason the EDT was developed to be used with children. Nonetheless, even among adolescent participants, question-based delay discounting, probability discounting, and the EDT were found to load onto a single factor, suggesting that a similar form of impulsive decision making underlies all three measures (Reynolds et al., 2008). Not unexpectedly, EDT is not significantly associated with the BIS-11 total score (Melanko et al., 2009). Evidence for the divergent validity of this measure was provided by nonsignificant correlations of EDT with behavioral measures of impulsive disinhibition and impulsive inattention (Reynolds et al., 2008).

In the studies reviewed above, the EDT has demonstrated good construct concurrent and divergent validity in adults and adolescents. Moreover, its association with treatment response highlights its potential clinical utility beyond conventional measures of delay discounting. However, continued research is needed to determine its reliability.

Balloon Analogue Risk Task (BART)

The BART is a computerized measure of risk-taking propensity that models real-world risk behavior with the potential for reward as well as the risk of harm (Leigh, 1999; Lejuez et al., 2002). In the task, the participant is presented with a small balloon and asked to pump the balloon by clicking a button on the screen. With each click, the balloon inflates .3 cm and actual money is added to the participant's temporary winnings. At any point, the participant has the option to press a button labeled "Collect \$\$\$," which deposits the amount in temporary winnings in the bank (i.e., it can no longer be lost) and ends the trial, at which point a new trial begins. However, each balloon is programmed to pop somewhere between 1 and 128 pumps, with an average breakpoint of 64 pumps. If the participant fails to press "Collect \$\$\$" before the balloon pops, all earnings for that balloon are lost and the next

balloon is presented. Risk taking is defined as the average number of pumps on unpopped balloons (Bornovalova et al., 2005; Lejuez et al., 2002), with higher scores indicating greater risk taking. In addition, this task provides a measure of latency in milliseconds between pumps number/percentage popped balloons.

One critique of the BART is that the repeated pumping needed to inflate a balloon may actually be working against impulsive processes (i.e., a more impulsive response may actually be to collect earnings more quickly, rather than delaying reward as the balloon grows larger). For this reason, Pleskac and colleagues (2008) reported on an automatic version in which the participant enters the desired number of pumps and then watches the balloon inflate. If the number entered is less than the predetermined explosion point, the participant receives the amount of money associated with the number of pumps, whereas if the number entered is greater than the popping point, the balloon pops and the reward is lost.

Reliability. There are two types of reliability data for the BART. Split-half reliability (or, more accurately, split-third), comparing scores across the first third, middle third, and final third of trials, typically indicates strong correlations $>.7$ among the blocks (Lejuez et al., 2002, 2003). Adult participants tend to demonstrate modest increases over the blocks (typically between one and three pumps; Lejuez et al., 2002, 2003), while scores tend to decrease among adolescents (Lejuez et al., 2007). Data also have indicated test-retest reliability among young adults when the BART is administered three times in the same session, with modestly significant increases across administrations ($T_2-T_1\Delta = 2.2$, $T_3-T_1\Delta = 2.3$) and reasonably robust correlations among these administrations ($T_1/T_2 r = .79$, $T_1/T_3 r = .62$, $T_2/T_3 r = .82$; Lejuez et al., 2003). Additional evidence of reliability was indicated when the task was presented twice across a 2-week period, with a nonsignificant increase across administrations ($T_2-T_1\Delta = 1.2$) and a reasonably robust test-retest correlation ($T_1/T_2 r = .77$; White et al., 2008).

Validity. The BART is considered to be one of a small number of gold-standard measures of risk taking (Harrison et al., 2005), with adult data indicating its link to risky sexual behavior (Lejuez et al., 2002, 2004) and risky substance use (Bornovalova et al., 2005; Lejuez et al., 2002, 2003, 2005b; Pleskac et al., 2008). In adolescent studies, performance on the BART (a youth version; Lejuez et al., 2007) was found to correlate significantly with a variety of real-world risky behaviors; specifically, an

increase in risk taking propensity is associated with increased frequency of substance use, gambling, delinquency behaviors, and risky sexual behavior (Aklin et al., 2005; Lejuez et al., 2005b, 2007). It is notable that relationships of the BART to self-report measures of disinhibition are inconsistent; findings indicate modest, though significant, relationships found with sensation seeking ($r = \sim .20$) but typically nonsignificant relationships with self-report and other behavioral measures of impulsivity (Bornovalova et al., 2005; Lejuez et al., 2007).

Iowa Gambling Task (IGT)

The IGT is a decision-making task originally developed to examine decisional processes associated with neuropsychological impairment (e.g., Bechara et al., 1994; Rogers et al., 1999). At the start of the IGT, the participant is given \$2000 and instructed to maximize earnings over the course of 100 decision-making trials. The participant is provided with four decks of cards on a computer screen. As described by Bechara et al. (2001), the decks are labeled A, B, C, and D at the top end of each deck. All cards are identical, and each card is associated with hypothetical payoffs or losses (although versions with real financial contingencies are available). Cards from decks A and B pay an average of \$100, but the decks also contain cards with higher losses, and cards from decks C and D pay an average of \$50, but the losses are smaller. Accordingly, 10 draws from decks A and B (the “disadvantageous” decks) lead to a net loss of \$250, while 10 draws from decks C and D (the “advantageous” decks) lead to a net gain of \$250 (Bechara et al., 1994; Buelow & Suhr, 2009). Several dependent variables from the IGT indicate risky decision making, but the most widely reported indices are the number or percentage of disadvantageous choices over 100 trials, where larger values represent greater riskiness.

During the task, the participant clicks on a card from any of the four decks. Once the card is selected, the computer makes a sound similar to that of a slot machine. The selected card appears as either red or black, indicating whether money was lost or gained, and the value of the reward or loss appears at the top of the screen. Following this feedback, the card disappears and the participant selects another card. Each deck of cards is programmed to have 60 cards (30 red and 30 black), although the participant is unaware of how many cards of each type are in each deck. Losses are equally frequent in each deck.

Reliability. Data supporting the reliability of the IGT are limited. Findings have shown modest

test-retest stability over the course of three administrations in a single testing session ($r = .57\text{--}.59$; Lejuez et al., 2003). Moreover, repeated administrations have been found to lead to marked practice effects in neurologically healthy participants, with the percentage of disadvantageous decks dropping from 46 to 34 to 28 in the three administrations (Lejuez et al., 2003; for a review, see also Buelow & Suhr, 2009). For example, adolescents with disruptive behavior disorders failed to show improvement over 1 week (Ernst et al., 2003). No test-retest data are available for individuals with more compromised functioning (e.g., current substance users), who might display differential patterns of learning across multiple administrations compared to controls.

Validity. The IGT originally was developed to examine the processes underlying real-world decision-making deficits observed in neurologically impaired patients (e.g., Bechara et al., 1994; Rogers et al., 1999). However, there is a growing literature to support IGT impairments among clinical groups characterized by impulsive and high-risk behavior. For instance, IGT impairments have been found among individuals dependent on alcohol, cocaine, opioids, and marijuana relative to healthy controls (Bartzokis et al., 2000; Bechara & Damasio, 2002; Bechara et al., 2001; Bolla et al., 2003, 2005; Ernst et al., 2003; Goudriaan et al., 2006; Monterosso et al., 2001; Verdejo-Garcia & Perez-Garcia, 2007), with polysubstance users demonstrating even greater impairment (Bechara & Martin, 2004; Grant et al., 2000). Pathological gamblers also have been found to perform more poorly than controls on the IGT (Goudriaan et al., 2006). Findings with regard to ADHD are inconsistent (e.g., Ernst et al., 2003; Malloy-Diniz et al., 2007).

Although impulsive groups have been found to exhibit deficits on the IGT, Bechara (2003) has argued that impulsivity and decision making are cognitively and anatomically distinct processes, both of which may be involved in the initiation and maintenance of disinhibited behavior (Petry, 2001). In decision-making paradigms there is a problem that needs to be resolved by weighing competing options, a function associated with the ventral-medial cortex and the orbitofrontal cortex (Bechara, 2003; Rahman et al., 2001). In contrast, impulsivity does not require a solution, but rather inhibition of a dominant cognitive, motor, or behavioral response, functions associated with the lateral orbital and frontal cortices and the anterior cingulate (Konishi et al., 1999; Lombardi et al., 1999). Consistent with these findings, the IGT was found

to be associated with rate of delay discounting ($r = .29\text{--}.37$; Monterosso et al., 2001) but not with behavioral measures of impulsive disinhibition ($r = .08$) or impulsive inattention ($r = .05$; Stanford et al., 2009). Only a weak association was found between the IGT and the BART ($r = .14$; Lejuez et al., 2003), with other unpublished work often showing negative correlations. Further, despite findings of IGT deficits in clinical and nonclinical groups characterized by risky, disinhibited behavior, the IGT is weakly associated with lifetime engagement in clinically relevant impulsive behavior in adults ($r = .13$; McCloskey et al., 2009) and adolescents (substance use: $r = .22$; delinquency: $r = .12$; Aklin et al., 2005). Thus, the IGT has shown strong convergent validity with decision-making tasks and good divergent validity with measures of other facets of impulsivity. Data supporting the relationship among the IGT and real-world risky behavior is evident, but a handful of published studies fail to find this relationship.

IMPULSIVE INATTENTION

Conners' Continuous Performance Test (CPT-II)

The CPT-II is a computerized measure that assesses the ability to inhibit an ongoing motoric response (Conners, 1994, 2000). Stimuli consist of single letters, which are presented for 250 milliseconds at a variable rate of one every 600–1500 millisecond. The task consists of 360 trials and requires 14 minutes to administer. Participants are instructed to press the space bar as quickly as possible in response to each stimulus presentation unless the stimulus is the letter X. Impulsive responses are defined as errors of commission (i.e., responses to X).

Generally, continuous performance tasks require the detection of relatively infrequent target stimuli and are therefore used as measures of attention, vigilance, or executive processing (Strauss et al., 2006). In contrast, the CPT-II is considered a measure of impulsivity because it consists of 90% target stimuli (i.e., 90% of trials are letters other than X), which establishes responding as the dominant response, making response inhibition more difficult (Edwards et al., 2007; Epstein et al., 1998).

Reliability. Internal consistency of the CPT-II was assessed using split-half reliability in a large sample of adults, adolescents, and children (Conners, 1994). For errors of commission, internal consistency was found to be good ($r = .83$). Test-retest stability was assessed in a small sample of healthy and clinical adults over approximately 3 months. Test-retest stability of errors of commission was found to be

marginal ($r = .60\text{--}.69$; Strauss et al., 2006). Practice effects have not been examined. Low test-retest stability may limit interpretability of data from repeated administrations of the CPT-II (e.g., as a clinical outcome or in repeated measures designs).

Validity. As noted previously, errors of commission (*false alarms*) are the only dependent variable from the CPT-II thought to reflect impulsivity. However, most validation studies of the CPT-II have examined other performance indices as measures of attention and vigilance deficits associated with ADHD; accordingly, there are limited data to support its use as a measure of impulsivity specifically. As general evidence of construct validity, several clinical groups characterized by impulsivity have been found to demonstrate increased errors of commission. The most robust findings come from ADHD samples, in which both children and adults with ADHD have been found to make more errors of commission than controls (Losier et al., 1996; Malloy-Diniz et al., 2007;). Adults with alcohol dependence demonstrated a similar pattern of deficits (Salgado et al., 2009), as did patients with bipolar disorder in the manic, but not euthymic, phase of illness (Bora et al., 2006). Thus, the CPT-II appears to differentiate among certain clinical groups characterized by impulsivity, providing preliminary support for its construct validity.

Commission errors were found to be modestly correlated with all ADHD symptom clusters in both children and adults (Epstein et al., 2003; Rybak et al., 2007), providing some support for commission errors as a marker of impulsive inattention, although commission errors do not appear to be uniquely related to impulsive symptoms, indicating a lack of specificity. Bolstering these findings, commission errors were significantly associated with the BIS-11 total score ($r = .44$) and the discounting rate on a measure of delay discounting ($r = .41$) in a sample of adolescent smokers (Krishnan-Sarin et al., 2007), although other studies have failed to replicate this relationship (Malloy-Diniz et al., 2007). As evidence of divergent validity, no association has been found between commission errors and the Wisconsin Card Sorting Task or the IGT (Salgado et al., 2009), suggesting the independence of commission errors from executive function and impulsive decision making.

Immediate Memory Test and Delayed Memory Test (IMT/DMT)

The IMT and DMT are variations on the continuous performance test paradigm developed to assess rapid

response impulsivity (Dougherty et al., 2003b). Like the other continuous performance tests, the IMT and DMT require the participant to respond only when they detect a matched pair of stimuli (a target trial). The IMT requires participants to compare successive stimuli, while the DMT requires participants to compare every fourth stimulus, disregarding three presentations of filler stimuli (Dougherty et al., 2003c). Therefore, there is a 500-millisecond delay between comparison stimuli in the IMT and a 3500-millisecond delay between comparison stimuli in the DMT (Dougherty et al., 2003b). Stimuli consist of five-digit numbers presented for 500 milliseconds at a rate of one per second. The IMT and DMT are administered as a single task, with alternating blocks of IMT and DMT. The entire task consists of 1100 trials (550 of each type) and requires 21.5 minutes to administer. The measure yields two indices of impulsivity: percentage commission errors (i.e., false alarms), defined as responding to a stimulus that matches four of five digits, and the ratio of commission errors to correct detections (henceforth referred to as the *IMT ratio* and the *DMT ratio*).

Of note, the Continuous Performance Task—Identical Pairs version (CPT-IP; Cornblatt et al., 1988), which was initially developed as a test of vigilance, attention, and effortful processing, has also been employed as a measure of impulsivity (Ferraz et al., 2009; Walderhaug et al., 2002). This task is very similar to the IMT but has a lower number of *catch trials* (i.e., stimuli that are similar but not identical to the previously presented stimulus). The IMT and DMT have a greater percentage of target and catch trials than the CPT-IP (33% vs. 20% targets, 33% vs. 18% false alarms), which allows for greater variation in performance over the 1100 trials. As a result, the IMT and DMT are sensitive to impulsivity and performance deficits in higher-functioning populations (Dougherty et al., 2003b), whereas the CPT-IP shows the greatest sensitivity in more impaired or neurologically vulnerable samples (e.g., Cornblatt et al., 1988, 1989).

Reliability. The IMT and DMT are commonly employed in repeated measure designs, and the authors have stated that evidence for test-retest reliability is provided by the consistency of scores across multiple administrations within the same day and over multiple days (e.g., Dougherty et al., 2000, 2005). However, because the authors have not reported test-retest correlations, it is difficult to determine the stability of scores in relation to conventional standards of test-retest reliability.

Validity. There is considerable literature to support the construct validity of both indices of impulsivity derived from the IMT and DMT. Percentage of commission errors and IMT/DMT ratios on both tasks have been found to differentiate patients with bipolar disorder, borderline personality disorder and other personality disorders, and disruptive behavior disorders from healthy controls (Dougherty et al., 1999, 2003a; McCloskey et al., 2009). Among patients with bipolar disorder, IMT and DMT ratios and the percentage of commission errors have been found to differentiate those with and without a history of suicide attempt; further, among bipolar suicide attempts, patients with more medically severe attempts demonstrated greater impulsivity on the IMT and DMT (Swann et al., 2005). However, patients with schizophrenia did not differ from healthy controls in errors of commission on the IMT and actually made fewer errors of commission on the DMT (Dougherty et al., 1998), which may indicate the specificity of this deficit to disinhibitory psychopathology.

As further evidence of construct validity, participants designated high in impulsivity on the basis of the I-7 score (Eysenck et al., 1985) made a larger percentage of commission errors on both the IMT and the DMT relative to participants low in impulsivity (Marsh et al., 2002). Moreover, the IMT ratio was found to increase following alcohol consumption (Dougherty et al., 2008), suggesting the measure's sensitivity to state-dependent variability in impulsivity. In all, both indices of impulsivity (i.e., percentage of errors of commission and IMT and DMT ratios) have shown strong evidence of construct validity.

On the other hand, support for the concurrent validity of the IMT and DMT is somewhat inconsistent. The association of the BIS-11 subscales to the ratio of commission errors to correct detections has been found to vary considerably, from nonsignificant associations among healthy adults (Stanford et al., 2009) to modest associations (motor: $r = .21$; attentional: $r = .17$; non-planning: $r = .18$) in a combined sample of individuals with personality disorders and healthy controls (McCloskey et al., 2009). The association of IMT and DMT ratios with the BIS-11 was stronger among hospitalized adolescents with disruptive behavior disorders (total: $r = .35-.37$; motor: $r = .08-.14$; attentional: $r = .25-.27$; non-planning: $r = .32-.35$). Given the inconsistency of findings, it is difficult to draw conclusions about the concurrent validity of the IMT and DMT based on their associations with the BIS-11.

With regard to behavioral impulsivity measures, IMT and DMT indices were found to correlate moderately with one another (percentage commission errors: $r = .62$; IMT and DMT ratios: $r = .71$; Dougherty et al., 2003b; Marsh et al., 2002), suggesting that the IMT and DMT assess related, but not overlapping, constructs. Higher percentages of IMT and DMT commissions were associated with likelihood of inhibitory failure on behavioral tasks of impulsive disinhibition ($r = .24-.33$; Marsh et al., 2002), as were higher IMT and DMT ratios ($r = .39-.46$; Dougherty et al., 2003b). The IMT ratio was not associated with performance on tasks of impulsive decision making (i.e., the IGT or Two Choice Impulsivity Paradigm; Dougherty et al., 2009; Stanford et al., 2009), providing evidence of divergent validity.

In summary, the IMT and DMT are commonly used measures of impulsive behavior that have been used in a wide range of clinical and nonclinical populations. Despite their widespread use, including in repeated measures designs, there is limited information available regarding the reliability of these measures. Both the IMT and DMT demonstrate strong construct validity, and good concurrent and divergent validity with behavioral impulsivity measures in normal and clinical samples.

Impulsive Disinhibition

STOP-SIGNAL PROCEDURE

The Stop-Signal procedure is a computerized measure designed to study inhibitory control over an already-initiated response (Logan, 1994; Logan & Cowan, 1984). Logan and Cowan (1984) suggest that inhibitory control reflects competing go and stop processes; behavior depends on the finishing time of each process. If the stop process begins early enough, the response will be inhibited, and if the process begins late, the response will be executed. The Stop-Signal Procedure was developed to examine the conditions under which inhibition is likely to fail or succeed.

Stimuli consist of four letters: two are assigned to one response and two are assigned to another response. Participants respond by pressing keyboard keys with the index and middle fingers of their dominant hand. Each stimulus presentation is preceded by a presentation of a fixation point for 500 milliseconds. Stimuli are presented for 500 milliseconds at a rate of one every three seconds. The stop signal is a 500-millisecond, 900-Hz tone, which occurs at 1 of 10 delays (50–500 milliseconds in 50-millisecond increments) following the presentation of the

target stimulus. The Stop Signal Procedure consists of 800 trials, 25% of which are stop trials. Each delay is used 10% of the time. The measure yields two indices of impulsivity: (1) the probability of responding at any given delay, with higher proportions indicating greater impulsivity, and (2) the stop-signal reaction time, or the latency to respond in stop-signal trials, with slower (larger) response times indicating greater impulsivity.

The stop-signal reaction time (SSRT) cannot be observed directly and is therefore derived. Because of the theoretical complexity of some scoring approaches, Logan and colleagues have proposed an alternative stop-signal procedure to estimate this value (Logan et al., 1997; the reader is referred to Logan & Cowan, 1984, for the standard computational methods). Unlike the original procedure, this modified Stop-Signal Procedure uses adjusting values for the delays based on the participant's performance. That is, the delay increases by 50 milliseconds if the participant inhibits successfully and decreases by 50 milliseconds if the participant responds, eventually converging on the delay interval at which the participant inhibits correctly 50% of the time. This value represents the estimated stop-signal reaction time.

Of note, a related test of disinhibition is the go/stop Task (Dougherty et al., 2005), which builds on the Stop-Signal procedure. Go/stop uses only visual stimuli (rather than visual and auditory stimuli) and incorporates a motivational component; participants earn money for fast, correct responses, and lose money for slow or incorrect responses. We focus on the SSRT primarily because it has been used in a wider range of subject populations.

Reliability. To our knowledge, the test-retest stability of the Stop Signal Task has only been evaluated in children with (Kindlon et al., 1995) and without (Kuntsi et al., 2001) externalizing disorders. As a brief review, the Stop Signal Task yields two indices of impulsivity. The SSRT refers to the length of time it takes to inhibit a go response 50% of the time on stop trials. The second index, probability of inhibition, refers to the likelihood that the individual will inhibit successfully on a stop trial. The test-retest stability for probability of responding has been found to be poor ($r = .60$), as has the test-retest stability of the SSRT ($r = .23$) over a 2-week test-retest period (Kuntsi et al., 2001). Moreover, participants tended to improve significantly (i.e., demonstrated less impulsivity) on both indices from the first to the second testing, suggesting the likelihood of practice effects (Kuntsi et al.,

2001; Rodriguez-Fornells et al., 2002). These findings highlight the need for future research on the reliability of this measure, particularly in adult samples. Further, they raise questions about the utility of this instrument in repeated measures designs or as an indicator of treatment response.

Validity. The concerns regarding the reliability of SSRT impulsivity indices should not be ignored, as an instrument cannot be valid without being reliable. However, the Stop Signal Procedure is widely used and has a considerable body of research to support its utility as a measure of a certain dimension of impulsivity. Impulsivity is implicated in the development and maintenance of many forms of psychopathology and problematic behavior, yet a prolonged SSRT appears to be specific to certain forms of psychopathology, providing support for the construct validity of this measure. The most consistent findings support the specificity of this type of impulsivity to ADHD: adults with ADHD have shown longer SSRTs than healthy controls (Aron et al., 2003; Bekker et al., 2005; Chamberlain et al., 2007; Lampe et al., 2007). There is preliminary evidence suggesting that this deficit is apparent in patients with trichotillomania and obsessive-compulsive disorder but not in anxiety disorders more broadly (Chamberlain et al., 2006; Oosterlaan et al., 1998). Pathological gamblers with a history of childhood ADHD demonstrated prolonged SSRTs, while pathological gamblers without childhood ADHD did not differ from controls (Rodriguez-Jimenez et al., 2006). Prolonged SSRTs have also been found in groups with cocaine dependence, alcohol dependence, and methamphetamine use, although it is unclear whether these deficits are the result of substance use, premorbid inhibitory deficits, or a combination of the two (Fillmore & Rush, 2002; Goudriaan et al., 2008; Monterosso et al., 2005).

The construct validity of likelihood of inhibition is somewhat less definitive. Although children with ADHD demonstrate a clear deficit on this index relative to healthy controls (see Oosterlaan et al., 1998, for a review), there has been little evidence of impairment on this index in adult patients with ADHD, borderline personality disorder, or bipolar disorder (manic phase; Bekker et al., 2005; Clark et al., 2007; Gruber et al., 2007; Strakowski et al., 2009). Given the lack of findings with these disorders and other forms of psychopathology commonly associated with significant cognitive impairment (e.g., schizophrenia, depression; Gruber et al., 2007; Huddy et al., 2009), it is possible that this index is not sufficiently sensitive to impairment in most

adult samples. Although the construct validity of this index of impulsivity was upheld by its relationship with childhood ADHD, continued research is needed to clarify the relationship of this index with other variables of interest.

To evaluate the concurrent validity of the SSRT, a number of studies have examined its associations with self-report and other behavioral measures of impulsivity. Consistent with findings across behavioral assessment measures, there was limited concordance in self-reports of impulsivity and stop-signal indices. For example, in most studies, the SSRT has not been found to correlate with self-report measures of impulsivity (e.g., total score or any single factor of the BIS-11, the I-7, or the Control subscale of the Multidimensional Personality Questionnaire; e.g., Enticott et al., 2006; Reynolds et al., 2006), although rare studies have found associations (e.g., Lampe et al., 2007). On the other hand, the SSRT has been found to correlate with several behavioral indices of impulsivity, including errors of commission on a go/no-go task ($r = .28$; Reynolds et al., 2006) and errors of commission on a degraded CPT ($r = .34\text{--}.50$; Strakowski et al., 2009), both measures of impulsive inattention. Notably, the SSRT has not demonstrated associations with other behavioral measures of impulsivity (e.g., delay discounting, BART) or performance-based measures of executive function (e.g., the Wisconsin Card Sorting Test), supporting its divergent validity (Reynolds et al., 2006; Wodushek & Neumann, 2003). Findings suggest good concurrent validity with other measures of impulsive inattention.

Several studies have examined the associations of the SSRT with clinical outcomes. The SSRT has been found to correlate with self-reported ADHD symptom severity, anger, and aggression in children and adults (Lampe et al., 2007; Oosterlaan & Sergeant, 1996; Wodushek & Neumann, 2003) as a predictor of clinical outcomes, providing preliminary support for its clinical utility. For example, the SSRT combined with performance on a card-playing task predicted 53% of the variance in relapse following treatment for pathological gambling, whereas self-reported impulsivity was not predictive (Goudriaan et al., 2008). These findings are especially surprising given that pathological gamblers have not been found to differ from controls on SSRT (Rodriguez-Jimenez et al., 2006). In another study, the SSRT was associated with poorer outcomes among obese children participating in a weight reduction intervention. The extent of overweight was correlated with the SSRT throughout

the study ($r = .46\text{--}.50$) and was associated with less weight loss at the end of treatment and at 12-month follow-up (Nederkoorn et al., 2007). In general, findings support the potential predictive validity of the SSRT in a number of clinical outcomes, although continued research is needed to determine whether it is associated with treatment outcome above and beyond other clinically relevant variables.

Future Directions

1. Impulsivity has been conceptualized as a stable, nonaffective trait, yet theory, research, and clinical experience suggest that impulsive behavior is especially likely to occur during periods of affective distress or tension. How can laboratory-based behavioral measures of impulsivity be applied to the question of distress-induced impulsive behavior?

2. There is evidence that some behavioral measures of impulsivity predict a poorer treatment response and relapse to smoking, providing preliminary support for their clinical utility. Are there other clinical applications for these measures, for example, in terms of primary prevention, assessment, diagnosis, or idiographic treatment planning?

3. Behavioral impulsivity tasks typically examine impulsive responding to obtain an appetitive reinforcer. For example, the delay discounting literature is almost exclusively invested in responding to delayed positive reinforcers. As a complement to these paradigms, consider the inverse, where an aversive consequence loses its value as a function of delay. How can current impulsivity paradigms be adapted to reflect negative reinforcement processes?

4. Psychometric standards have not been established for behavioral assessment, as they have for conventional measurement approaches. To what extent should conventional standards of reliability and validity be applied to behavioral measures?

Notes

1. Since its initial development, the UPPS has been used as a 44-item measure (Lynam & Miller, 2004), a 45-item measure (Anestis et al., 2007; Magid & Colder, 2007), and a 46-item measure (Whiteside et al., 2005). We are not sure how to account for this variation, though it may reflect minor variations in the factor loadings of certain items across samples. We have chosen to include validity data from all UPPSs, as we do not believe that the removal or addition of a single item would markedly alter the pattern of the

- findings. In Table 38.2, we present the means and internal consistencies for the original 45-item measure using the largest sample available ($n = 70$, 82.9% female; Anestis et al., 2007). The reader is referred to Lynam and Miller (2004) for normative data for the 44-item measure in college and community samples.
- Notably, recent work has expanded the urgency construct to include positive urgency, the tendency to behave rashly when in a positive mood. The Positive Urgency Measure (PUM; Cyders & Smith, 2007) is often used in conjunction with the UPPS to understand relationships among impulsivity-related constructs and engagement in risky behavior (e.g., Cyders et al., 2009).
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Legal Aspects of Impulse Control Disorders

Jon E. Grant and Brian L. Odlaug

Abstract

Impulse control disorders present unique legal issues for the criminal justice system.

These behaviors require that the courts differentiate an uncontrolled act from an uncontrollable act. Kleptomania is by definition a disorder linked to illegal behaviors, whereas pathological gambling and compulsive sexual behavior are two impulse control disorders that are often associated with illegal activities for some individuals. Can and should a psychiatric disorder be used as a defense for criminal responsibility? This chapter will discuss the legal aspects of certain impulse control disorders and how courts have struggled to understand these complex behaviors from the criminal justice perspective.

Keywords: courts, gambling, impulse control, insanity, law, legal, shoplifting, theft

Introduction

Impulse control disorders present unique legal issues for the criminal justice system, patients, and clinicians. Not only do these disorders often result in illegal behaviors, but the behaviors in turn raise complex forensic issues. This chapter will discuss the legal aspects of certain impulse control disorders and examine the complicated status of impulsivity and impulsive behaviors under the law.

The law has grappled with the field of impulse control disorders in the area of criminal responsibility. Mental disease alone does not excuse a defendant from criminal responsibility. The mental disease must bear a relationship to the criminal behavior such that the defendant is morally not culpable. How to determine that relationship has been one of debate for decades in the law. In response to this difficult question, the law has established tests of criminal responsibility. These are often referred to as *insanity defenses*, and each state has adopted different tests with different names.

Although the insanity defense has existed since the twelfth century, initially it was not considered an

argument for the defendant to be found not guilty, but instead a way for a defendant to mitigate a sentence. The seminal case in insanity came from England and is now called the *M'Naghten rule*. In 1843, Daniel M'Naghten, an Englishman who was apparently a paranoid schizophrenic under the delusion that he was being persecuted, shot and killed Edward Drummond, secretary to British Prime Minister Robert Peel. M'Naghten was under the delusion that Drummond was Peel. M'Naghten was found not guilty on the grounds that he was insane at the time of his act. The case led to a standard regarding the defense of insanity. The M'Naghten rule states: "To establish a defense on the ground of insanity, it must be clearly proved that, at the time of the committing of the act, the party accused was laboring under such a defect of reason, from disease of mind, as not to know the nature and quality of the act he was doing; or if he did know it, that he did not know he was doing what was wrong" (*M'Naghten's Case*, 1843). The test to determine if defendants can distinguish right from wrong is based on the idea that they must know the difference in order to be convicted of a crime.

The *irresistible impulse defense* is another test of criminal responsibility designed to address defendants who know that their acts are against the law but who cannot control their impulse to commit them. This test is often combined with the M'Naghten rule in many jurisdictions. It suggests that the defendant is not liable if, by reason of mental illness, he or she is unable to exert control over his or her actions (Weil, 1989). Generally, this is construed to apply only to impulses that arise suddenly and are acted upon without reflection. How can one differentiate an uncontrolled act from an uncontrollable act? Also, courts have been loath to believe that most mental illnesses impair the will to such an extent that the person is a mere robot giving in to his or her impulses.

A slightly different standard was devised by the American Law Institute and is currently used in 18 states: “*a person* is not responsible for criminal conduct if at the *time* of such conduct as a *result* of mental disease or defect he lacks substantial capacity either to appreciate the wrongfulness of his conduct or to conform his conduct to the *requirements of law*.” This standard asks whether defendants have a substantial incapacity to appreciate the criminality of their conduct or to conform their conduct to the law (Bromberg, 1992).

The Federal Insanity Defense Reform Act holds: “It is an affirmative defense to a prosecution under any Federal statute that, at the time of the commission of the acts constituting the offense, the defendant, as a result of a severe mental disease or defect, was unable to appreciate the nature and quality or the wrongfulness of his acts. Mental disease or defect does not otherwise constitute a defense” (18 US Code, Section 17, “Insanity Defense”(a) “Affirmative Defense”). The federal statute therefore does not use the irresistible impulse test, and it is the defendant who must prove his or her inability to appreciate the nature and quality of the crime.

Kleptomania

No other psychiatric disorder (except drug use disorders) has the status of kleptomania. That is, the disorder itself is defined by an illegal activity. Whereas someone can be diagnosed with pedophilia simply by having urges or thoughts about children (without ever having acted on the behavior), or someone can have pyromania and yet never set a fire, a person can be diagnosed with kleptomania only if he or she steals.

The diagnostic criteria for kleptomania are (1) recurrent failure to resist impulses to steal objects

that are not needed for personal use or for their monetary value; (2) increasing sense of tension immediately before committing the theft; (3) pleasure, gratification, or relief at the time of committing the theft; (4) stealing is not done to express anger or vengeance and is not in response to a delusion or a hallucination; (5) stealing is not better accounted for by conduct disorder, a manic episode, or antisocial personality disorder (APA, 2000).

Not All Shoplifting Is Kleptomania

Not all shoplifters meet diagnostic criteria for kleptomania. The exact prevalence of shoplifting is unknown (as many who steal are never caught), but one study found that 10% of randomly chosen customers followed while shopping were observed stealing (Astor, 1969). In fact, it is estimated that more than \$13 billion worth of goods are stolen from retailers each year, which translates into more than \$35 million per day (National Association for Shoplifting Prevention, 2010). The majority of shoplifters are described as amateurs with sporadic activity, who have no known history of criminal activity, and who steal for their own consumption rather than for resale (Cox et al., 1990). Studies involving apprehended, legally referred shoplifters indicate that shoplifting may be more common in women (ranging from 52% to 100%) than in men. But as with kleptomania, these rates may be falsely elevated because women may be more likely than men to be referred for psychiatric evaluation. Male shoplifters are more likely to be apprehended during adolescence and early adulthood, whereas female shoplifters are more likely to be apprehended during puberty/early adulthood and around the time of menopause (McElroy et al., 1991).

Rates of kleptomania among people who are arrested for shoplifting have ranged from 0% to 8% (McElroy et al., 1991). According to the DSM-IV, fewer than 5% of shoplifters are identified as having kleptomania (APA, 2000). These rates may be falsely low due to incomplete psychiatric evaluations, lack of strict diagnostic criteria for kleptomania, and selection bias in these samples (McElroy et al., 1991).

A study that compared kleptomaniacs to shoplifters interviewed directly after apprehension found that 58% of the shoplifters were male compared to only 32.4% of the individuals with kleptomania (Sarasalo et al., 1996). Although none of the shoplifters met DSM criteria for kleptomania, approximately one-fifth had not stolen for personal use and had eventually discarded the object (Sarasalo et al., 1996). The study also found that both groups

reported the same degree of impulsivity and “a feeling of not being oneself.” On the other hand, kleptomaniacs reported a relatively greater number of previous thefts compared to shoplifters, which supports the compulsive aspect of kleptomania. The study found that many shoplifters, although they do not meet criteria for kleptomania, share characteristics with kleptomaniacs and therefore may benefit from treatment (Sarasalo et al., 1996).

Adolescent Stealing

Given that both typical shoplifting and kleptomania may start at a relatively early age, it is important to briefly examine adolescent stealing. A recent study of adolescents ($n = 3999$) found that the overall prevalence of stealing was 15.2%, and 29 students (0.72% of the entire sample, 4.6% of those who steal) endorsed stealing symptoms consistent with a DSM-IV diagnosis of kleptomania. For all students who reported stealing, the stealing behavior was associated with a wide range of antisocial behaviors such as drug and alcohol use, regular smoking, and poor school performance (Grant et al., in press). Another study examining the prevalence of impulse control disorders in a college population ($n = 791$) found that 0.38% of students reported criteria consistent with kleptomania (Odlaug & Grant, 2010).

Legal Aspects of Kleptomania

A study of 22 adults diagnosed with kleptomania revealed that they stole, on average, 2.3 times a week and had urges 3.7 days per week (Grant & Kim, 2002). Studies have reported that 64% to 87% of kleptomania subjects have been apprehended (McElroy et al., 1991; Sarasalo et al., 1996). In a study of 101 individuals with kleptomania, 69 (68.3%) had been arrested at least once as a result of their shoplifting (mean of 2.1 ± 1.8 arrests per subject with an arrest history; Grant et al., 2009b). In addition, 36.6% had been arrested but not convicted, and 20.8% had been convicted and incarcerated (Grant et al., 2009b). Of those who were apprehended, 79% reported that urges to steal were virtually abolished, but only for an average of 3.5 days. Therefore, apprehension does not appear to provide a sustained treatment for the urge.

Kleptomania is a severe disorder characterized by illegal behavior. People who suffer from this behavior are often apprehended and not infrequently jailed (Grant et al., 2009a). Suicidal thoughts are common in kleptomania, particularly when there are legal repercussions. Clinicians are

advised to have their patients seek legal advice when apprehended.

Kleptomania and the Court System

In Ohio, a defendant was caught stealing multiple clothing items from a store. The defense experts diagnosed her with kleptomania, stating that she did not steal for the monetary value of the items. She was found guilty. On appeal, the court found that the preplanned nature of the crime and the fact that she did not steal random items argued against an irresistible impulse to steal and therefore upheld the verdict (*State v Weber*, 1999). The verdict suggests that if the courts could be presented with a more archetypal case of kleptomania, the diagnosis might be a defense against theft.

Instead of kleptomania being a complete defense against criminal charges, courts have considered whether it should be used to mitigate a sentence. In 1997, a defendant accused of stealing from a department store was sentenced to 11 months in the county jail. On appeal, the defendant reported that she had a long history of shoplifting and had been diagnosed with kleptomania. The court used her history of shoplifting as an enhancement factor and her diagnosis of kleptomania as a mitigating factor. In the end, the sentence was upheld on appeal but she was allowed to serve it on probation (*State v Downey*, 1997).

Other courts have also not been willing to consider kleptomania as a mitigating factor in sentencing. One court, when refusing to dismiss a case based on a kleptomania defense, compared kleptomania to drug addiction and described crimes committed by drug addicts as motivated by factors such as illness, but stated that having an illness was not a valid defense of the behavior (*People v Meyers*, 2005).

The problem with kleptomania as a mitigating factor is that the diagnosis necessitates a history of stealing that can be characterized as kleptomania. This history then becomes a possible enhancement factor in sentencing, as it shows a long history of criminal behavior that courts may use to increase the defendant’s sentence (Novak, 2010). Therefore, raising the kleptomania defense may worsen instead of aiding a defendant’s case.

Pathological Gambling

Although gambling is generally not illegal, people meeting criteria for pathological gambling are more likely to have legal issues. In fact, one of the DSM-IV criteria for pathological gambling includes illegal behaviors: the individual “has committed

illegal acts such as forgery, fraud, theft, or embezzlement to finance gambling" (APA, 2000).

Pathological gambling has long been associated with crime (Blaszczynski & McConaghay, 1987; Lesieur, 1987; Rosenthal & Lorenz, 1992). The prevalence of criminal activity among pathological gamblers has been estimated to range from 20% to 80% (Blaszczynski, 1994; Blaszczynski et al., 1989; Brown, 1987). In one study involving 109 pathological gamblers, 55% reported having committed a crime related to gambling and 21% had been charged with a crime (Blaszczynski et al., 1989). Illegal activities of pathological gamblers often include writing bad checks, embezzlement, robbery, blackmail, tax fraud, and prostitution (Petry, 2004).

Whether there is a causal relationship between criminal behavior and problem gambling remains unclear. Some gamblers resort to illegal activities to fund their gambling or pay debts (Blaszczynski & McConaghay, 1994). As losses mount, the pressure to commit illegal acts increases. In two surveys of Gamblers Anonymous attendants, 46% to 56% reported that they had stolen something to gamble, and 39% reported having been arrested (Lesieur & Anderson 1995; Thompson et al., 1996).

Alternatively, both crime and gambling may simply be behaviors associated with antisocial personality disorder. Approximately 15% to 40% of individuals with pathological gambling suffer from comorbid antisocial personality disorder (Argo & Black, 2004; Black & Moyer, 1998; Slutske et al., 2001) (compared to rates of 1%-3% in the general population). Similarly, approximately 26% of prison inmates are pathological gamblers (Templer et al., 1993). In a study of gamblers calling a helpline, those who reported gambling-related illegal behaviors were more likely to have a severe gambling problem, owe debts to acquaintances, have received mental health treatment, have a substance use disorder, and have features of antisocial personality disorder (Potenza et al., 2001).

Among adolescents, groups with serious gambling-related problems are twice as likely to report involvement in illegal activities (Jacobs, 2004). Illegal activities among adolescent gamblers include stealing from their families or from others. In fact, illegal activities may be more prevalent among youthful gamblers due to their limited ability to generate funds and due to peer pressure.

Gambling and the Courts

In *United States v Iaconetti* (1999), the defendant pled guilty to the charge of cocaine possession with

intent to distribute. His defense at sentencing was that he suffered from pathological gambling, had enormous debts from gambling, and sold cocaine to help pay those debts. The defendant did not have a history of criminal behavior prior to his gambling addiction. The court found that a departure from federal sentencing guidelines was appropriate given that the criminal behavior was "causally connected" to the gambling.

In *United States v Grillo* (2004), gambling was again raised as a means of reducing the sentence of a defendant who was found guilty of mail theft and fraud. The defense claimed that when the defendant had money to gamble, he did not steal; he stole only when he had no funds for gambling. The court determined that a reduced sentence was allowed only if the conduct stemming from the mental disorder constituted the crime itself, but not if the mental disorder either had a direct causal connection to the crime or provided a motive for the crime.

The *Grillo* case shows that courts do not want to support a defense that might be abused. The court stated that many crimes could be committed for a variety of motives and that the courts should not allow all those motives to affect sentencing. In addition, the court aligned itself with the Seventh Circuit, which has held that the mental disorder must significantly impair the defendant's capacity to control his or her conduct at the time of the offense (*United States v Roach*, 2002). The *Grillo* court also explicitly stated that this defense would preclude a reduced sentence in the case of a compulsive shopper who stole because he or she had run out of money.

These cases demonstrate that although a defendant may receive a reduced sentence due to pathological gambling in some jurisdictions, it is rare.

Gamblers as Plaintiffs

Although pathological gambling may provide a limited defense against criminal charges, the courts have also considered whether a pathological gambler has any cause of action under the law.

In *Broan v Argosy Gaming Company* (2004), the plaintiff brought suit against the Argosy Casino, claiming that he had a gambling addiction and that Argosy was negligent in failing to exercise reasonable care after being notified by the plaintiff's wife that the plaintiff was a pathological gambler who was ruining the family finances. In fact, the plaintiff's gambling had led to his inability to make mortgage payments, disconnection of telephone and water services, and loss of insurance. The court held

that not only does a gambler not have a cause of action against a casino, but that a family member (i.e., a spouse) also fails to have a cause of action.

In *Williams v Aztar Casino* (2003), David Williams began gambling at a casino and soon developed a pathological gambling problem. At the time his problem became unmanageable (he was \$160,000 in debt), his girlfriend contacted the casino and informed them that Williams' compulsive gambling had led to financial debt and depression and that he was contemplating suicide. In response to her pleas, two members of the casino's Responsible Gaming Committee approached Williams in the casino to discuss his gambling habits. Later that night, he checked into a local mental health facility, where he was subsequently committed. The casino then sent Williams a "cease admissions" letter barring him from the casino. Williams, however, returned to the casino without difficulty. He then sued the casino, claiming breach of the duty of care. Although the court's decision focused largely on jurisdictional issues, the court reinforced the idea that casino operators are not under a duty of care (under common law) to protect gambling addicts from their own addictive and injurious behavior. Therefore, if states wish to allow such suits, they must provide a statutory means for plaintiffs to sue casinos.

Compulsive Sexual Behavior

Although compulsive sexual behavior is not inherently illegal, it is often accompanied by illegal activities, including the use of illegal substances (Kafka, 2010). Research has found lifetime rates of substance use disorders in 34% to 71% of individuals with compulsive sexual behavior (Black et al., 1997; Carnes, 1998; Kafka & Hennen, 2002; Raymond et al., 2002; Wines, 1997). The behavior itself appears to be quite common, although large epidemiological studies assessing prevalence in the general population have yet to be obtained. In a study of 791 college students, 29 (3.67%) reported symptoms consistent with proposed diagnostic criteria for compulsive sexual behavior (Odlaug & Grant, 2010). Individuals with compulsive sexual behavior report purchasing the services of prostitutes, viewing child pornography, and even engaging in sexual violence (Kafka & Hennen, 2002; Kafka & Prentky, 2002).

Compulsive Sexual Behavior and the Courts

In *United States v McBroom* (1998), the defendant was charged with possession of child pornography. At sentencing, the defendant showed that he

suffered from compulsive sexual behavior, that he was "obsessed" with Internet pornography, and that his compulsion reduced his mental state and led to the commission of the offense. The court found that his compulsion reduced his ability to control his behavior and was integrally linked with his crime.

Conversely, in the case of *Winston v Maine Technical College System* (1993), a tenured English instructor who kissed an 18-year-old student in one of his classes asserted that he was "sexually obsessive" and unable to control his impulses. Records indicated that the defendant had had previous inappropriate sexual encounters with students and had talked to his department chair prior to this current incident about seeking out prostitutes and being bothered by these impulses. The court ruled against the instructor, stating that disability protections did not apply to his case: "We further note that the DSM-III-R label has not been officially identified to include sexual addiction, that defendant's psychiatrist's opinion is based on a very broad 'process' addiction model representing the view of a 'subset' of physicians, and that the Americans with Disabilities Act of 1990 (ADA) also specifically excludes 'sexual behavior disorders from the term 'disability.'" The question remains whether inclusion of sexual compulsion in the DSM would change the legal system's view of this behavior.

Summary

The area of impulse control disorders raises unique and unsettled questions of law. First, what does it mean legally to be unable to control one's impulses? The courts have started to grapple with this issue in terms of criminal liability, but it is far from settled. The courts have even hinted at the heterogeneity within impulse control disorders. Perhaps individuals whose impulse control disorder is linked to criminal behavior (e.g., those with compulsive sexual disorder) should be treated differently under the law than those whose criminal behavior is merely a possible extension of the impulse control disorder (e.g., pathological gamblers who embezzle).

Furthermore, as neuroscience advances, what role will this information play in legal arenas? For example, if one could show that there was an objective abnormality in brain functioning at the time of the criminal behavior, would and should that affect sentencing? In one case of pyromania, neuroimaging (a single photon emission computed tomography scan showing perfusion deficits) was used to argue that the ability to resist the fire-setting behavior was

compromised (Grant, 2006). The neuroimaging findings were used to reduce the defendant's sentence (unpublished court opinion). Will this, in turn, provide an opportunity for the law to further revise criminal statutes in keeping with biological knowledge?

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Impulse Control Disorders and Impulsivity: Future Directions

Marc N. Potenza and Jon E. Grant

Abstract

Although research funding for impulse control disorders has been lacking, the last several years have seen an explosion in the amount of research and clinical information on these fairly common and debilitating behaviors. This information has been helpful in developing improved prevention and treatment strategies for many people. Despite these advances, substantial gaps remain in our understanding of impulse control disorders. The field of impulse control disorders therefore remains an important frontier in clinical research.

Keywords: funding, research, advances, treatment, prevention

Despite the description of impulse control disorders for more than a century, they had arguably received relatively little attention from researchers and clinicians for decades. Over the past decade, however, considerable research has begun to examine these disorders, as evidenced by the contributions to this volume. Despite these efforts, substantial gaps remain in our understanding of impulse control disorders. For example, formal diagnostic criteria in the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., Text Revision; DSM-IV-TR; American Psychiatric Association, 2000) exist only for a subset of disorders (intermittent explosive disorder, kleptomania, pathological gambling, pyromania, and trichotillomania), with the remainder relegated to a “not otherwise specified” category. The absence of formal diagnostic criteria for impulse control disorders involving compulsive sexual behaviors, compulsive shopping, problematic Internet use, and pathological nail biting or skin picking inhibits research, prevention, and treatment efforts for individuals suffering from these conditions. Furthermore, there exists debate regarding how best to categorize these disorders (e.g., as

impulse control disorders, addictions, obsessive-compulsive disorders, or disorders specific to the domain of impaired control, such as compulsive sexual behaviors [or hypersexuality] as a sexual disorder and binge eating disorder as an eating disorder). This debate occurs with a relative deficiency of information about how the impulse control disorders relate to one another and to other mental health conditions. It has been proposed that one of the best-characterized impulse control disorders, pathological gambling, should be moved from its current placement within the category of “Impulse Control Disorders Not Elsewhere Categorized” to an “Addictions and Related Disorders” category in DSM-V based on existing data (Holden, 2010; Petry, 2006; Potenza, 2006; Potenza et al., 2009). However, the paucity of information on other disorders, such as problematic Internet use, has precluded similar recommendations. Therefore, diagnostic criteria for a broader range of impulse control disorders are needed. It would be logical for experts to convene and develop provisional criteria that could be field-tested and refined based on data gathered and then utilized in clinical and research settings.

Progress in the study, prevention, and research of impulse control disorders has arguably been hampered by limited funding. Researchers in this field have reported that no institute within the National Institutes of Health considers impulse control disorders to be a significant priority. This situation does not appear likely to change in the foreseeable future given the slow recovery from the recent economic downturn. Unless impulse control disorders have a higher priority, existing gaps in knowledge will probably be addressed slowly. As such, basic information (e.g., regarding the prevalence of these disorders and the basic sociodemographic and clinical correlates) may not be obtained quickly. Additionally, prevention and treatment efforts may lag behind those of many other psychiatric disorders, especially given the paucity of empirically validated treatments for impulse control disorders. For example, no drugs currently carry an indication (as approved by the U.S. Food and Drug Administration) for any of the formal DSM-IV-TR impulse control disorders.

Like the impulse control disorders, the construct of impulsivity has received more research and clinical attention over the past decade. Nonetheless, significant gaps remain in our understanding of impulsivity, its core components, and their relevance to theoretically related constructs and specific aspects of different psychiatric disorders. Research over the past decade has found that impulsivity factors into two or more dissociable domains (e.g., response and choice impulsivity) and that these components may relate to specific aspects of psychiatrically relevant behaviors in predictive, consequential, or interactive ways (de Wit, 2008; Potenza & de Wit, 2010; Potenza & Taylor, 2009; Verdejo-Garcia et al., 2008). However, assessing impulsivity is a complex undertaking. For example, self-report and behavioral assessments of impulsivity, even within the same domain, may not correlate substantially with one another and thus may be differentially related to clinically relevant measures like treatment outcome (Krishnan-Sarin et al., 2007). Furthermore, behavioral and self-report measures of impulsivity may factor separately from one another and from measures of theoretically related constructs (e.g., risk taking), suggesting that a broad range of measures may help us better understand how impulsivity relates to specific behaviors and disorders (Meda et al., 2009). As impulsivity may represent an important endophenotype for multiple psychiatric disorders, and one that may link more closely to underlying biological mechanisms than to psychiatric diagnoses, such efforts warrant further

investigation (Fineberg et al., 2010). Additionally, developmental changes in impulsivity are important to consider with respect to the development and progression of psychiatric disorders, and changes in impulsivity may occur with the development of psychiatric conditions. With respect to this latter point, it has been proposed that in many conditions (e.g., substance use disorders, impulse control disorders), impulsivity may transition to compulsivity as the disorders become more entrenched (Brewer & Potenza, 2008; Everitt & Robbins, 2005; Jentsch & Taylor, 1999). Thus, understanding these constructs, their core components, their underlying biological mechanisms, and changes within these over time seems particularly relevant to mental disease and health. The importance of such considerations is highlighted by their relevance to the treatment of psychiatric disorders, with impulsivity predicting the treatment outcome in some studies (Krishnan-Sarin et al., 2007) and with changes in impulsivity correlating with changes in disorder-specific measures (e.g., the severity of problem gambling in pathological gambling) in others (Blanco et al., 2009).

One experienced researcher recently described the field of impulse control disorders as an important remaining frontier in clinical research. While significant advances have been made over the past decade, significant work remains to be done. A research society devoted to the study of impulsivity and impulse control disorders (the International Society for Research on Impulsivity and Impulse Control Disorders [ISRI]; www.impulsivity.org) has been formed and has held annual research meetings since its inception in 2004. Through sustained and directed research efforts, there is great potential to gain information that will be helpful in developing improved prevention and treatment strategies for many people.

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