Behavioral Addictions

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Faculty
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Faculty Disclosure
Contributing faculty, Mark Rose, BS, MA, LP, has disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

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The division planners and director have disclosed no relevant financial relationship with any product manufacturer or service provider mentioned.

Audience
This course is designed for mental health practitioners who may intervene in diagnosing and treating behavioral addictions in their patients.

Accreditations & Approvals
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Designations of Credit

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NetCE designates this continuing education activity for 6 NBCC clock hours.

NetCE designates this continuing education activity for 15 continuing education hours for addiction professionals.

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Course Objective

The purpose of this course is to provide clinicians with the knowledge and skills to appropriately identify, diagnose, and treat behavioral addictions.

Learning Objectives

Upon completion of this course, you should be able to:

1. Outline the evolution of and concepts forming the basis of behavioral addictions.
2. Compare and contrast explanatory models of addiction and their applicability to behavioral addictions.
3. Describe the factors underlying child and adolescent vulnerability to behavioral addictions.
4. Discuss psychologic and pharmacologic treatments that are generally appropriate for behavioral addiction.
5. Analyze the epidemiology, comorbidity, and risk factors of gambling disorder.
6. Evaluate the diagnosis and treatment of gambling disorder.
7. Review the pathophysiology, diagnosis, and treatment of addictive sexual behavior.
8. Describe Internet gaming disorder and how it is diagnosed and treated.
9. Explain how binge eating disorder is identified and appropriately treated.
10. Outline the epidemiology, risk factors, and clinical characteristics of compulsive buying disorder.
11. Analyze factors related to the diagnosis and treatment of compulsive buying disorder.
12. Discuss the epidemiology, comorbidities, and pathophysiology of trichotillomania.
13. List approaches to the treatment of trichotillomania.
14. Identify patients with kleptomania and create an effective treatment plan for these patients.
15. Describe behavioral addictions induced by dopamine agonists and other medications and disorders.

EVIDENCE-BASED PRACTICE RECOMMENDATION

Sections marked with this symbol include evidence-based practice recommendations. The level of evidence and/or strength of recommendation, as provided by the evidence-based source, are also included so you may determine the validity or relevance of the information. These sections may be used in conjunction with the course material for better application to your daily practice.

INTRODUCTION

The term "addiction" is traditionally used to describe the pathologic behavioral patterns a subset of persons exhibit from exposure to substances with central nervous system (CNS) activity (e.g., heroin, cocaine, alcohol). However, addiction is not a unitary construct; it incorporates common features that include repetitive engagement in rewarding (at least initially) behaviors, loss of control (spiraling engagement over time), persistence despite consequences, aversive states when ingestion is halted or substantially cut back, and an appetitive urge or craving state prior to engaging in the behavior. Many of these pathologic behaviors have long been classified as impulse control disorders, including pathologic gambling, intermittent explosive disorder, kleptomania, pyromania, and trichotillomania. Research suggests that several of these impulse control disorders more accurately represent behavioral addictions.

As a distinct area of study, behavioral addiction is recent, and extensive knowledge advances have been made since 2010. Growing evidence suggests that behavioral and substance addictions overlap in clinical expression (e.g., craving, tolerance, withdrawal symptoms), comorbidity, neurobiologic profile, heritability, and treatment. Behavioral and substance addictions also share similar features in natural history, phenomenology, and adverse consequences. This course will address the most common non-substance behavioral addictions, including pathologic gambling, compulsive sexual behavior, compulsive buying, and compulsive video gaming.

BEHAVIORAL ADDICTION: EVOLUTION AND CONCEPTS

In some behavioral addictions, the published research is vast but confusing. Prominent investigators in this field view behavioral addictions through differing paradigms, which has interfered with expert consensus and efforts to advance the field. Disagreement on diagnostic criteria has prevented standardized measurement of treatment outcomes. Debate has also focused on whether behavioral addictions qualify as addictions or are better explained as disorders of impulse control or compulsivity, and whether "Internet addiction" as a unified construct is valid.

EVOLUTION IN THE CONCEPT OF BEHAVIORAL ADDICTION

Early Characterizations

The concept of addiction involving non-substance behaviors as repeated urges to engage in counterproductive activities was introduced in 1990. At the time, behavioral addictions were proposed to encompass obsessive-compulsive disorder (OCD), "compulsive spending" (including gambling), "overeating" (binge eating), "hypersexuality," and kleptomania. It was thought these behaviors were linked to addiction by poor impulse control and self-regulation, which led to repetitive engagement of the behavior despite negative consequences [1; 2]. The concept of an obsessive-compulsive spectrum of disorders was proposed in 1993; disorders that featured an inability to control or delay repetitive behaviors were thought to fall on a spectrum from impulsivity to compulsivity [3]. Numerous psychiatric and neurologic disorders were included in this spectrum [4].

Further research suggested that impulsivity and compulsivity appeared in substance and behavioral addictions at different stages, and the "impulsive-compulsive disorder" model was proposed. In this model, impulsivity was dominant in earlier-stage addiction, when behavior is motivated and reinforced by reward, and compulsivity was dominant in later stages, when behavior is motivated and reinforced by avoidance of negative emotional states [5].

Expansion of Core Features

An influential 2006 study broadened the definition and core features of addiction as 1) a state of craving or urge that immediately precedes the behavior, 2) impaired ability to control the behavior, and 3) the behavior continues despite negative consequences [6]. Criterion 3 suggested addiction was no longer tethered to substance use. This was cemented in 2011, when the American Society of Addiction Medicine released their definition of addiction. The core features of this definition are [7]:

- The inability to consistently abstain
- Impairment in behavioral control
- Craving
- Diminished recognition of significant problems with one's behaviors and interpersonal relationships
- A dysfunctional emotional response

Numerous repetitive problematic behaviors became suggested as behavioral addictions, but criticism that excessive normal behaviors were becoming overpathologized ensued [4; 8; 9]. Concerns were raised that such persons could become stigmatized, based in part on study results suggesting that "brain disease" explanations of drug addiction had unforeseen consequences on clinician and public attitudes—the inability of persons with addiction to control behavior being hard-wired into their brains was perceived to make them more dangerous [10].

The DSM-5 and Classification Issues

A major step forward in the recognition of behavioral addictions as addictive disorders came in 2013 with the American Psychiatric Association (APA) publication of the fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5) [11]. In the DSM-5, pathologic gambling was renamed gambling disorder, becoming the first recognized behavioral addiction. This followed decades of research and data demonstrating similarities to substance addictions [8]. Internet gaming disorder and its proposed diagnostic criteria were placed in Section III (Emerging Measures and Models); the body of evidence was extensive, but standardized diagnostic criteria remain absent [12].

Decisions in the DSM-5 concerning behavioral addiction were based on evidence published before 2013 in an emerging and quickly evolving field. While some criticism reflected the premature state of some evidence and lack of consensus, other criticism was directed at decisions to omit behavioral addictions that were viewed as subjective and biased [4].

INTERNET-MEDIATED BEHAVIORAL ADDICTION

Internet addiction is the concept of addiction to Internet use itself, as opposed to the concept that some addictions are mediated by Internet use. The description and diagnostic criteria of Internet addiction disorder first appeared in 1996. Internet addiction disorder was considered an impulse control disorder, based on an inability to control Internet use and resulting in impaired functioning. An Internet addiction disorder typology was proposed, consisting of [13]:

- Cybersex addiction
- Cyber-relationship addiction
- Internet compulsions
- Information overload
- Computer addiction

This work in a newly evolving, unexplored area could not foresee the current reality of Internet centrality in daily life. Many aspects of Internet use deemed pathologic in 1996 have become normative [14]. The Internet is now considered the medium of choice in many gambling, gaming, sex, and other addictions, but not itself the object of addiction, and the concept of Internet addiction has been abandoned [15].

The DSM-5 unnecessarily contributed to this confusion in the introductory text for Internet gaming disorder by stating that it was also commonly referred to as Internet use disorder or Internet addiction [11]. This message was unclear, but some features unique to the Internet may promote excessive or addictive behaviors, including [16; 17; 18; 19; 20; 21]:

- Accessibility through high-speed broadband connections
- Affordability, as the Internet lacks previous cost restraints
- Anonymity, which can encourage behaviors inhibited offline by stigma and fear of detection and increase perceived control over the content, tone, and nature of online experience. Anonymity can increase the comfort level of users with fears of social rejection by removing the ability to look for and detect disapproval or judgment in facial expressions or body language.
- Convenience of use in familiar, comfortable environments, reducing perceptions of risk
- Escapism. Online gaming, gambling, buying, or sex is reinforced by a subjectively experienced "high," with habitual pursuit a core feature of addiction. Relief from negative emotions and distress related to real-world problems further reinforces the behavior.
- Immersion can facilitate a dissociation-like state in which the user loses track of time, feels like someone else, blacks out, or experiences a trance-like state, a reinforcing effect for some.
- Disinhibition. The core reward for some. Users tend to lower their defenses and emotionally reveal themselves faster online than offline. Socially inhibited users can find the disinhibiting environment and perceived connection to others powerfully reinforcing.

Other contributors to online impulsivity include invisibility and the absence of authorities (e.g., police, parents, teachers) that communicate power and hierarchy offline and constrain behavior [21]. Freed of inhibitions that maintain offline behavior within the boundaries of civility, social acceptability, and legality, users may act on impulses online they would otherwise attempt to resist [22].

COGNITIVE AND BEHAVIORAL ASPECTS OF BEHAVIORAL ADDICTION

As with substance addiction, behavioral addictions are defined by the presence of cognitive, affective, and behavioral abnormalities that reflect maladaptive alteration of underlying neural structures—the core pathology. Functional magnetic resonance imaging (fMRI) and other brain imaging methods can identify these core pathologic features and their similarity to those of substance addiction. Addiction research is moving toward classifying psychopathology on dimensions of behavior and neurobiology and away from DSM-type symptom checklists. This is expected to improve assessment and diagnostic validity and treatment efficacy [23].

Impulsivity

The ability to delay, withhold, or interrupt a behavioral response is a key aspect of executive function. Suppressing an inappropriate immediate response allows time for other processes (and predicting consequences of possible actions) to promote appropriate behavioral choices. Effective inhibitory control is required for adaptive functioning, such as restraining impulses for spiteful, peculiar, and/or inappropriate expression [11; 24].

Impulsivity is complex. It is broadly defined as a predisposition for rapid, unplanned reactions to internal or external stimuli without forethought or regard for negative consequences to self or others [25]. In persons with early-stage substance or behavioral addiction, impulsivity is a powerful mechanism thought to drive pursuit of immediate reward, and it is a temperament vulnerability factor for substance use disorders [26; 27; 28].

Those with prominent impulsivity traits tend to favor immediate rewards over long-term gains, to have poor capacity to delay gratification or resist urges, to quickly react with poorly planned response to stimuli, to be unreflective in decision-making, to dismiss the negative consequences of one's actions, and to be disinhibited, novelty- and thrill-seeking, and impatient [29]. Impulsivity includes several distinct neurocognitive mechanisms and domains.

Domains of Impulsivity

The four major domains of impulsivity are motor, choice, reflection, and delay discounting. Motor impulsivity (also known as response/rapid-response impulsivity) is defined as an impaired ability to stop motor response following changes in environmental circumstances. Impaired motor response inhibition is highly prominent in attention deficit hyperactivity disorder (ADHD) (the archetypal disorder of impulsivity), pronounced in trichotillomania, and observed in gambling disorder [30; 31].

Choice impulsivity is characterized by difficulty in delaying gratification and a tendency to choose immediate small rewards at the expense of larger but delayed rewards. This type of impulsivity is consistently observed in substance use disorder and when money, health, and/or freedom are rewards [32; 33; 34].

Reflection impulsivity is the diminished ability to collect salient information from the environment before a decision. Reflectively impulsive persons make choices rapidly, rather than waiting for more relevant information to become available. Reflection impulsivity commonly occurs in substance use disorder and gambling disorder [35].

The subjective value of a reward decreases as a function of delay in its delivery; the value of reward is discounted with longer delay. With Internet gaming disorder, the immediate reward of online gaming increasingly comes at the cost of abandoned social and professional activities that yield greater long-term rewards [31; 36]. This is referred to as delay discounting.

Measuring Impulsivity

Impulsivity-related behaviors are measured by neuropsychologic testing, personality scales, and neuroimaging. These assessments can be repeated over time to evaluate treatment response [37].

Neuropsychologic tests measure performance (i.e., functioning) in specific cognitive domains (***Table 1***). Testing during neuroimaging can identify the functional integrity of brain regions that mediate performance on specific neuropsychologic tasks.

Personality scales measure the extent that statements involving impulsive behaviors are endorsed. These statements include: "I make decisions quickly," "I often buy things I cannot afford," or "I act without thinking" [25].

Neuroimaging uses non-invasive technologies to identify brain regions involved in inhibitory control, presence of abnormality, and possible changes with treatment [25]. Neuroimaging has helped identify frontostriatal circuitry as contributing to action inhibition, affective inhibition, cognitive flexibility, and delay discounting. It is now known that frontostriatal circuitry and dopaminergic transmission are common neural substrates that underlie inhibitory control function [38; 39].

COMMON IMPULSIVITY DOMAINS AND NEUROPSYCHOLOGIC TESTS		
Impulsivity Domain	**Neuropsychologic Test**	**Cognitive Function Measured**
Motor inhibitory control	Stop-signal task	Ability to withhold or cancel a planned motor response
Affective inhibitory control	Emotion regulation task	Ability to suppress a default emotional response to unpleasant stimuli
Cognitive flexibility	Reversal learning task	Perseverative response to previously rewarding stimuli when stimulus association rules have changed
Delay discounting	Delay discounting task	Degree of preference for smaller immediate rewards over larger delayed rewards
Source: [25]		*Table 1*

Compulsivity

Compulsivity is less well-defined and investigated than impulsivity, but it is used to describe repetitive and functionally impairing overt or covert behaviors that lack adaptive function. These habitual, stereotyped behaviors are performed inflexibly to avoid perceived negative consequences and persist despite being harmful [40; 41].

Compulsive behaviors are inappropriate and have no obvious relationship to a goal. When reward-driven substance use or behavioral pursuits persist over an extended period, reward-based learning mechanisms are thought to develop into compulsive behaviors [26].

Compulsivity appears distinct from impulsivity, but research demonstrates overlapping neural networks involved in both [4]. Deficits in cognitive flexibility may also serve as markers in disorders of addiction to drug or non-drug rewards. There are two subtypes of compulsivity (reversal learning and attentional set shifting) [42]. Reversal learning is the capacity to flexibly switch choices in response to immediate changes in the environment or setting. The orbitofrontal cortex are involved in this type of compulsivity. Attentional set shifting is the ability to shift response sets to a previously irrelevant dimension and involves the lateral prefrontal cortex (PFC).

EXPLANATORY MODELS OF ADDICTION

Explanatory models of psychiatric disorders, including behavioral and substance addictions, are developed to provide a conceptual framework for understanding the disorder and identifying an effective treatment approach. Theoretic models can propose an explanatory basis for entire categories of disorders or specific disorders, a pathogenic process within a larger pathophysiology, or a specific symptom or feature. In behavioral addictions, diverse models have been developed to explain symptoms and clinical course and the interpersonal, cognitive, psychologic, and neurobiologic dysfunction experienced by patients. The most relevant models serve as an organizing foundation for treatment approaches.

GENERAL MODELS OF ADDICTION

Reward-seeking is a central component of addictions. Early reward-centric models focused on the nucleus accumbens, where dopamine is released in response to natural rewards like sex or food. Drugs of abuse were thought to "hijack" the nucleus accumbens by triggering dopamine release far exceeding that of natural rewards (e.g., food, exercise, sex). The reward deficit disorder model posits that addictive behaviors develop from the need to compensate for impaired reward signaling in the mesolimbic dopamine pathway [43].

Although simplistic based on current understanding, earlier biologic models provided an important foundation for later expansion. For example, nucleus accumbens and dopamine function are now known to be involved in reward-based learning, reward anticipation and valuation, salience attribution (i.e., assigning degrees of relevance to items, decisions, or behaviors), and loss processing [43]. More recent models include the reward deficit disorder model, Gray's reinforcement sensitivity theory, the attachment theory development model, and the rejection sensitivity theory.

The Reward Deficit Disorder Model

The reward deficit disorder model describes addictions as chronic relapsing disorders characterized by a compulsion to seek and take the drug or experience the behavior, loss of control over stimuli intake, and emergence of a negative emotional state (e.g., dysphoria, anxiety, irritability) when unable to access the stimulus. The negative emotional state is termed motivational withdrawal syndrome. A key component of addiction is negative reinforcement, which describes engagement in potentially harmful behaviors to alleviate a negative emotional state [44].

"Reward deficit disorder" refers to multiple motivational mechanisms and the progression from impulsivity (positive reinforcement) to compulsivity (negative reinforcement). Compulsivity is thought to result from stress within forebrain structures and dysregulation of neurocircuits involved in reward [44].

Gray's Reinforcement Sensitivity Theory

In Gray's reinforcement sensitivity theory, behavioral addictions are understood from the context of brain system sensitivity to punishment and reward. Accordingly, punishment sensitivity regulates responses to stimuli perceived as potentially dangerous, leading to their avoidance (behavioral inhibition). Reward sensitivity directs behavior toward appetitive stimuli that provide immediate compensation (behavioral activation) [45; 46].

The Attachment Theory Developmental Model

Behavioral addictions have also been examined through the lens of attachment theory. While not specifically a model of addiction, attachment theory helps explain how early environment experiences may shape beliefs and cognitions that increase risks of behavioral addiction. This model posits that the nature and quality of child attachment to a primary caregiver(s) shape the personality, beliefs in responsiveness and trustworthiness of others, and level of interpersonal security in adolescence and adulthood. The primary attachment relationship is thought to form the template from which subsequent relationships are developed [47; 48; 49].

A child with responsive, consistent early caregiving develops an expectation that others will be available and supportive when needed. In contrast, children raised in negligent, rejecting caregiving environments form negative expectations in future relationships and social interactions. Attachment style influences personality traits, interpersonal behaviors, and competencies that directly impact satisfaction in romance and friendships, emotional functioning, interpersonal communication, and social behavior [47; 48; 50].

Attachment style may also influence motivation for pathologic behavior. In persons with attachment anxiety, attachment needs are activated by relational or environmental stressors. Some persons with insecure early attachments may display attachment avoidance, which is the absence of desire for closeness in important relationships. In the context of Internet-mediated addictions, the high dependency in attachment anxiety leads individuals to seek comfort and connection online [49; 51; 52].

In one study of college students, attachment anxiety (both maternal and paternal) significantly predicted pathologic Internet use, but attachment avoidance did not. Maternal attachment anxiety was a significant predictor of male pathologic Internet use, while paternal attachment anxiety was most relevant in female pathologic Internet use. It is important to note that this study examined college students in the United States, where parental roles and attachment patterns may differ from those in earlier international studies [49].

Rejection Sensitivity

Another interpersonal model is rejection sensitivity, a cognitive-affective worry of rejection that impairs well-being and interpersonal function. Persons who doubt acceptance by others fear and expect rejection, are more hostile and aggressive in relationships, and experience more unstable and dissatisfying relationships that end sooner. They respond to social rejection with hostility, depression, emotional regression, jealousy, and/or over-politeness [35]. Susceptible individuals may turn to Internet-mediated activities first as a way to avoid social interactions; these behaviors may then evolve into addictions.

BEHAVIORAL ADDICTION MODELS

Most theoretical models address behavioral addictions broadly; a few are specific to Internet-mediated behavioral addictions. Many borrow from substance addiction models to address the motivational, behavioral, and cognitive factors associated with other behavioral addictions. However, a few models specific to behavioral addictions have emerged. Many psychologic therapies have been incorporated in behavioral addiction models, because patients with specific behavioral addictions show cognitive, behavioral, and personality dysfunctions successfully treated in other patient populations by the same therapy [53].

The Components Model of Addiction

The components model of addiction defines addiction as any behavior with all six core addiction components [17; 54]:

- Salience: The behavior dominates activity, focus, thinking (preoccupation, cognitive distortion), feelings (craving), and behavior (deterioration of socialized behavior).
- Mood modification: The subjective experience of euphoria or relief from aversive emotional states reinforces the behavior.
- Tolerance: Escalation in frequency or stimulus intensity is required to deliver the desired level of mood modification.
- Withdrawal symptoms: Unpleasant emotional effects occur when the behavior is discontinued, interrupted, or decreased, including feeling shaky, restless, moody, depressed, or irritable.
- Conflict: Interpersonal friction occurs when the behavior supersedes obligations and responsibilities to others. Conflict can be intrapsychic, from guilt or loss of control.
- Relapse: There are repeated failed attempts to quit, cut back, or control the behavior, with previous addictive behavior patterns quickly restored.

The Cognitive-Behavioral Model

The cognitive-behavioral model addresses the role of motivational drives for reward-seeking and stress-reduction; behavioral control (related to executive inhibition); and decision-making (involving weighing the pros and cons of engaging in motivated behaviors) in addictive behaviors.

Reward and Motivation

Motivational drives linked to reward-seeking importantly contribute to addictive behavior, and diminished executive function/cognitive control over these motivational drives contributes to decision-making that leads to persistent engagement in the behaviors. Enhanced reward sensitivity and decreased loss sensitivity may increase urges to gamble (in gambling disorder) or online game (in Internet gaming disorder) [53].

Executive Control and Behavioral Inhibition

Executive systems exert cognitive and behavioral control over motivational drives, allowing inhibition of desires and control or moderation of reward-seeking behaviors [55; 56]. Subjects with behavioral addiction show diminished response-inhibition and cognitive-control responses to addiction-specific stimuli, reflecting cognitive bias and altered set-shifting related to compulsive aspects of addictions. This is a reflection of alterations in executive function and neural processes that underlie attention, response inhibition, and behavioral flexibility [57; 58].

Decision-Making

In deciding between pursuit of immediate reward and delayed consequences, individuals with behavioral addictions show strong tendencies for deciding on immediate reward. Decision-making is the final "check point" before a behavior is enacted [53].

Interaction of Cognitive Domains

Increased sensitivity to winning or pleasure can enhance urges to gamble, online game, or engage in sexual activity. Impaired capacity for executive control leads to inadequate control over reward-seeking behaviors, permitting urges and craving to dominate decision-making and lead to addictive behavior patterns. This promotes disadvantageous decision-making for immediate gratification over long-term gains. Gambling or gaming reinforces reward-seeking behaviors, further eroding executive-control abilities and perpetuating addictive behaviors [53].

UNIFIED MODELS OF INTERNET-MEDIATED BEHAVIORAL ADDICTIONS

Two theoretical models have been developed to describe the underlying mechanisms in the development and maintenance of Internet-mediated addictive behaviors. Both models share common components, are theoretically sound, and have empirically confirmed elements.

The Theoretical Model of Internet Gaming Disorder

The theoretical model of Internet gaming disorder links decision-making favoring immediate reward despite long-term negative consequences and motivation-seeking (craving) as drives to experience pleasure and/or reduce stress. The third domain is diminished executive control (inhibition and monitoring) over motivation-seeking, consistent with research of executive functioning in individuals with substance use disorders. This model refers to reward-centered models of substance addiction, such as incentive salience theory. Treatment interventions are suggested to target specific cognitive and motivational factors [53].

The Interaction of Person-Affect-Cognition-Execution (I-PACE) Model

The I-PACE model is a theoretic framework for specific Internet-mediated behavioral addictions (e.g., gaming, gambling, pornography/cybersex, buying). According to this model, these addictions are the result of interactions between predisposing factors, moderators, and mediators with impaired executive functioning. Conditioned processes (learning and memory) strengthen these associations in the addiction process. Biopsychologic, personality, and affective factors combine and interact to influence an individual's predisposition for Internet-mediated addictions. Interactions between predisposing affective/cognitive factors and pathologic neuropsychologic functioning during Internet-mediated addictions are bidirectional [59].

Predisposing biopsychologic factors increase vulnerability to emotional disorders and behavioral addiction. Negative early childhood experiences of trauma, emotional or physical abuse, and/or social isolation are linked to insecure attachment styles, which correlate with Internet-mediated behavioral addictions [60]. Adverse early events increase individual vulnerability to intense stress reactivity, mental disorders, and addictive behaviors in adolescence and adulthood [61; 62].

Predisposing personality factors include high impulsivity, shyness, neuroticism, low self-esteem, conscientiousness, self-directedness, and procrastination. Affective/cognitive responses to environmental stimuli influence the preference of specific Internet genres. For example, a perceived lack of social support, feelings of isolation, and loneliness are linked to the use (and potential misuse) of Internet genres with prominent communication features (e.g., social networking sites) [63; 64]. Specific Internet-mediated addictions are associated with specific personality profiles, such as greater rates of ADHD and impulsivity.

The neural correlates of Internet-mediated addictions reflect a maladaptive interaction between cue-reactivity/craving and reduced prefrontal/executive functioning, also observed in substance addictions. Decisions to reduce craving and increase mood through specific Internet genre use are thought to be the result of increasing dominance of immediate reward and decreasing consideration of longer-term negative impacts [59].

CHILD AND ADOLESCENT VULNERABILITY TO BEHAVIORAL ADDICTION

For many years, experts have expressed concerns over child and adolescent susceptibility to pathologic behavior patterns involving the Internet, video games, and other digital media use. Substantial changes in brain function and structure occur during adolescence into young adulthood. Key developmental characteristics of adolescent brains increase the likelihood of thrill-seeking and risky behaviors associated with substance use and behavioral addictions. Adolescent behavior is marked by intense affective expression and impulsive reactivity. During the transition from childhood to young adulthood, the process of enhanced myelination decreases gray matter and increases white matter. The increase in white matter is mostly uniform, but diminished grey matter (through synaptic pruning) is more selective. Myelination increases the speed of neural signaling and underlies the timing and synchrony of neuronal firing patterns that convey meaning [65].

The PFC region is involved in executive processes important in risk-reward decision-making, but is among the last brain regions to mature. Compared with mature adults, this aspect of brain development renders adolescents more vulnerable to addictions, other risky behaviors, and mental health disorders [43]. The brain mechanisms that mediate cognitive control, impulsivity, and sensitivity to reward mature during adolescence and young adulthood, and this is the period when substance use initiation and other problem behaviors most commonly emerge [66].

Immature connections between the PFC, the nucleus accumbens, and the amygdala are thought to largely influence goal-directed behaviors in adolescents [67; 68]. Reduced prefrontal cognitive control may allow greater influence of affective systems over decision-making and behavior, which increases adolescent vulnerability to social and peer pressure [69]. Addiction can interrupt the normal neurodevelopmental trajectory from adolescence to early adulthood, and addiction in adolescence can be both a cause and effect of cognitive, affective, and behavioral dysfunction.

OVERVIEW OF GENERAL BEHAVIORAL ADDICTION TREATMENT

PSYCHOLOGIC THERAPIES

Psychologic therapies are the most extensively studied treatment approaches in behavioral addiction. The core components of many psychologic therapies are applied across behavioral addictions, while other therapies are modified for specific addictions. This section gives a broad overview of psychologic therapies used in the treatment of behavioral addictions in general. Tailored variants are discussed in later sections.

As with any patients, it is important to assess for native language (or preferred language). Patients with lower English proficiency will benefit from materials in their preferred language and/or the use of an interpreter.

Cognitive-Behavioral Interventions

Cognitive-behavioral therapies (CBTs) have evolved over the past 50 years. Behavioral therapy emerged as the "first wave" in the 1950s, followed by a second wave of cognitive therapy in the 1970s. Cognitive therapy and behavioral therapy were combined in the 1980s to become CBT. Since the mid-1990s, a range of third-wave approaches have been introduced, including acceptance and commitment therapy, cognitive-behavioral analysis system of psychotherapy, and functional analytic psychotherapy [70].

Cognitive-Behavioral Therapy

CBT is empirically supported with demonstrated efficacy across a range of substance use disorders [71; 72]. CBT may work by strengthening executive control over behaviors through acquisition of cognitive skills. In behavioral addictions, CBT can help individuals improve their inhibitory control ability, recognize maladaptive cognitions, and learn and employ skills for more adaptive decision-making [73; 74].

CBT and CBT-related therapies can help patients identify distortions and errors in thinking and the mistaken, irrational beliefs that underpin maladaptive functioning. Patients learn to identify these distortions by monitoring their internal dialogue (e.g., "You can't do that," "Don't even try") and remembering childhood pronouncements (e.g., "You'll never amount to anything," "You're lazy and stupid") that compromise current functioning. Assessing the validity of patients' conclusions and exploring different ways to view themselves and their capabilities are both potent CBT interventions. Through role-playing or in vivo exposure, patients can experiment with alternative views and behaviors. CBT-based approaches also teach coping skills, symptom management, relapse prevention, impulse control, and anger management [75].

CBT for Internet-mediated addiction (CBT-IA) addresses coping styles, Internet-related expectancies, attentional biases, cue-reactivity/craving, executive function, and inhibitory control. The patient's Internet behavior is monitored and analyzed for situational, emotional, and cognitive contexts, and the reinforcing effects of Internet use are considered to help understand the cognitive assumptions and distortions that underlie situational triggers. CBT-IA identifies coping responses and high-risk situations for addictive Internet use, as well as the expectancies, illusions, and reinforcing effects. Cognitive restructuring and reframing target Internet-related cognitive biases [59].

Attentional biases can be decreased by attentional retraining. When combined with the computerized Go/No-Go task (a binary pass/fail test), this approach can improve inhibitory control and executive functioning. Cue-exposure therapy is found to reduce conditioned associations and craving intensity [59].

CBT has evidential support as a treatment for several behavioral addictions. However, there is a nationwide shortage of clinicians with specialized training in CBT in the United States. Even clinicians who believe they are delivering CBT-based interventions often do not deliver the core components of empirically supported CBT [76].

Cognitive Enhancement Therapy

As discussed, patients with behavioral addictions often show cognitive dysfunction involving elevated impulsivity, impaired cognitive control, and cognitive inflexibility, and therapies that target these domains may be helpful. Cognitive enhancement therapy (CET) involves the repeated practice of cognitive tasks involving problem-solving, response inhibition, visual tracking, and discrimination skills. These skills are practiced several hours per week over several months [56]. CET has resulted in significant improvements in impulsivity and delay discounting in patients with stimulant addiction, and similar strategies should be considered in targeting cognitive dysfunction in persons with behavioral addictions [77].

Cognitive Bias Modification

Persons with gambling or Internet gaming disorder show attentional biases towards Internet cues, and therapies that target such biases may be effective as treatment [78]. Cognitive bias modification specifically targets automatic/implicit processes such as attention bias and approach bias.

Mindfulness-Based Stress Reduction

Stress intolerance and poor stress coping are strongly associated with behavioral addictions. Mindfulness-based stress reduction interventions target stress reduction, and efficacy is supported in substance addictions. This approach may be particularly helpful in patients with behaviors driven by negative-reinforcement motivations [79].

Dialectical Behavior Therapy

Dialectical behavior therapy combines elements of CBT with mindfulness and other approaches into a structured treatment approach that emphasizes skills training in mindfulness, distress tolerance, and emotion regulation. Dialectical behavior therapy provides an atmosphere of validation with psychoeducation and coping skills training—core competencies for interpersonal functioning that ordinarily would have been learned growing up in a healthy family environment. The four primary skill sets are mindfulness, emotion regulation, distress tolerance, and interpersonal effectiveness [75].

Mindfulness helps patients cultivate present-moment awareness, enabling them to observe and describe what is occurring without judgment. Mindfulness effectively counteracts viewing the present through the lens of distorted hopes, expectations, and past experiences of rejection and abandonment. Many patients believe they miss their childhood/family/lover, but they actually miss their idealized version of the past, not the reality.

Effective emotion regulation helps patients to not over-react when facing emotions that threaten to overwhelm. Patients learn to become more aware of their feelings, the precursors to losing emotional control, and how to downregulate arousal states to relieve the pressure for impulsive behavior. Up-regulation is also taught, so patients do not ignore faintly recognized but painful reactions alerting them something is not right.

Dialectical behavior therapy also helps patients learn multiple methods of tolerating distress without resorting to self-defeating or self-destructive behavior. One method involves self-soothing by engaging the five senses when experiencing acute interpersonal anxiety or distress. This can involve listening to or singing a favorite song; watching a travel video of a beautiful location; savoring the taste of a favorite food; lighting a candle with a favorite scent; and/or touching or rubbing soft material or a smooth stone.

Interpersonal effectiveness is built by establishing a repertoire of skills that improve unstable relationship patterns. Patients learn skills related to self-respect, assertiveness, inhibition of emotions, and setting and maintaining personal limits and boundaries. Interpersonal effectiveness is the result of psychoeducation about commonly held relationship myths that encourages patients to begin dismantling maladaptive beliefs by developing statements that challenge their narratives.

Clinical psychologists administering dialectical behavior therapy are available after hours for support calls; during these contacts, patients are asked a series of standardized questions that encourage the best use of problem-solving strategies that apply dialectical behavior therapy principles. Typically, therapist-led dialectical behavior therapy consists of 2 introductory psychoeducational sessions; 16 core skill-learning and skill-building sessions on mindfulness, emotion regulation, and distress tolerance; and 2 final review and relapse prevention sessions [80].

Motivational Interviewing

The concept of motivational interviewing evolved from experience in the treatment of problem drinkers and was first described in 1983. Motivational interviewing is a systematic, evidence-based approach to complex behavior change. It is unique in eliciting and exploring patients' own arguments for change, evoking intrinsic motivation, and helping patients to resolve their ambivalence about change [81]. It is a directive but nonconfrontational method to bypass unproductive struggles. The therapist engages the patient through empathic listening, open-ended questions, reflective statements, affirmations, and summary statements. By decreasing denial and enhancing motivation, motivational interviewing is a pragmatic, practical approach that focuses on actual change [75].

Motivational interviewing helps empower patients with responsibility for their behavior. By allowing patients to resolve their ambivalence using an approach that gently pushes them, they are more inclined to acknowledge the consequences of their behavior and engage in treatment. More aggressive strategies, sometimes guided by a desire to "confront denial," can fail because they push patients to make changes for which they are not willing or ready [82].

Interpersonal Psychotherapies

Interpersonal Psychotherapy

Interpersonal psychotherapy (IPT) was originally developed as a treatment for patients with depression, but it has been modified for other clinical populations. It is a brief, focused therapy that targets problem resolution and symptom improvement within four social domains: grief, interpersonal role disputes, role transitions, and interpersonal deficits. Treatment occurs in three phases [80]:

- Developing a thorough understanding of interpersonal contexts that contributed to the primary diagnosis and identifying interpersonal problem areas
- Helping the patient make interpersonal changes in the identified problem areas
- Reviewing progress and consolidating gains in treatment to prevent relapse

Group Psychodynamic Interpersonal Psychotherapy

Group psychodynamic interpersonal psychotherapy (PIPT) differs from IPT in its de-emphasis of social roles (e.g., role disputes), with greater emphasis placed on present interactions among group members and the therapist. Group PIPT uses cyclical relational patterns and circumplex models (versus social roles) to understand interpersonal patterns. It also applies specific models to elucidate patient attachment needs, negative affect, and behavioral addiction as a coping mechanism [80].

Twelve-Step Programs

Introduced in 1935, Alcoholics Anonymous (AA) is the original and best-known 12-step program. It was followed by Narcotics Anonymous (NA), introduced in the 1950s. Twelve-step programs constitute a specific form of group-mediated recovery from substance and behavioral addictions. They are member-led groups of people with similar addictions who are committed to helping each other—new members in particular—achieve and maintain sobriety. The 12 steps refer to a specific program of recovery. In a very brief description, members of a 12-step program come to understand their powerlessness in controlling the addiction; develop a relationship(s) with a power outside oneself (i.e., a sponsor [mentor], the 12-step group, a therapist, a Higher Power of their definition, or any combination) to best protect against relapse and ensure continued recovery; identify and work through character defects; amend past harms; and become useful to others struggling with addiction. Since the introduction of AA and NA, several 12-step programs have been adapted for specific behavioral addictions [75].

The exact mechanisms of 12-step group involvement that underlie their efficacy are multiple and difficult to disentangle. For example, admitting being powerless over the addiction could permit greater control over the addictive behavior through changes in decision-making processes. Social interactions likely influence efficacy through contact with similarly motivated peers with a shared goal and social norm of abstinence, and access to sponsors for guidance in developing skills for managing crises. Specific aspects may operate through different mechanisms; influences on decision-making and risk-taking behaviors may occur through 12-step meetings and discussions with sponsors [83].

Combined Approaches

Preliminary studies suggest that combining several therapeutic strategies may be more effective than a single approach. Such approaches may include CBT, cognitive bias modification, CET, and/or mindfulness-based stress reduction, delivered in group, individual, family therapy, or school-based formats, with different strategies complementing aspects of the others. Combining behavioral and pharmacologic therapies can also be considered.

PHARMACOTHERAPIES

Almost all behavioral addictions were originally understood through the obsessive-compulsive spectrum model. By extension, and based on efficacy in OCD, selective serotonin reuptake inhibitors (SSRIs) became the most widely used drug class in the treatment of behavioral addictions, but they showed minimal benefit. More recently, neuroscience research identified similarities in the core features of behavioral and substance addictions, and the obsessive-compulsive spectrum model was discarded in favor of better-fitting addiction disorder models. Naltrexone, an opioid receptor antagonist with efficacy in substance use disorders, has suggested benefit in behavioral addictions, but there is evidence of possible adverse effect [22; 84].

The brain opioid system may contribute to social attachment and bonding, and brain opioid theory of social attachment posits that endogenous opioids may mediate reward effects in social connection and closeness. The possible effect of exogenous (drug) opioids on social reward is a new area of study. One trial randomized healthy volunteers to naltrexone or placebo and then to the alternate treatment 10 days under double-blind conditions. Participants noted significantly less sense of social connection on naltrexone, which was not explained by changes in emotional state. The findings support endogenous opioid contribution to social bonding and raise unexplored questions of whether diminished social bonding contributes to patient drop-out in naltrexone studies [85; 86].

GAMBLING DISORDER

Gambling is a popular activity in many cultures, and most people have gambled at some point or do so infrequently, without harm. A subgroup of individuals unable to control their gambling can develop gambling disorder, with potentially severe financial, emotional, relationship, occupational, and possible legal consequences. The accessibility, affordability, and anonymity of Internet gambling may facilitate disordered behavior in predisposed gamblers [87]. A key determinant in the development of gambling disorder is the structural characteristics of the game [88].

EPIDEMIOLOGY

In North America, gambling disorder affects 0.42% of the general population in their lifetime (0.64% among men, 0.23% among women) and 0.16% in the past year [89]. In epidemiologic studies, women represent about 32% of people with gambling disorder and show a telescoping phenomenon of later onset and more rapid progression also observed in women with alcohol use disorder [84].

Financial and marital problems are common among people with gambling disorder. Many of these patients also engage in illegal behaviors, including stealing, embezzlement, and fraud (e.g., writing bad checks), to fund their gambling or to cover past losses. Usually resulting from financial and legal problems, suicide attempts are common and have been reported in 17% of people in treatment for gambling disorder [84].

Among treatment-seeking disordered gamblers, women initiate gambling at an older age than men (31.3 years versus 22.4 years) and have a significantly shorter time from gambling initiation to meeting DSM criteria for gambling disorder (8.33 years versus 11.97 years). As noted, this phenomenon is consistently found in women with substance use disorders as well [90].

Individuals with subclinical but problematic gambling are referred to as "problem gamblers." Studies conducted from 2000 to 2015 found past-year adult problem gambling rates of 2% to 5% in North America [91]. Problem gamblers account for almost one-third of total industry revenue [92]. Those with problem gambling are heterogeneous, and a problem-gambling typology was developed to help account for these differences and consists of three subcategories [10]:

- Behaviorally conditioned problem gamblers
- Emotionally vulnerable problem gamblers
- Antisocial, impulsive problem gamblers

COMORBIDITY

Gambling disorder is highly comorbid with other mental health disorders, particularly substance use disorders, and shows a heritability rate of 50% to 60% [93]. Data published before widespread access to online gambling indicate that, compared with non-pathologic gamblers, pathologic gamblers show a six-fold greater risk for substance use, a four-fold greater risk for illicit substance use disorder, and a three-fold greater risk for mood disorders [94]. At-risk gamblers have higher rates of psychiatric comorbidity relative to the general population [95].

RISK FACTORS

Many attributes of online gambling have led to assumptions that Internet gambling may facilitate earlier onset, more rapid progression, and greater consequences related to gambling disorder compared with off-line gambling [95]. Land-based gamblers, characterized as older, female, and more likely to be divorced, show greater use of electronic gambling machines and table games and greater problem gambling rates than online gamblers [95]. Mixed (off-line and online) gamblers, characterized by younger age, less likely to be married, and more likely to consume alcohol and illicit drugs when gambling, show a propensity to greater involvement in gambling, sustaining higher losses and consequent harms [95]. Online gamblers, consistently identified as male, younger age, higher in education and income, and employed full-time, are significantly less likely to report gambling-related problems than other gambler subgroups [95]. Unlike other behavioral addictions, online gambling is not associated with greater risk of problem or disordered behaviors, which seems more likely to develop in gamblers who prefer offline gambling in casinos or other venues [96].

In contrast to expectations of greater gambling severity and psychopathology in lower income (vs. middle/higher income) groups, a robust positive relationship is found between alcohol use disorder and gambling disorder in middle/higher income (vs. lower income) groups [97]. Gamblers preferring rapid continuous forms in land-based venues can be motivated by emotional vulnerabilities to engage in low-skill games and may be more likely to bypass responsible gambling options to persist despite losses. Setting predetermined limits is more easily achieved with Internet gambling because registration before play is required [95].

In Australia in 2010, gambling generated $19 billion in revenue; 55% came from electronic gaming machines in clubs and hotels, and 40% of electronic gaming machine revenue came from problem gamblers (only 0.6% of the population) [10]. Termed the "crack cocaine of gambling," electronic gaming machines are considered the most hazardous form of gambling, because they incorporate features that promote gambling-related cognitive distortions to perpetuate gambling, including near-win (or near-miss) outcomes and "loss disguised as a win." These features activate the sympathetic nervous system and reward-related neural circuitry, amplified further in disordered gamblers [98; 99].

"Losses disguised as wins" undermine the decision-making capacity of disordered gamblers already experiencing cognitive distortions, so they are less likely to make rational choices. Gamblers often misinterpret near-wins as evidence they are mastering the game, which fosters an illusion of control [10].

ASSOCIATED FEATURES

Smoking status is associated with more severe gambling and psychiatric symptoms. Among patients seeking treatment for gambling disorder, daily smokers (compared with non-daily smokers) were more likely to have histories of substance use disorder treatment, more severe gambling, family and social problems, and more frequent psychiatric symptoms, especially anxiety-related symptoms [100; 101]. In a study of the association between smoking and gambling in adolescents, those with "at-risk/problem gambling" who smoked were more likely to report poor grades, lifetime use of cannabis and other drugs, current heavy alcohol use, depression, aggressive behaviors, and less likely participation in extracurricular activities [102].

Gambling disorder is associated with elevated rates of suicidal thoughts and behavior. Among non-treatment seeking subjects with subsyndromal gambling disorder, suicidality was identified in 18.4% [103]. Suicidality is significantly associated with mood and anxiety disorders, higher nicotine consumption, and greater impairments in decision-making and cognitive flexibility [103].

PATHOPHYSIOLOGY

Disordered gambling is associated with several cognitive impairments, including diminished reward sensitivity from reduced reward circuit activity, reduced ability to delay reward or learn from negative consequences, stronger physiologic responses to near-wins, and poor error monitoring. The neuropsychologic impact of near-wins is a key feature in developing and maintaining gambling addiction [10].

Subjects with gambling disorder have been compared to healthy controls in numerous neuropsychologic and neuroimaging studies. Brain structure abnormalities, and corresponding functional deficits, have been identified in individuals with gambling disorder in the insula (altered near-miss cognitive interpretation), ventral striatum (impaired reward-based cognitive flexibility), frontotemporolimbic circuits (impaired concentration, memory, executive functions), PFC (cognitive rigidity, impulsivity, compulsivity), dorsolateral PFC (risky decision-making), ventrolateral PFC (impaired reward-based cognitive flexibility), and orbitofrontal cortex (risky decisions, impairments in assessing future consequences, and reward-based cognitive flexibility) [104; 105]. Neurochemical systems implicated in gambling disorder pathophysiology include dopaminergic, glutamatergic, serotonergic, noradrenergic, and opioidergic systems [105].

In one study of gambling disorder and control subjects, reactivity to gambling cues was imaged by fMRI. In gambling disorder subjects, gambling cues reliably induced craving to gamble and increased neural reactivity in the anterior cingulate cortex and insula [106]. Craving level was strongly linked to:

- Hyper-connectivity between the nucleus accumbens and insula
- Hypo-connectivity between the nucleus accumbens and medial PFC

Gambling cues activated brain responses in reward-related circuits. The intensity of cue reactivity (as craving) was influenced by impaired PFC control over the limbic system. As gambling abstinence increased, craving elicited by the gambling cues decreased.

Gray's theory provides a framework to understand behavioral addictions in terms of the sensitivity of brain systems to punishment and reward [45].

Patients with gambling disorder with higher reward sensitivity have been found more likely to dropout from treatment than those with lower reward sensitivity. In a community sample of gamblers, high reward sensitivity was associated with greater gambling frequency and social motives for gambling. Disordered gamblers in therapy are commonly urged to sever their ties with people and places that may trigger a relapse; when confronted by this, patients with stronger social bonds to gambling may terminate treatment. This is even more likely in early-stage gambling disorder, in which the patient attains the sought-after immediate reward and has not yet experienced distress or consequences [45].

DIAGNOSIS

According to the DSM-5, gambling disorder is categorized as a substance-related or addictive disorder. Diagnosis of gambling disorder requires four or more of the following [11]:

- Often preoccupied with gambling
- Need to gamble with increasing amounts of money to achieve excitement
- Repeated unsuccessful efforts to reduce or stop gambling
- Restlessness or irritability when trying to reduce or stop gambling
- Gambles in response to negative moods
- Chases losses
- Lies to others to conceal the extent of gambling
- Jeopardizes relationships, career, or educational opportunities because of gambling
- Relies on others to escape negative financial consequences of gambling (i.e., "bailouts")

The exclusion of illegal activity from the DSM-5 criteria was not an endorsement to downplay its relevance. The presence of illegal behavior in patients with gambling disorder is directly linked to more severe pathologic outcomes and resistance to treatment; specific and more intensive treatment may be required for these patients [107].

In differential diagnosis, it is important to rule out medication side effects when new-onset disordered gambling symptoms follow initiation of dopaminergic medication in Parkinson disease [11].

Gambling disorder is included in the *International Classification of Disease, Eleventh Revision* (ICD-11), published in 2018 [108].

TREATMENT

Despite serious psychosocial and financial consequences, most disordered gamblers do not seek treatment or may seek treatment only after being pressured by family members. Patient motivation and treatment adherence importantly contribute to successful treatment outcome for patients with gambling disorder [109].

Prominent barriers to seeking help for gambling problems include wanting to handle the problem by oneself, shame/embarrassment/stigma, unwillingness to admit the problem, and issues with treatment itself. Unwillingness to admit to the problem may be more prevalent than reported. Other barriers include a lack of knowledge about treatment options and practical issues involving treatment attendance [110].

A main challenge in gambling disorder treatment is the high patient dropout, ranging from 43% to 80% depending on treatment modality and setting [109]. Identifying factors that improve treatment engagement is important. One study found patients receiving antidepressants plus group CBT had the longest treatment retention, regardless of gambling disorder severity or demographic factors. Given the limited benefit of antidepressants in gambling disorder, this finding was unexpected but suggests using multiple treatment modalities may reduce patient dropout [109].

Psychologic Interventions

Many disordered gamblers can trace the course of their disease to an early win, followed by increasing gambling frequency and expenditure. Even early wins of $100 to $500 can increase excitement about gambling, distort self-perceptions of skill or expected returns, and strengthen irrational beliefs about gambling (e.g., illusion of control) [111; 112]. Many erroneous beliefs and inaccurate perceptions are rewarded, learned, or become habitual during disordered gambling, and several cognitive distortions are emblematic of gambling disorder, including [70; 113]:

- The gambler's fallacy (i.e., belief that a string of losses must predict an imminent win)
- The availability heuristic (i.e., selective recall of wins over losses)
- Failure to recognize net losses with intermittent small wins (e.g., spending $400, a $50 win is deemed a success, despite the $350 net loss)
- The idea of intensely needing to win makes winning likely
- Beliefs about luck

Therapies targeting erroneous cognitions, gambling urges, and motivations are effective. A meta-analysis of behavioral therapy outcomes in gambling disorder found an effect size of 2.01 at the end of treatment and 1.59 at follow-up (mean: 17 months). Efficacy seems to diminish over time, but the effect sizes were robust [83; 114].

Cognitive-Behavioral Therapies

In the treatment of gambling disorder, cognitive therapy is based on the idea that gambling-related cognitive distortions contribute to disordered behavior. With this approach, the therapist helps patients identify and correct gambling-related cognitive distortions, with the goal of improving gambling abstinence by greater use of logical behavioral choices. One cognitive approach provides up to 20 weekly sessions until the patient stops gambling. Short-term outcomes show superior improvement relative to control groups. A frequent component is relapse prevention, which identifies high-risk situations for relapse and maladaptive beliefs related to control over gambling [112; 115].

Behavioral therapies target gambling urges primarily with exposure-based therapies. Exposure therapy is based on classical conditioning paradigms, whereby gambling urges are eliminated through cue exposure with extinction processes. Exposure therapy is thought by some to be more beneficial in gambling disorder than other behavioral therapies, such as aversive therapy [70].

CBT focuses on changing gambling-related cognitions and behaviors; it is effective and improves treatment engagement and outcomes [113]. Brief, four- to six-session CBT interventions show some promise in problem and disordered gambling, but they require further evaluation in broader samples of treatment-seeking gamblers [112; 116]. An eight-session CBT intervention for gambling disorder may follow the specific therapeutic focus outlined here [94; 112]:

- Recognizing gambling triggers that precipitate gambling episodes: days/times, people, cues (cash), events (celebrations, arguments), and emotions (negative affect)
- Examine gambling episodes using functional analysis
- Increase alternate pleasurable activities
- Manage anticipated and unanticipated triggers
- Relaxation techniques to manage responses to gambling urges
- Interpersonal conflicts, practice gambling refusal skills
- Cognitive biases and distortions with gambling disorder
- Relapse prevention, coping with major life events

Variants of CBT have been adapted for patients with gambling disorder.

EVIDENCE-BASED PRACTICE RECOMMENDATION

According to a Cochrane Review, cognitive-behavioral therapy (CBT) is effective in reducing gambling behavior and other symptoms of pathologic and problem gambling immediately following therapy. However, the durability of therapeutic gain is unknown.

(https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD008937.pub2/full. Last accessed April 12, 2021.)

Level of Evidence: Meta-Analysis

Functional analysis teaches patients with gambling disorder to identify triggers, related feelings, and resultant behaviors; examine the positive and negative consequences of their triggers; and identify cognitive biases, such as luck-related beliefs in their gambling behaviors [83]. The process of developing improved strategies to identify and manage triggers related to craving may increase PFC control over motivational drives involving subcortical brain regions [83]. Increased awareness and understanding of irrational cognitions, such as those related to luck, near-miss events, and chasing behaviors, may increase the balance in activity of brain circuits that code conflicting motivational states [83; 117].

Skills training in CBT can include interpersonal skills and conflict resolution methods through role-playing and skills training. These skills are intended to help the person recognize that gambling may provide short-lived reward and enjoyment but problematic long-term consequences of debt, interpersonal conflicts, and/or legal system involvement. This type of intervention further addresses finance management and debt settlement—important concerns in gambling disorder [118].

Brief Motivational Interventions

Brief motivational intervention shows promise in reducing gambling when delivered alone or with other therapies. Brief motivational interventions are often directed at patients with subclinical diagnoses, defined as negative gambling-related consequences without meeting the criteria for gambling disorder. Brief motivational intervention is useful for engaging patients with poor problem recognition in further treatment or for spurring self-directed change. Therapy sessions focus on personalized feedback, brief advice, review of options (e.g., treatment options, change goals), and building self-efficacy, delivered in an empathic manner. In one study, telephone brief motivational intervention combined with a self-help workbook was effective in improving gambling outcomes in problem gamblers [119]. Single-session stand-alone motivational sessions are effective compared with assessment-only control groups [116]. Direct comparisons of motivational therapy and CBT suggest both interventions are equally effective compared with control groups [120].

Gambling Disorder and Comorbid Problem Drinking

Changes in alcohol use patterns during gambling treatment were examined in a study of 163 patients with gambling disorder followed 36 weeks pre-, during, and post-treatment. Overall, alcohol use was decreased during and after treatment, but 31% showed risky drinking during and after treatment. Latent risky drinking was predicted by recent at-risk drinking, male sex, younger age, lower gambling severity, treatment condition, and gambling during treatment. This greater-drinking profile suggested ambivalence about gambling changes during treatment, and those less motivated to change their gambling may be less motivated to reduce their alcohol use [121]. One study found that problem gamblers with co-occurring lifetime alcohol dependence demonstrate addictive behavior across multiple domains and resistance to externally motivated treatment approaches [122].

Treatment for patients with gambling disorder and comorbid risky alcohol use may differ from non-risky drinkers. Disordered gamblers with low-risk alcohol use tend to have positive outcomes with group CBT, while high-risk drinkers tend to respond better to motivational interviewing. Screens for alcohol problems, such as the Alcohol Use Disorders Identification Test (AUDIT), may be used to help guide treatment selection [123].

Pharmacologic Interventions

The most studied medication class in gambling disorder is SSRIs, but results have been mixed at best [84]. Based on possible efficacy in impulsivity treatment, several mood stabilizers have been evaluated in gambling disorder treatment. In one study, lithium was superior to placebo in reducing gambling symptoms during 10 weeks of treatment in patients with gambling disorder and bipolar spectrum disorders. However, olanzapine was found no more effective than placebo [84].

Opioid Receptor Antagonists

Multiple studies have demonstrated the efficacy of opioid receptor antagonists in the treatment of gambling disorder. Opioid antagonists dampen gambling-related excitement and cravings by decreasing dopamine neurotransmission in the nucleus accumbens and interconnected reward and motivational neurocircuitry [84].

In the initial study, naltrexone (mean dose: 188 mg/day) demonstrated superiority to placebo in reducing gambling-related behaviors, thoughts, and urges, and was particularly effective in gamblers with more severe gambling urges [124]. A second naltrexone study confirmed the findings of the initial study over a longer (18-week) period [125]. The efficacy of as-needed placebo or naltrexone (50 mg) and psychosocial support on reduction of gambling severity, gambling-related thoughts and urges, and gambling frequency and expenditures was assessed among 101 problem gamblers over 20 weeks [126]. Overall, the as-needed naltrexone provided no substantial benefit over psychosocial support.

Nalmefene is a long-acting analog of naltrexone administered by injection. Nalmefene efficacy was demonstrated in 207 subjects treated for 16 weeks, with significant reductions in gambling urges, thoughts, and behavior observed in 59% with nalmefene, compared with 34% with placebo [127]. A second nalmefene trial failed to show statistically significant differences from placebo [128]. Subsequent research found low-dose nalmefene significantly more effective than higher doses. Over 16 weeks, nalmefene (25 mg and 50 mg per day) led to significantly greater reductions in pathologic gambling severity than placebo. With nalmefene (25 mg/day), 59.2% of patients were rated as "much improved" or "very much improved," compared with 34.0% with placebo. Nalmefene at a dosage of 25 mg/day was effective and showed few adverse events, but 50-mg and 100-mg doses had intolerable side effects [129].

Catechol-O-Methyl-Transferase Inhibitors

Catechol-O-methyl-transferase (COMT) is an enzyme that degrades dopamine to regulate cognitive functioning in the PFC. As discussed, PFC-mediated cognitive dysfunction is implicated in the pathophysiology of gambling disorder, and a small uncontrolled study evaluated the COMT inhibitor tolcapone in 24 patients with gambling disorder who also received fMRI before and after treatment. Compared with controls, the subjects with gambling disorder showed underactivation during executive planning tasks pretreatment. Tolcapone was associated with statistically significant reductions on measures of pathologic gambling, the extent of which significantly correlated with increased activity in brain areas previously underactive during executive planning challenge [130].

Dopaminergic Modulators

Dopamine binding is positively correlated with gambling severity and impulsivity, and dopamine release in disordered gamblers is related to excitement and worsened behavioral performance. The dopamine-1 (D1) receptor antagonist ecopipam was evaluated in patients with gambling disorder when taken as needed for nine weeks. Compared with placebo, ecopipam led to statistically significant reductions in Yale-Brown Obsessive Compulsive Scale Modified for Pathological Gambling (PG-YBOCS) total score (25.6 baseline vs. 14.0 at study end) and subscales of thought-urge and behavior. Ecopipam may reduce pathologic gambling behavior [131].

Glutamate-Modulating Agents

Another promising area of pharmacotherapy in gambling disorder is glutamate-modulating agents. *N*-acetylcysteine (NAC), one such agent, was studied in subjects with gambling disorder over eight weeks; responders during open-label were then randomized to NAC or placebo for six weeks. In the open-label phase, 59% experienced significant symptom reductions and were classified as responders. At the end of the double-blind phase, 83% of those assigned to NAC remained responders (vs. 28.6% assigned to placebo) [132].

Cigarette smoking is highly prevalent in gambling disorder, and nicotine dependence is associated with greater gambling severity. Imaginal desensitization plus NAC was evaluated in patients with gambling disorder and nicotine dependence. After six weeks, subjects randomized to NAC or placebo both showed significant benefit from imaginal desensitization, but at three-month follow-up, NAC led to significant additional benefit in gambling severity reduction compared with placebo [133].

Other glutamate agents, such as topiramate, have not shown benefit [84].

Gamblers Anonymous

Founded in the 1950s, Gamblers Anonymous (GA) is a mutual help fellowship based on the 12-step program of AA. With meetings in most North American communities, GA is established worldwide as a resource for people struggling with gambling problems. GA groups are peer-led and abstinence-focused and represent the most widely available option for recovery from gambling disorder [134]. Some GA meetings welcome family and friends to attend "open meetings," recognizing the impact of disordered gambling often extends far beyond the patient and that support from non-gambling family and friends can be integral to recovery. Local groups can be found online at http://www.gamblersanonymous.org/ga/content/about-us [112].

GA has a unique culture of recovery, distinct from AA and NA in specific areas. For example, GA recognizes that for many new members, addressing the crippling financial consequences is vitally important for their recovery. Senior members who confronted and resolved their own financial consequences guide and support the new members through these challenges [134].

GA members tend to have more severe gambling symptoms, are more motivated for treatment, and have greater involvement with professional gambling treatment. GA involvement is positively associated with abstinence and improvements in abstinence, and with increased readiness for change, adaptive coping skills, and higher leisure activity involvement [134].

GA can be used alone or combined with other interventions. Individuals who engage in GA and professional treatment have better gambling outcomes than those who receive professional care alone. GA is also a valuable resource if remitted gambling problems recur in the future [33].

The potentially severe consequences of gambling disorder elevate the need to identify factors that support gambling abstinence. In a study of GA members, the greatest predictors of gambling abstinence were participation and involvement in GA, support from family and friends, and connections with other GA members. Relapse was strongly predicted by high gambling urges and erroneous gambling cognitions [92]. Of note, GA meeting attendance may be helpful, but involvement with GA meetings and members carried the greatest protection from relapse. GA and CBT are the two most widely used approaches for gambling disorder. A review of published evidence concluded that combining GA and CBT may enhance therapy engagement and reduce relapse risk [118].

ADDICTIVE SEXUAL BEHAVIOR

Persons with sexual addictions can spend vast amounts of time pursuing sexual encounters to the detriment of their marital, occupational, financial, and physical well-being and function. When human immunodeficiency virus (HIV) or other sexually transmitted infection (STI) exposure is possible, the behavior can be life-threatening. The lack of consensus of how to conceptualize problematic sexual behavior has invited explanatory paradigms using obsessive-compulsive, impulse control, addiction, and biopsychosocial models [75; 135].

Reports in the medical literature of patients seeking professional help for extremely frequent or hypersexual urges and behaviors date to the early 1800s [136]. In addition to well-characterized manifestations of sexual addiction, more recently developing contexts include methamphetamine-driven hypersexuality in the gay community and the use of location-based online dating platforms. Arguably the most important development in the field of addictive sexual behavior is Internet-mediated sex addiction, more frequently involving pornographic videos and masturbation [15].

The terms sex addiction, sexual addiction, addictive sexual behavior, compulsive sexual behavior, hypersexual disorder, sexual compulsivity, and sexual impulsivity describe roughly the same phenomenon [137]. Other related terms include [11; 138; 139]:

- Paraphilias: Behaviors deemed socially unacceptable that involve non-human objects, suffering of self or partner, children, or a non-consenting person (e.g., fetishism, exhibitionism, pedophilia)
- Nonparaphilic: More typical sexual behaviors that include compulsive sexual acts with multiple partners, constant fixation on a partner that may be considered unobtainable, compulsive masturbation, compulsive use of pornography, and compulsive sex and sexual acts within a consensual relationship
- Geo-locating social media: Online dating applications (apps) that use geo-positioning satellite (GPS) technologies in smart phones to provide the proximity of users from each other

EPIDEMIOLOGY, RISK FACTORS, AND COMORBIDITY

Addictive sexual behavior may be relatively common, and while no epidemiologic data are available, researchers estimated a lifetime prevalence of 2% to 6% and rates of 2.0% to 3.7% among American college students [140]. Some data suggest addictive sexual behavior may be more common with Internet mediation. A Swedish study estimated the prevalence of "Internet sexual problems" was 17.8% in men and 6.7% in women, higher than studies from the pre-Internet era [22].

Addictive sexual behavior is more common in men, with onset usually in late adolescence. It can involve a wide range of sexual behaviors, often including a mixture of paraphilic and nonparaphilic behaviors. Sexual urges and behaviors are often distressing and uncontrollable, triggered by certain mood states, and result in feelings of shame [141; 142].

Comorbidity is common in addictive sexual behavior, including substance use disorder (64%), affective disorders (14% to 39%), anxiety disorders (50%), and childhood conduct disorder (44%). In the family histories of patients with addictive sexual behavior, 40% had substance use disorder in at least one parent and 36% had sexual addiction in at least one parent [143; 144].

Addictive sexual behavior is consistently linked to high-risk sexual behaviors, including multiple partners, unprotected sex, and sex under the influence of drugs or alcohol. The adverse consequences and risks of addictive sexual behavior include HIV/STI infection, unwanted pregnancies, infertility, loss of relationships, social isolation, loss of self-esteem, job loss, legal issues, financial problems, significant personal distress, and impairment in daily functioning [137].

Patients diagnosed with sex addiction differ in common personality characteristics from those diagnosed with gambling disorder. Sex addiction is associated with higher education level and lower novelty-seeking, harm avoidance, persistence, and self-transcendence. Being employed and lower cooperativeness scores also predicted the presence of sex addiction. However, patients with gambling disorder or sex addiction do not differ in comorbid psychopathology [145].

Gender Differences

In one of few Internet pornography addiction studies involving women, Internet pornography users rated pornographic images as more arousing and reported greater craving for sex than non-users. Craving, sexual arousal, sensitivity to sexual excitation, problematic sexual behavior, and severity of psychologic symptoms were predictors of Internet pornography addiction. In contrast, current relationship status, number of sexual contacts, satisfaction with sexual contacts, and use of interactive cybersex were not associated with addiction. The reinforcing nature of sexual arousal, learning mechanisms, and cue reactivity and craving in the development of Internet pornography addiction in women were found consistent with those reported in heterosexual men [146].

In a large community survey, 51% of women and 90% of men reported using Internet pornography. Internet pornography use was associated with sexual dissatisfaction from perceived addiction (perceived compulsivity, effort to access, distress from pornography use) and sexual function problems (sexual dysfunction, compulsion, avoidance). This pattern held for men and women [147].

Geo-Locating Social Media

Growing in popularity and market share, geo-locating social media gives users the ability to find each other for casual sexual encounters ("hook-ups"). Unlike standard online dating apps, location-matching apps typically do not require users to create a profile about themselves; instead, users upload one or more photos. The simplicity of appearance-based selection and proximity accounts for the increasing growth in popularity of location-based dating apps. One of the first such apps, Grindr, was introduced in 2008 specifically for gay and bisexual males, followed by other apps for gay men, lesbians, and heterosexuals (Tinder, Bumble, and others) [139; 148].

INTERNET PORNOGRAPHY ADDICTION

Excessive and addictive Internet pornography use by younger men has received little attention outside of behavioral and sexual addiction research, but data suggest this has become a rapidly growing problem with serious consequences that requires fuller discussion.

Epidemiology of Adverse Effects

High-speed broadband Internet access became widely available in the early 2000s. Until the early to mid-2000s, erectile dysfunction (ED) prevalence in sexually active men younger than 40 years of age was low, consistently 2% to 4%. In contrast, studies conducted since 2005 show sharply increased prevalence of ED and low sexual desire in men younger than 40 years of age [149]. For example, new ED diagnoses in active-duty servicemen doubled from 2004 to 2013 [150]. In 2014, 53.5% of men 16 to 21 years of age had symptoms of sexual dysfunction, including ED (26%), low sexual desire (24%), and problems with orgasm (11%) [151]. In 2016, a study of persons 16 to 21 years of age followed over two years found persistent problems in boys/men of low sexual satisfaction (47.9%), low desire (46.2%), and ED (45.3%). Over time, sexual problems declined in women but not men [152]. A 2015 study found ED with low desire for partnered sex was common in men (mean age: 36 years) seeking help for Internet pornography use with excessive masturbation [153].

ED in men younger than 40 years of age is typically diagnosed as psychogenic and linked to stress, depression, generalized anxiety, or performance anxiety. Organic ED, diagnosed in 15% to 20% of cases, is linked to neurologic, hormonal, or anatomic conditions or drug side effects. ED in older men is more often diagnosed as organic, associated with smoking, alcohol use, diabetes, obesity, hypertension, or cardiovascular disease [154]. None of the known correlative factors of psychogenic ED adequately account for the rapid increase in youthful sexual dysfunction. The historical correlates of organic ED in older men have not changed proportionately, or have decreased, in younger men over the past 20 years [149; 155].

PATHOPHYSIOLOGY

The bulk of recent neuroscience research in sexual addiction has studied addictive Internet pornography use and, unfortunately, almost exclusively involves male research participants. Nonetheless, this research has importantly clarified pathogenic and pathophysiologic processes, the marked similarities to substance addictions, and the pathogenesis and pathophysiology of Internet pornography addiction, hypersexuality, and Internet pornography use-induced sexual dysfunction. The importance of this research is underscored by findings of a long-term study that Internet pornography may have the highest addiction potential of any online activity (with Internet gaming a close second) [156].

Cue reactivity, or the level of sexual arousal to Internet pornography, is the greatest single predictor of Internet pornography addiction, severity of the addiction, and impairment from the addiction. Hours of use alone is unrelated to addiction. Cue reactivity is an established correlate of addictive behavior across addiction disorders [149; 157]. Internet pornography addiction severity is directly related to cue-induced activation of the dorsolateral PFC and thalamus [149; 157].

Male sexual response is actually complex. One core mechanism is the hypothalamic nuclei, which regulates sexual behavior and erections by integrating brain and peripheral input. Pro-erectile input comes from mesolimbic dopamine pathways—the ventral tegmental area and the nucleus accumbens. The ventral tegmental area and nucleus accumbens form the core of a set of integrated circuits, termed the "reward system," that detects reward stimuli and regulates response to sex and other rewards. The mesolimbic dopamine pathway receives excitatory input from other limbic structures and inhibitory input from the PFC [149; 158; 159]. The reward system promotes memory and repeat of critical pro-survival behaviors such as sex, eating, and social connections.

Internet Pornography as Supranormal Stimulus

High-speed, broadband Internet pornography is substantially more sexually arousing than other forms of pornography or fantasy and may become more immersive with three-dimensional technology. Novel sexual visuals trigger greater arousal, faster ejaculation, and more semen and erection activity than familiar visuals; arousal is further increased by ability to self-select material with ease. As an amplified mimic of stimuli with evolutionary salience that human brains have evolved to pursue, Internet pornography is a supernormal stimulus and unique activator of reward, impulsivity, and for some, addiction [149; 160; 161].

Pathologic Learning and Hyper-Reactivity

Reward system vulnerability to pathologic learning can make Internet pornography use self-reinforcing. The neural substrates of sexual learning and addiction overlap. Development of addictive Internet pornography use promotes neuroplastic change. The mesolimbic dopamine system receives glutamate inputs from cortical and limbic regions. Glutamate synapses associated with seeking and obtaining a specific reward become altered, enhancing mesolimbic dopamine system response to the specific reward. With the mesolimbic dopamine system sensitized to sexual reward, hyper-reactivity develops to Internet pornography cues [149; 161; 162].

Decreased Reward Sensitivity (Hypo-Reactivity)

Reward system hyper-reactivity to Internet pornography cues develops concurrently with hypo-reactivity and down-regulation of response to natural rewards (i.e., partnered sex) [163]. Persons with addictive Internet pornography use show strong preferences for, but rapid habituation to, novel sexual images. This often fuels the search for even more novel sexual images and escalation to hardcore and extreme pornography, indicating decreased reward sensitivity, increased habituation and tolerance, and greater stimulation needed for sexual arousal [149; 161; 162].

Addictive Internet pornography use is correlated with lower functional connectivity between the striatum and the PFC. Dysfunction in this circuitry is linked to behavioral choices that disregard potentially negative outcomes, including reduced executive control function during exposure to pornographic images [149; 164; 165; 166].

Sexual Conditioning and Dysfunction

Sexual arousal conditioned to Internet pornography cues promotes sexual dysfunction [157]. Users crave the high levels of novelty, intensity, and arousal of Internet pornography that are difficult to sustain in partnered sex. When high stimulation expectations and requirements are not met, partnered sexual stimulation is ineffective. In some users, a preference for novelty arises from the need to overcome declining libido and erectile function, leading to conditioned preferences for Internet pornography use [167; 168].

Pornography use is inversely related to satisfaction with partners, as measured by affection, physical appearance, sexual curiosity, and sexual performance [169]. The caudate is involved in approach-attachment behaviors and motivational states associated with romantic love [170]. Putamen activation is associated with sexual arousal and penile tumescence [171]. Users of more extensive Internet pornography show smaller right caudate and lower putamen activation when viewing sexually explicit still photos. This reflects down-regulation of natural neural response to sexual stimuli from intense, prolonged exposure to pornographic stimuli [172]. Men with greater use of degrading or extreme pornography report more frequent concerns of sexual performance, penis size, and ability to maintain an erection (compared with moderate or non-users) [149; 173].

Early sexual conditioning impacts sexual arousal templates. A critical developmental period of sexual behavior forms around initial experiences with sexual arousal and desire, masturbation, intercourse, and orgasm. Younger age of onset for regular Internet pornography use and greater preference for pornography over partnered sex is associated with persistence of this pattern. In 2014, nearly 50% of college-age men reported they were first exposed to Internet pornography before 13 years of age, compared with 14% in 2008 [174; 175]. Young adults with addictive Internet pornography use show greater impairments in sexual arousal and erectile function in intimate relationships, but not with pornographic material.

ASSESSMENT

In contrast to the body of research in other behavioral addictions, the published literature on sexual addiction is highly qualitative in nature. There are fewer controlled studies of treatment, but far greater material that examines the nuances of clinical assessment and care of these patients. In fact, a main criticism of research in other behavioral addictions is the lack of such qualitative work [176].

Areas to inquire about in order to identify hypersexuality subtypes during patient assessment include [136]:

- Paraphilic interests
- Behaviors that are not themselves sexual but may suggest a hypersexual subtype. For example:
  - Non-sexual forms of avoidance or procrastination (may suggest problematic sexual behavior as a maladaptive avoidance strategy)
  - Non-sexual symptoms of personality disorders, by embedding the problematic sexual behavior as a single feature of a more general problem
  - With distress attributed to sexuality, probe to determine if sexual behavior falls within peer norms and/or the individual's distress unrelated to sexuality
  - Partner characteristics, such as recent or long-standing inhibitors of full sexual functioning

Clients may lack the vocabulary to describe their sexual interests, and it can be helpful to canvas and list the pornographic websites they prefer. Other general assessment questions for patients with sexual behavior concerns include [136]:

- How much of your concern is about the amount of sex, what kind of sex, or the kinds of people you want sex with?
- Some people hire sex workers for simple, physical sex, but other people want other kinds of relationships with the sex workers. What is your ideal?
- Which porn websites do you prefer?
- Masturbating several hours per day is certainly higher than average. What would you do with that time if not masturbating?
- How do you deal with stress? What has worked for you? What has not?
- Why did you get married? How has it matched up with what you expected?
- What would your partner's sex life be like if you never met?
- How does religion influence your life aside from sex?
- How much of the problem is about risking harm to yourself (or others), and how much involves what it means about you as a person?
- Finding sex is what attracts our attention, but for many people the problem is more about finding relationships. How do these go together for you?

EVIDENCE-BASED PRACTICE RECOMMENDATION

According to the British Association for Sexual Health and HIV, sexual history-taking should take place in a confidential, private environment.

(https://www.bashhguidelines.org/media/1241/sh-guidelines-2019-ijsa.pdf. Last accessed April 12, 2021.)

Strength of Recommendation/Level of Evidence: 1D (Strong recommendation based on non-analytic evidence)

Sexual Dysfunction from Internet Pornography Use

A simple assessment for Internet pornography-related sexual dysfunction is asking the patient whether he can achieve a satisfactory erection and climax when masturbating without Internet pornography. If he cannot, but can easily achieve these goals with Internet pornography, then his sexual dysfunction may be associated with its use. Healthcare providers should also screen for relationship problems, low self-esteem, depression, anxiety, post-traumatic stress disorder, stress, and other mental health problems, but should avoid assuming a psychologic cause of otherwise unexplained sexual dysfunction in men younger than 40 years of age. The relationship between psychosocial factors and sexual dysfunction in younger men may be bidirectional and co-occurring or Internet pornography-induced [149].

Possible Medical Causes of Hypersexuality

The first step in the treatment begins with accurate diagnosis that rules out medical causes of hypersexuality. Neurologic disorders associated with hypersexual behavior include Alzheimer disease (4.3% to 9% of these patients experience sexual disinhibition as a result of disease effects on the frontal and temporal lobes), frontotemporal degeneration (associated with impaired regulation of socially acceptable behaviors), and Kleine-Levin syndrome (causes hypersomnia that may lead to abnormal behavior, such as hypersexuality). Some medications or illicit drugs can elevate sexual drive, such as cocaine, methamphetamine, and dopamine agonists used in Parkinson disease [138; 177; 178].

DIAGNOSIS

Variously worded diagnostic entities describing addictive sexual behavior have appeared in the iterations of the DSM and the ICD [179]. Diagnostic criteria for hypersexual disorder were created and proposed for the DSM-5. These criteria are [138; 180]:

- Over a period of at least six months, recurrent and intense sexual fantasies, urges, or behaviors (specify if masturbation, pornography, sexual behavior with consenting adults, cybersex, phone sex, or strip clubs) in the context of three or more of the following criteria:
  - Excessive time consumed by sexual fantasies and urges, and planning for and engaging in sexual behaviors, repetitively interferes with other important (non-sexual) goals, activities, and obligations
  - Repetitive engagement in such sexual activity as a means to regulate dysphoric mood states of anxiety, depression, boredom, or irritability
  - Repetitively engaging in such sexual activity in response to stressful life events
  - Repeated but unsuccessful efforts to control or significantly reduce these sexual fantasies, urges, or behaviors
  - Repetitive engagement in sexual behaviors while disregarding the risk for physical or emotional harm to self or others

- Presence of 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
- Sexual activities not due to the direct physiologic effects of a recreational drug or medication, a co-occurring general medical condition, or to manic episodes
- Age of at least 18 years

Not included in the diagnostic criteria, Kafka also proposed that seven sexual outlets (i.e., orgasms) per week represented a cutoff point for excessive sexual activity, based on his review of published research [75].

The APA rejected the recommendation of its own Sexual and Gender Identity Disorders Work Group to include hypersexual disorder in the DSM-5. The APA stated an objection to the implicit normative reference to the "right amount" of sexuality (e.g., the cutoff of seven sexual outlets per week suggested in the supporting review but not in the diagnostic criteria) [181]. The DSM-5 task force also stated there were insufficient data to support inclusion of hypersexual disorder [75].

An evaluation of various proposed diagnostic criteria for sexual addiction found common core domains [75]:

- Excessive sexual behavior, generally outside the context of sustained intimate relationships
- Intense and persistent urges to perform such sexual behavior, similar to drug craving in substance addiction
- Continuation of sexual behavior despite the potential to cause significant personal, occupational, or financial consequences and/or harm to physical health
- Difficulty stopping the behavior despite repeated attempts or significant negative consequences

The ICD-10-CM was introduced to the U.S. healthcare system in 2015. It is the most widely used mental disorders classification worldwide and permits a hypersexual disorder diagnosis for coding and reimbursement. In the United States and other countries, ICD diagnostic codes are mandated by international treaty, unlike the DSM-5, which lacks such mandate. As such, a diagnosis of hypersexual disorder can be used even though it is not included in the DSM-5 [179].

Diagnostic criteria for compulsive sexual behavior disorder also appear in the ICD-11, published in 2018. These diagnostic criteria are [108]:

- A persistent pattern of failure to control intense, repetitive sexual impulses or urges resulting in repetitive sexual behavior. Symptoms may include:
  - Repetitive sexual activities become a central focus to the point of neglecting health, personal care, or other interests, activities, and responsibilities
  - Numerous unsuccessful efforts to control or significantly reduce repetitive sexual behaviors
  - Continued engagement in sexual behaviors despite adverse consequences
  - Continued engagement in sexual behaviors even when the individual derives little or no satisfaction from it
- Pattern evident for at least six months
- Pattern leads to marked distress or significant impairment in personal, family, social, educational, occupational, or other important areas of functioning

Distress that is entirely related to moral judgments and disapproval about sexual impulses, urges, or behaviors does not fulfill these criteria. Behaviors better categorized as a paraphilic disorder (e.g., exhibitionistic disorder, voyeuristic disorder, pedophilic disorder) also do not qualify.

STANDARD TREATMENT OPTIONS FOR SEXUAL ADDICTION			
Therapies	**Treatment Target**	**Mechanisms**	**Disadvantages**
Cognitive-behavioral therapy	Behavioral control, management of negative affect, social skills, relapse prevention	Functional analysis of sequence and triggers of behavior, thought records, skills building	Does not address motivation, personal meaning, or underlying character structure
Psychodynamic therapy	Self-concept and interpersonal relationships, self-awareness, underlying personality organization	Exploration of personal meaning of symptoms in context of personal history	Does not provide concrete skills to change behavior
Group therapy	Shame, stigma, social isolation, denial, rationalization	Social support, group confrontation of denial, peer sharing of experiences	Does not provide individualized, in-depth treatment
Medication treatment of comorbidity	Comorbid anxiety, depression, OCD, impulsivity, psychosis, mania	Established treatments of comorbid conditions can reduce symptoms of sexual addiction	Works best in sexual addiction secondary to or strongly exacerbated by comorbid condition
SSRIs	Anxiety, depression, obsessional ideation, sex drive	Reduces dysphoric affect, may reduce sex drive	Largely safe, but not without side effects
Anti-androgens	Destructive sex drive in male repeat sex offenders	Greatly reduces or eliminates sex drive	Severe possible side effects such as pulmonary embolism, bone mineral loss
OCD = obsessive-compulsive disorder; SSRIs = selective serotonin reuptake inhibitors.			
Source: [75]			*Table 2*

TREATMENT

Standard approaches for the treatment of sexual addiction include a variety of psychologic and pharmacologic options (***Table 2***). Understanding the characteristics of persons seeking help for out-of-control sexual behavior and differences among patients can help guide clinician decision-making for better patient-treatment matching.

Hypersexuality Subtypes

In patients seeking help for problematic sexual behavior, clinicians are repeatedly recommended to use a patient-tailored, multimodality approach, but there is little published guidance for matching clinical presentation with treatment options. Thus, a group of prominent sexual disorder researchers and clinicians developed a hypersexuality typology that links clinical profile and symptom clusters to suggested treatments. Diverse hypersexual behavioral patterns suggest they may be unrelated phenomena; assumed commonality is erroneous [136].

Paraphilic Hypersexuality

The paraphilic hypersexuality subtype has key features of paraphilic interests and a high frequency of one or more hypersexuality behaviors. Many of these patients lack the strong, internal directedness most men have in their sexual interest(s). Instead, they tend to test out or go along with partners' sexual interests. Interactions with sex workers are based on sex, and for many, a desire to become a part of the sex worker milieu [136].

Paraphilic hypersexuality is remarkable for the sizeable number reporting gynandromorphophilia, a rarely discussed erotic interest in persons with both male and female anatomy (usually full breasts and intact penis) typified by incompletely transitioned male-to-female transgender women. For many, this specific enduring erotic interest leads to confusion over sexual orientation or gender identity and hesitant self-reference as "mostly heterosexual" or as bisexual [136].

This clinical characterization of gynandromorphophilia risks pathologizing attraction to transgender women and the women themselves. Care should be taken to avoid doing so in practice settings. Methods to change paraphilic interests into euphilic attraction have no evidence or support. Treatment suggestions include integration of interests that can be expressed alone or with consenting partners, and harm reduction for those interests that cannot. Medication may be highly relevant. SSRIs can reduce libido and impulsivity, but can also delay or prevent ejaculation and lead to seeking greater stimulation sufficient to trigger orgasm [136; 182].

Avoidant Masturbation

The primary presenting complaint in patients with avoidant masturbation is the extreme time spent masturbating to pornography. These patients report masturbating many hours daily, job termination from online pornography or masturbation at work, academic failure, and social relationships and other important activities abandoned in favor of masturbation. Paraphilic interests are rare [136].

Many of these patients acknowledge masturbating to avoid tasks or chores. Anxiety or dysthymia are common; masturbation is the cause of, or the coping skill for, negative emotions. Avoidant masturbation does not always interfere with relationship satisfaction or sexual activity, but many have little interest in partnered sex and prefer masturbating to pornography [136].

Avoidance and procrastination motives drive the masturbation. In the gay male community, avoidance may be expressed as excessive time frequenting bathhouses, perusing online hook-up sites, or engaging in anonymous sex with large numbers of partners. With avoidance motivation, treatment is more productive when it addresses avoidance and procrastination instead of masturbation. Motivational interviewing can be very beneficial [136].

Chronic Adultery

In clinical settings, patients with chronic adultery are almost always male and outliers in the frequency of extramarital sexual activities involving one-time hook-ups, on-going relationships, or sex workers. Extreme time involvement seeking or engaging in sex is usually absent [136].

Chronic adulterers frequently state their wives or partners have a condition or history that inhibits sexual enjoyment, such as coital pain disorders (i.e., dyspareunia), low libido, sexual abuse history, or conservative/religious backgrounds. When partners present for therapy, their histories and libido levels usually match their husband's description. Patients may seek treatment motivated by secondary gains from appearing sincere about changing their behavior, without an actual desire to change. In practice, this is less common than assumed; chronic adulterers are generally motivated to change by the time they present for help [136].

Marital distress may be a consequence of most other hypersexuality subtypes, but it is antecedent to chronic adulterer behaviors. While couples therapy is strongly indicated, because many marital issues are unresolvable without partner engagement, spouses often instruct the chronic adulterer to go fix his problem and are unwilling to participate. Mismatching atypically high and low sex drives often predate the marriage; the sex drive discrepancy goes unaddressed, and the high libido is chronically expressed outside the marriage. Demanding the adulterer fix his problem in therapy also serves to distract from or excuse unaddressed issues of the spouse [136].

Sexual Guilt

Patients with sexual guilt self-label as hypersexual or sex addicted and express intense distress, but lack evidence of sexual behavior that exceeds peer group norms. Highly conservative (usually religious) family environments are typically reported; some willfully adopt moralistic standards in adulthood that exceed the dictates of their religious.

Initially presenting with hypersexuality complaints, further assessment will identify sexual guilt and broadly expressed anxiety over sexual urges or interests. Among these patients are androphilic men who reject or resist their sexual orientation and seek treatment for their perception of addiction or compulsion to have sex with men. Psychoeducation and permission-giving is beneficial, and several therapy approaches can address inaccurate beliefs about sexuality and peer norms. These beliefs may reflect explicit religious involvement or other deeply held ideologies, such as gender roles.

The Designated Patient

Romantic partners of designated patients demand they get help and instigate sex disorder clinical assessments. These demands may reflect highly restrictive sexual beliefs with zero-tolerance for masturbation, pornography, or non-procreative sex. Designated patients may self-label as sex addicts (or a similar term) but lack the sexual behavioral patterns. These patients state their partners explicitly believe the problem is theirs to solve. Designated patients are best helped by including the partner in psychoeducation on healthy masturbation and pornography use and communication and assertiveness training. Sex-positive clinicians quickly see partner demands as unnecessarily restrictive. Some patients know this, but agree to the restrictions for the benefit of the relationship.

Better Accounted for as a Symptom of Another Condition

Hypersexuality can be a symptom or sequelae of several non-sexual diagnoses, including some personality disorders, hypomania, developmental delay, disinhibiting brain injuries, neurologic disease, and medication side effects. Borderline personality disorder is the most common associated syndrome and can present as any hypersexuality subtype. In general, personality disorders are distinguished from hypersexualities by the presence of other personality disorder symptoms and not by differences in sexual behaviors.

Psychologic Interventions

Cognitive-Behavioral Therapy

CBT can help the patient identify and work through triggers of sexually addictive behavior, permission-giving thoughts that weaken inhibition of the behavior, and the negative consequences of the behavior. Specific CBT approaches have been modified to treat sexually addictive behavior [183; 184].

Motivational Interviewing

Motivational interviewing serves to help the patient consider the pros and cons of maintaining the addiction in the context of their overall goals and priorities [185]. Motivational interviewing is less focused on actual behavior change than on bolstering motivation to enter the change process. Advantages of motivational interviewing include the non-confrontational and nondirective nature, which can be more effective in those with low motivation to change [75].

Psychodynamic Therapy

Psychodynamic therapy has relatively few published clinical studies in sexual addiction treatment, but it appears to offer unique advantages [184]. Specifically, psychodynamic treatment can address the factors related to the individual's unique personal history and personality organization and can clarify the specific meaning the addictive behavior holds for the patient. For instance, sexual addiction can reflect a rebellion against a puritanical and authoritarian parent; a form of self-soothing in the context of parental abandonment; a narcissistic need for constant affirmation of one's attractiveness; or a desperate attempt to stave off feelings of emptiness. In most cases, sexually addictive behavior is thought to reflect a disturbance in the core self-concept and/or the capacity for intimate relationships [75].

Psychodynamic psychotherapists recognize the inherent human need for close intimate relationships to help regulate negative and positive emotions, and how this need must be reconciled with feeling dependent on people who remain, in large part, outside of one's control [186; 187]. For those with less ability or an inability to tolerate this dilemma, often resulting from maladaptive early attachment, an addictive object can provide a convenient end-run around the vulnerability and relative powerlessness inherent in intimate relationships. In this way, sexually addictive behavior can serve as a transitional object [75].

According to psychodynamic theory, sexually addictive behavior serves as a self-soothing and emotionally regulating "other" that remains under the individual's near-total control. The sexual partners in sexually addictive behavior are rarely experienced as "subjects"—three-dimensional people who can make an emotional impact on the sexually addicted individual outside of his or her control. Instead, they are fleeting "objects," used to gratify an urge and then discarded. Paradoxically, by insisting on total control over the attachment object, the sexually addicted individual loses control and ends up controlled by their addiction. Psychodynamic therapy can help the patient to understand how these dynamics play out in his or her life and to subsequently develop healthier relational patterns [75].

Group Psychotherapy

Group psychotherapy can be highly effective in the treatment of substance and behavioral addictions and, in some cases, is more effective than individual therapy alone [184]. Group therapy serves multiple purposes and probably works through a combination of mechanisms. It provides social support during the difficult change process, information about other group members' experiences and coping strategies, support for a positive new group identity, and confrontation of the denial and rationalizations associated with sexually addictive behavior [75]. Group therapy helps individuals feel less isolated and reduces feelings of shame [143].

Couples Therapy

Due to the negative impact of sex addiction on intimate relationships, couples therapy may offer both the patient and their partner guidance on dealing with this disorder [138].

Pharmacologic Interventions

Few pharmacotherapy studies in sex addiction have been published. The research that has been published focuses on male patients. Clinical experience indicates close monitoring is needed in treating compulsive sexual behavior, as some individuals may increase risky sexual behavior to compensate for the dampening effects of medication on sex drive [84].

In a study of gay and bisexual men with complaints of compulsive sexual behaviors, 12 weeks of citalopram led to greater reductions in desire for sex, frequency of masturbation, and hours of pornography use per week compared with placebo. However, the number of sexual partners during treatment did not differ [188].

Naltrexone was evaluated over eight weeks in non-addicted, methamphetamine-using, binge-drinking men who have sex with men at high risk for HIV. Naltrexone led to significantly greater reductions in overall and condom-less receptive anal intercourse than placebo. Among frequent methamphetamine users, naltrexone led to greater reductions in methamphetamine-using days. Higher naltrexone adherence also led to greater reductions in binge drinking days [189].

The importance of close patient monitoring is underscored by a case report of three heterosexual men (24 to 35 years of age) who developed new-onset problematic sex behaviors during paroxetine (an SSRI) treatment for pathologic pornography use and masturbation. After 10 weeks of paroxetine, all showed marked reductions in anxiety and pathologic pornography use and masturbation and return of normal sexual function. However, by three months, all had new-onset sexual behaviors; none had histories of risky sexual behaviors, affairs, paying for sex, or mania/hypomania symptoms during treatment [190]. Two were in long-term relationships (one married); one was single. All described pride in overcom-

ing inhibitions to take sexual risks with success and felt strongly motivated to continue. Both patients in relationships felt guilt but also elation over new sexual frontiers as both had remained with their first and only sex partner. None wanted to phase-out paid sexual services by increasing pornography and masturbation, which they described as isolating, time-consuming, and humiliating [190].

In this study, the authors explain that an imbalance in reward reactivity and threat reactivity promoted sexual risk-taking; problematic behaviors resulted from increased impulsivity or increased anxiety [190; 191]. All three patients regulated their high pretreatment anxiety levels with pathologic pornography use and masturbation. Paroxetine helped decrease amygdala reactivity, which decreased anxiety and subsequently decreased pathologic pornography use and masturbation, with new compulsive sexual behaviors potentially emerging from diminished anxiety [190].

When sexually disordered behavior presents a grave risk to the individual or society, such as persistent uncontrolled pedophilia, anti-androgen therapy has been effective for drastically reducing sexual drive. The side effects with this drug class are notable, and such medications are rarely used outside of serious circumstances in a forensic context [75].

Twelve-Step Programs

Sex Addicts Anonymous (SAA) is a support group with a purpose of helping others with sex addiction find recovery. This organization operates similarly to AA and uses the 12-step program of recovery. Another 12-step program is Sex and Love Addicts Anonymous (SLAA) [138]. Most SAA, SLAA, and Sexual Recovery Anonymous ("S" meetings) groups admit new members only after screening and approval by the group members. Meetings focus on the goal of helping the new member stop or control his/her pathologic sexual behavior and learn new coping strategies. "S" meeting groups can play an important role in the recovery of patients by helping them become honest with themselves and their families in an atmosphere of support and fellowship [135].

INTERNET GAMING DISORDER

The specific game genre is essential to understanding the behavioral addiction of Internet gaming disorder. The characteristics of genres attract online gamers with specific psychologic and personality features. Gamers at risk for Internet gaming disorder gravitate to genres that deliver potent reward and reinforcement. This disorder develops from the interaction of gamer and genre factors over time.

Massive, multiplayer, online role-playing games (MMORPGs) are unique in pathogenic potential among game genres. A 2015 report described a case of two brothers (22 and 19 years of age), high academic achievers from an intact, upper-middle class family who lived at home. Both began MMORPGs two years before hospital admission for Internet gaming disorder. In the initial months, time spent gaming progressed from 2 to 4 hours to 14 to 18 hours daily. Gaming interfered with their sleep and daily routine. Both deteriorated to the point that when engaged in MMORPGs, they urinated and defecated in their clothes, did not change clothes for days, did not bathe, skipped meals, and did not answer the phone. Fixated on gaming, they were indifferent to the presence of a burglar robbing their home. Both failed their classes, became abusive and violent when their gaming was disrupted, and were admitted to inpatient care by their parents [192].

INTERNET GAMING GENRES

Despite a large variety of available games online, players typically prefer a specific game genre. With Internet gaming disorder, genre greatly matters, and MMORPGs are the most frequently reported genre among people seeking help for an Internet gaming problem [193].

Massive, Multiplayer, Online Role-Playing Games

MMORPGs are played online by thousands of gamers simultaneously, spanning vast time zone differences without spatial or temporal boundaries. The design of MMORPGs borrows from operant conditioning, incorporating random, unpredictable reward structures to maximize player engagement and time involvement. The same intermittent schedule of rewards is featured in slot machines and other gambling modes [192].

In MMORPGs, players assume the role of a fictional character and take control of that character's actions while simultaneously interacting with other gamers in a shared, virtual world. This virtual world persists independently of the players; events and interactions between other players continue when one player goes offline [8]. To advance, the player's character acquires new powers, skills, special weapons, or currency as rewards for succeeding in a mission or special quest. Online social interactions occur among players in-game or by regrouping into clans (guilds), a persistent, virtual hierarchical social network of characters with shared backgrounds and objectives [8]. The aspects of a permanent world, reinforcement schedule, and online social interaction contribute to the addictive potential of MMORPGs [194].

The uniquely problematic potential of MMORPGs is under-recognized. A 2016 report stated that only 36.7% of surveyed Canadian psychiatrists believed there was an association between psychopathology and MMORPG engagement [195].

First-Person Shooter

First-person shooter (FPS) is an action game genre and differs markedly from MMORPGs. Players engage in combat with enemies, primarily using firearms. The shoot-to-kill style of FPS games is considered highly arousing and violent, but little research has studied Internet gaming disorder in excessive FPS players [193].

Real-Time Strategy

Real-time strategy games are strategic, combat-orientated games that often use forms of strategic simulation. In real-time strategy game matches, strategic decision-making ability directly influences the results and scores. For example, a gamer might hold a combat commander position that requires use of multiple strategies to take over an enemy fortress [193].

Other Genres

Other game genres include arcade games, online sports games, casual games, and other action games [193]. These games are rarely associated with Internet gaming disorder.

PATIENT CHARACTERISTICS AND GENRE PREFERENCE

To investigate Internet gaming disorder characteristics, genre preference, and their interaction, 212 Korean patients (18 to 23 years of age, 97.6% male) underwent extensive psychologic testing after meeting criteria for Internet gaming disorder [168]. The researchers found five common themes [193]:

- Involvement more than 4 hours per day or more than 30 hours per week
- Young Internet Addiction Scale (YIAS) score >50
- Irritable, anxious, and/or aggressive when forced to stop
- Behavioral problems, distress, and financial problems from excessive gaming
- Maladaptive disruption of diurnal rhythm (i.e., sleep all day, game all night), irregular meals, failure to maintain personal hygiene, school truancy or job loss

Patient preferences were for MMORPGs (39.2%), real-time strategy games (34.9%), FPSs (16.0%), and other genres (9.9%) [193].

MMORPGs

Of all genres, MMORPG players have the highest Internet gaming disorder severity, partially explained by the variable reinforcement schedule (similar to gambling). Among gamers, MMORPG players show the greatest levels of social anxiety and social avoidance, suggesting that social dysfunction drives gamer motivation to escape interpersonal anxiety and problems. Gamers with high social anxiety and avoidance are often stress intolerant and easily distressed over interpersonal and academic problems. The MMOPRG alternate reality space provides an escape [193].

Some studies show that MMORPG gamers motivated to escape reality experience greater consequence than gamers with other motives for gaming, and escapism is a strong predictor of Internet gaming disorder [194; 196]. Other research shows that MMORPGs interfere with player development of social and emotional skills; this may exacerbate deficient interpersonal skills to create a vicious cycle [194; 197].

Other studies comparing Internet gaming genres found MMORPG players spent significantly more hours playing, had worse health (e.g., more cigarettes smoked, less physical exercise, worse sleep quality), and greater interference in real-life socializing and academic work [197]. MMORPG players (compared with those playing other genres) more frequently displayed signs of dysfunctional use [8; 194].

Escapists use MMORPGs to avoid interpersonal and life stressors, and in-person social support is adversely impacted by the high time involvement. Social support increases online but decreases offline. Offline social support has the strongest impact on favorably affecting gamer well-being, and greater online social support for offline problems did not offset the negative effect of diminished offline support in MMORPG escapists [198].

First-Person Shooter

Social anxiety is lowest in FPS gamers. FPS players control the game character from a first-person view, making self-confidence crucial for high scores and engagement in competition. Most online FPS players do not play in isolation, and social interaction motivation highly predicts FPS time involvement. FPS players are drawn to competition and challenge [199]. The highly immersive nature and potential for online competition, achievements, and social interaction can reinforce excessive FPS involvement. Social-seeking behavior is inversely related to social anxiety and avoidance [193].

Real-Time Strategy

Among the four genre groups, self-esteem was highest in the real-time strategy group, suggesting that the strategic simulation information tasks are more rewarding in gamers with stronger self-esteem. Real-time strategy involves many challenging tasks requiring rapid switching, multiple information sources, and multiple actions for which substantial cognitive flexibility is needed [200]. Successful players must cope with simultaneous and rapidly evolving game situations in real-time, while managing funds, resources, and information concerning their opponent. When managing multiple, simultaneous game strategies, self-esteem can have a decisive effect on judgment and decision-making by reducing gamer anxiety and enhancing performance. Compared to MMORPG players, the lower social anxiety and avoidance scores of real-time strategy players may be related to higher self-esteem. Thus, relatively high self-esteem and low social anxiety and avoidance are the main characteristics in the real-time strategy group [193].

Others

Compared to the other three genres, players of arcade, online sports, and casual games had higher depression scores, with Beck Depression Inventory scores showing "moderate depression," compared with "mild depression" average scores in the other

three groups. Games in this category are relatively simple in design and low in complexity, and this game preference is probably motivated by wanting a less challenging game, possibly influenced by the presence of low volition or energy, impaired concentration, or depressed mood [193].

PREVALENCE

The pooled prevalence estimates from more than 30 countries showed problematic Internet game use in 8% to 12% of young persons and addictive use in 2% to 5% of children, adolescents, and college students [17]. Nationally representative American samples show an Internet gaming disorder prevalence of 8.5% among those 8 to 18 years of age [201; 202].

RISK FACTORS

Internet gaming disorder is associated with increased neuroticism, aggression/hostility, avoidant and schizoid interpersonal tendencies, loneliness, introversion, social inhibition, boredom inclination, sensation-seeking, low agreeableness, diminished self-control, narcissistic personality traits, low self-esteem, state and trait anxiety, and low emotional intelligence. These personality factors are not specific to pathologic gaming and contribution to, or direction of, causality is unclear from the research designs [17]. Gamers with Internet gaming disorder show high rates of comorbidity, including ADHD, generalized anxiety disorder, panic disorder, depression, social phobia, school phobia, and various psychosomatic symptoms [17].

ASSOCIATED FEATURES

Hostility, defined as thoughts, feelings, and behaviors expressing the negative affect state of anger, including aggression, irritability, rage, and resentment, is relatively common in adolescence. It is linked to the onset of substance use disorders, and the relationship between hostility and Internet gaming disorder in adolescence is bidirectional. Hostility may lead to Internet gaming escape-avoidance and withdrawal as a coping mechanism for emotional distress, while the anonymity and absence of oversight can make the Internet an outlet for more hostile adolescents to express aggression unacceptable offline [203; 204]. As an ineffective form of coping, Internet gaming disorder could lead to poor adjustment in early adulthood, precipitating hostility [203]. The emotional regulation strategies (e.g., suppressing emotions) that characterize individuals with Internet gaming disorder may be contributing factors to their hostile tendencies [205].

In one study, adults (mean age: 35.8 years) were assessed for "social media addiction" (presence of core addiction elements), Internet gaming disorder, and risk factors for both. Social media addiction was associated with being female and single, younger age, ADHD, OCD, anxiety, and lower levels of depression. Internet gaming disorder was positively associated with being male and single, younger age, ADHD, OCD, and depression, and inversely related to anxiety symptoms. ADHD, OCD, anxiety, and depression contributed to the variance in social media addiction (15%) and Internet gaming disorder (7%) after controlling for age, sex, relationship status, and education [206]. Social media addiction and Internet gaming disorder had low inter-correlation and were associated with different variables, which along with many other studies dispels the idea of a unified construct of Internet use disorder or Internet addiction [206].

Adverse Outcomes

Numerous studies indicate that, for a player subgroup, excessive online gaming can lead to diverse negative psychosocial consequences, including sacrificing work, education, hobbies, socializing, time with partner/family, and sleep, as well as increased stress, absence of real-life relationships, lower psychosocial well-being, loneliness, worse social skills, decreased academic achievement, increased inattention, aggressive/oppositional behavior and hostility, maladaptive coping, decreases in verbal memory performance, maladaptive cognitions, and suicidal ideation [17].

One study evaluated children enrolled in grades 3 to 8 over two years. Higher Internet game involvement, lower social competence, and greater impulsivity were risk factors for developing Internet gaming disorder, while depression, anxiety, social phobia, and declining academic performance were outcomes of Internet gaming disorder. The prevalence rate of Internet gaming disorder was 9%. The authors concluded that Internet gaming disorder can develop without premorbid disorders and persist for years in young Internet gamers [207].

When considering the applicability of cross-cultural epidemiology and risk factors in Internet gaming disorder, cultural differences in attitudes towards Internet use in East Asia and elsewhere should be noted. For instance, parents in East Asia tend to pathologize behaviors that interfere with family or educational pursuits, possibly inflating Internet gaming disorder prevalence rates in Taiwan and South Korea [208]. Individuals who appear addicted to using social media and social networking sites are often engaged online to psychologically compensate for the lack of offline social rewards [15].

Psychologic Factors

Significant predictors of pathologic gaming among adult MMORPG players include expressing "true self" in game, higher levels of social anxiety, larger numbers of in-game social supports, and fewer supportive offline relationships. The number of in-game supports was significantly associated with time spent playing [209].

CBT that addresses specific gaming-related cognitions in Internet gaming disorder may have greater benefit than general approaches that solely address "preoccupation with gaming" (a DSM-5 criterion for Internet gaming disorder). Maladaptive cognitions associated with Internet gaming disorder have been identified in cross-sectional studies, but the extent to which these cognitions predict future pathologic gaming behavior requires longitudinal data. In 465 Internet gamers (84% male, mean age: 26.2 years) followed over 12 months, those who developed Internet gaming disorder had significantly higher baseline scores in perfectionism, cognitive salience, and guilt compared with gamers remaining non-problematic. Pathologic gamers who successfully transitioned to being non-problematic gamers had lower baseline perfectionism scores compared with those whose use remained problematic. Cognitive changes accounted for 28% of variance in scores beyond contributions from gender, age, and gaming frequency. Maladaptive gaming-related cognitions could be assessed to better inform treatment targets for CBT [210].

Specific achievement- and immersion-related motives have been identified as strong predictors of problematic online gaming, addiction symptoms, and adverse impact on daily life. Of these motives, problematic use was consistently associated with "advancement" (e.g., the desire to gain power, progress rapidly, accumulate in-game symbols of wealth or status) and "mechanics" (i.e., interest in the rules and system to optimize performance). Immersion-related motives highlighted "escapism." Escapers are motivated by immersion in a virtual reality instead of by reaching game-specific objectives; the game is a dissociative method to escape offline thoughts and problems. Social-related motives (e.g., playing to meet people, teamwork) were unrelated to problematic use [8].

Internet gaming disorder is linked to impulsivity and poor self-regulation, supported by the multidimensional UPPS-P model of impulsivity that describes impulsivity as five related but specific dimensions [211]:

- **U**rgency: Acts rashly during intense positive and negative emotions
- **P**remeditation: Considers consequences of an act before engaging in that act
- **P**erseverance: Capacity to remain focused on boring and/or difficult tasks
- **S**ensation seeking: Tendency to enjoy and pursue new and exciting activities
- **P**ositive urgency: Tendency to act rashly under extreme positive emotions

Many studies highlight the strong links between these impulsivity factors and psychiatric disorders such as substance use disorders, gambling disorder, compulsive buying, suicidal ideation/attempts, and aggressive behaviors.

The "urgency" subtype of impulsivity may predict pathologic MMORPG use. Urgency, poor premeditation, and poor perseverance impede the executive functioning required for self-control, inhibitory control, decision-making ability, and resistance to cognitive interference [8].

Internet gaming disorder symptom severity may be highly relevant in adolescents 16 to 18 years of age, which precedes the age of highest Internet use (18 to 24 years). In one study, individuals more hostile at 18 years of age continued MMORPG involvement with higher Internet gaming disorder symptom severity after two years. These results agree with other longitudinal studies that show stability in addictive behavior during adolescence and onset of Internet gaming disorder coinciding with increased real-life problems [207; 212]. In contrast, Internet gaming disorder is more transient in adults; this disparity may reflect age-related differences in the progression of addiction [203; 213].

Among 3,041 adult recent Internet gamers (60% male), 13.8% met DMS-5 criteria for Internet gaming disorder. Compared with healthy gamers, the Internet gaming disorder group used gaming significantly more often to escape reality and specific issues or as a coping mechanism. They also scored significantly higher on measures of obsession-compulsion, depression, somatization symptoms, interpersonal sensitivity, and impulsivity. Adults with Internet gaming disorder experienced more frequent difficulty in concentrating, loneliness, and insomnia, and were five times more likely to attempt suicide and exhibit aggression. Unlike youth with Internet gaming disorder, no game genre was dominant in adult Internet gaming disorder [214].

Maladaptive Cognitions in Internet Gaming Disorder

Internet gaming disorder-related cognition is more complex than "preoccupation." The primary underlying cognitive factors in Internet gaming disorder (and therapeutic targets for CBT) include [215; 216]:

- Persistent overvaluation of video gaming rewards, activities, and identities
- Over-reliance on gaming to meet self-esteem needs
- Gaming as a method to gain social acceptance
- Maladaptive, inflexible adherence to internal rules dictating gaming behavior

In a study of adolescents, those with Internet gaming disorder reported significantly more maladaptive gaming beliefs than non-disordered respondents. The association between gaming cognitions and Internet gaming disorder symptoms was very large and independent of gaming activity and psychologic distress. The distinct problematic cognitions of adolescents with Internet gaming disorder should be addressed in any therapeutic intervention [217].

PATHOPHYSIOLOGY

Neuroimaging studies comparing patients with Internet gaming disorder to matched controls have repeatedly identified the following pathophysiology [8; 194; 216; 218]:

- Resting-state imaging: Alteration in brain regions that mediate attentional control, impulse control, emotional regulation, and sensory-motor coordination
- Presentation of gaming images (cue exposure): Alteration in reward inhibition mechanisms and loss of control
- Functional studies: Abnormal dopamine release, altered dopamine levels, decreased dopamine transporter availability, and decreased dopamine receptor D2 occupancy that reflect an overall reward deficiency involving decreased dopaminergic activity

- Structural studies: Decreased white matter density in regions involved in decision-making, behavioral inhibition, and emotional regulation; altered orbitofrontal cortex gyrus with greater Internet gaming salience and loss of control; ventral striatum (centrally involved in brain reward mechanisms) volume alterations related to changes in reward sensitivity
- Treatment studies: Reduction in Internet gaming craving and reductions in associated brain activity

In aggregate, these findings show that Internet gaming disorder is associated with neuroadaptation of reward and reinforcement pathways that develop from prolonged hyper-stimulation and structural alteration in brain regions that mediate executive functioning. While Internet gaming disorder pathophysiology highly resembles that of substance addiction, this research is early-stage and such conclusions are tentative [218; 219].

Addicted Internet gamers show patterns of physiologic arousal deficits that differ by game genre. In addicted MMORPG gamers, cardiovascular activity (as measured by heart rate, blood pressure, and skin conductance) significantly decreases during gaming compared with pre-game baseline but significantly rose after gaming. Addicted FPS gamers had significant increases in blood pressure during gaming, decreasing significantly post-game. Non-addicted MMORPG and FPS gamers showed increased blood pressure during and after gaming [220].

ASSESSMENT AND DIAGNOSIS

Clinical assessment of patients with suspected Internet gaming disorder is necessary to explore the clinical severity and negative biopsychosocial impact via inquiry (***Table 3***).

The DSM-5 Task Force asked the Substance-Related Disorders Work Group to consider several potential behavioral addictions for inclusion in the DSM-5. The Work Group suggested Internet gaming disorder placement in Section III (Emerging Measures and Models). While the body of published evidence was extensive, the research did not apply standard diagnostic criteria; substance use disorder criteria were borrowed by some studies, and impulse control disorder or pathologic gambling criteria were borrowed by others. The DSM-5 included proposed criteria for Internet gaming disorder with the hope of standardizing emerging work in the field [12]. In this proposed framework, a diagnosis is given when five or more of the following criteria are met [11]:

- Preoccupation with Internet games
- Withdrawal symptoms when Internet gaming is taken away (e.g., irritability, anxiety, sadness)
- Tolerance (i.e., the need to spend an increasing amount of time engaged in Internet games)
- Unsuccessful attempts to control participation in Internet games
- Loss of interest in previous hobbies and entertainment
- Continued excessive use of Internet games despite knowledge of psychosocial problems
- Deception of family members, therapists, or others regarding the amount of Internet gaming
- Use of Internet games to escape or relieve a negative mood
- Has jeopardized or lost a significant relationship, job, or educational or career opportunity because of participation in Internet games

The ICD-11 formally includes as diagnostic entities gaming disorder (predominately online) and gaming disorder (predominately offline) [108].

CLINICAL INTERVIEW QUESTIONS TO ASSESS INTERNET GAMING DISORDER	
Presenting problem	When did you begin to notice problems with your gaming? How long have you been gaming? How much time do you spend gaming each day? Week? Month? What was going on in your life when you began gaming? What was going on in your life when you began to have difficulties with your gaming?
Biologic factors	Are you experiencing or have you previously experienced any health concerns? If so, please describe. How have these health concerns been impacted by your gaming? What treatment have you received for these health concerns? Does your gaming interfere with your sleep/meal schedule? What exercise patterns do you engage in? What drugs and/or medications do you take? How much? How often? In the past?
Psychologic factors	What types of gaming do you engage in? What do you like/dislike about gaming? How do you feel before, during, and after gaming? What are your thoughts before, during, and after gaming? Have you ever used gaming to help improve your mood or change your thoughts? In what places at home or elsewhere do you usually engage in gaming? Have you ever felt anxious, depressed, or isolated when not gaming?
Social factors	Has your gaming caused concerns with your family? Has your gaming caused concerns with your significant other? Has your gaming caused concerns with your social activities and friendships? Has your gaming interfered with your performance at school or work? What are your social/leisure/hobby activities?
Relapse prevention	Do you believe that you have a problem with your level of gaming? What do you see as the benefits and costs of continued gaming? What is your level of commitment to change your current gaming patterns? What plans have you implemented in the past to deal with your level of gaming? Have these plans worked?

Source: [202] *Table 3*

A comprehensive understanding of patient motivation is necessary for optimal treatment selection. The Motives for Online Gaming Questionnaire (MOGQ) was developed to assist in this assessment. The MOGQ consists of 27 statements, such as "I play online games because gaming helps me to forget about daily hassles," which are rated from 1 (almost never/never) to 5 (almost always/always). Motivational factors identified by the MOGQ include social, escape, competition, coping, skill development, fantasy, and recreation [12; 221].

TREATMENT

Psychologic Interventions

In the published research to date, psychologic treatment is the dominant modality for Internet gaming disorder, with CBTs the most-studied.

Cognitive-Behavioral Therapies

As discussed, maladaptive cognitions are a core feature of Internet gaming disorder and include self-regulation deficits, preference for a virtual life, cognitive bias, impaired cognitive control abilities, cognitive deficits, poor cognitive error processing, and decision-making deficits [202]. CBT addresses

these cognitions and is the most widely used psychologic treatment for Internet gaming disorder. The first stage of CBT treatment for Internet gaming disorder deals with the behavioral aspects of addicted gaming, and subsequent stages gradually shift the focus toward the development of positive cognitive assumptions. During therapy, addicted gamers identify false beliefs and learn how to modify them into more adaptive ones. CBT also trains patients to monitor their thoughts and to identify affective and situational triggers associated with their addictive gaming behavior [202].

In the treatment of Internet gaming disorder, CBT typically involves 8 to 28 sessions that address psychoeducation, problem identification, healthy communication, Internet awareness, and cessation techniques. Treatment of Internet gaming disorder may also include short-term therapy, group therapy, and systemic therapy with parents/teachers/peer support and multilevel interventions that incorporate motivational interviewing [202].

Although CBT has some efficacy in Internet gaming disorder, the results do not appear to differ from other non-specialized psychologic treatments for Internet gaming disorder [222]. One study found that combinations of CBT and medications showed an advantage over monotherapies [223]. To improve patient response and clinical outcomes with standard psychologic approaches, therapy modalities have been developed that target the cognitive pathologies and underlying neural substrates specific to Internet gaming disorder [224].

Motivational Enhancement Therapy/CBT

CBT is combined with motivational enhancement therapy (MET/CBT) to move patients from ambivalence to engagement concerning behavioral change. MET/CBT is structured by stages:

- Contemplation stage: Rapport building during the initial sessions includes a detailed interview and case formulation.
- Preparation stage: The therapist creates an empathetic environment to deliver psychoeducation, including instruction on managing physiologic and emotional arousal through relaxation techniques and a cost-benefit analysis of gaming addiction.
- Contract stage: With the patient and parents (if appropriate), the therapist addresses behavior modification of gaming, reducing time spent online and promoting healthy activities.

MET/CBT has led to decreased symptoms and improved school performance in Internet gaming disorder [202; 225].

Craving Behavioral Intervention

Based on cognitive neuroscience and behavioral addiction research, craving behavioral intervention was developed for patients with Internet gaming disorder to reduce cravings and enhance coping skills, which may improve therapeutic outcomes and prevent relapse. Craving behavioral intervention is conducted weekly in a group format with eight to nine participants. Topics for each weekly session include [62]:

- Perceiving subjective craving
- Recognizing and testing irrational beliefs regarding craving
- Detecting craving and relieving craving-related negative emotions
- Coping with cravings and altering participant fulfillment of psychologic needs
- Learning time management and skills training for coping with cravings
- Reviewing, practicing, and implementing skills
- Mindfulness training

Short-Term Treatment of Internet and Computer Game Addiction

Short-term treatment of Internet and computer game addiction (STICA) is a disorder-specific, CBT-based, individual and group treatment lasting three months. The STICA treatment protocol and focus of treatment sessions is organized into three phases [226]. In the early phase, the focus is on patient education on the mechanisms and effects of Internet gaming disorder (e.g., learning theories, development and consequences of Internet gaming disorder, the vicious cycle of addiction). Interventions promote social communication and bonding in the therapy setting. In the middle phase, patients are taught to identify triggers of dysfunctional Internet use (e.g., emotional states, maladaptive cognitions, daily stress) by keeping diaries. A functional analysis of addictive behavior is done, with the goal of enabling functional computer and Internet use by appropriate problem-solving strategies. Patients establish an in-person social network and build a repertoire of alternative activities. Sessions may include exposition training (i.e., confronting and deleting access to critical genre elements, such as the self-created avatar) and skills training (e.g., coping with stress and problems, social skills, building alternative activities) [226]. The last phase is termination and relapse prevention. During this phase, treatment tools are transferred to daily life, including functional computer/Internet use. Relapse prevention tools are provided and practiced.

The efficacy and stability of outcomes following STICA are under evaluation. Early results indicate that nearly 70% of initially enrolled patients completed the three-month treatment, with six-month follow-up showing significant decreases in Internet use, online time, and psychiatric symptomatology [227; 228].

Pharmacologic Interventions

Few pharmacotherapy studies have been published in Internet gaming disorder. In one study, adolescents and adults with Internet gaming disorder were randomized to bupropion sustained-release, escitalopram, or a no-treatment control group. After six weeks of therapy, bupropion and escitalopram significantly improved all clinical symptom scores compared with controls. Bupropion led to greater improvements than escitalopram in Internet addiction severity, depression, ADHD, behavioral inhibition and activation, and global functioning. Bupropion and escitalopram were effective in reducing Internet gaming disorder symptoms, but bupropion was more effective in improving attention and impulsivity, important in the management of Internet gaming disorder [229].

Eleven patients with Internet gaming disorder received bupropion treatment for six weeks. Pretreatment MRI imaging showed higher brain activation in the left occipital cuneus, left dorsolateral PFC, and left parahippocampal gyrus compared with healthy controls. After six weeks of bupropion treatment, craving for Internet gaming and cue-induced brain activity in the dorsolateral PFC decreased from pretreatment in patients with Internet gaming disorder [230].

Atomoxetine and methylphenidate were compared in 86 adolescents with Internet gaming disorder and ADHD. After 12 weeks of treatment, both atomoxetine and methylphenidate reduced symptom severity; this reduction correlated with reductions in impulsivity. These results suggest impulsivity may contribute to the development of Internet gaming disorder [168].

BINGE EATING DISORDER

In the terminology for addictive patterns of eating, "food addiction" is seen as a misnomer that conveys the largely disproven concept that certain foods possess inherent properties that promote addictive behavior. More appropriate terms are "eating addiction" or "addictive eating disorder," because they correctly associate addictive patterns of food consumption with behavior originating from individual predisposition [231].

There is increasing concern that consumption of food with high sugar content may be "addictive" and promote weight gain. Claims that sugar has addictive potential are based on animal studies, but direct human evidence for symptoms of sugar-related substance dependence is lacking [232]. A study evaluated 1,495 university students for potential "food addiction" (with DSM-IV substance dependence criteria applied to food) involving high-sugar foods. In this group, 12.6% met food addiction criteria; of these, 5% mainly consumed sugar-laden foods. Overweight/obesity was unrelated to sugary food preference. "Food addiction" was concluded to result from unique individual characteristics that determined reward response to food and promoted excessive eating [232].

EPIDEMIOLOGY

The lifetime prevalence of binge eating disorder in American adults is 0.8%, based on DSM-5 criteria [233; 234]. A World Health Organization survey of more than 24,000 adults in 14 mostly upper-middle and high-income countries found lifetime prevalence of binge eating disorder ranging from 0.2% to 4.7%, with the United States second in prevalence only to Brazil [235].

Binge eating disorder is more common in women (3.5%) than men (2%), and in younger and middle-aged adults than those older than 60 years of age [80]. The prevalence is higher in obese than non-obese persons. The lifetime prevalence of binge eating disorder appears slightly lower among Latino and Asian Americans (1.9% and 2.0%, respectively) compared with the general population; the prevalence is similar among Hispanic and non-Hispanic whites [234; 236].

While binge eating disorder is found across all weight categories, binge eating is a strong risk factor for obesity, and roughly 25% of those seeking treatment for obesity meet binge eating disorder criteria [237].

Binge eating disorder has a higher lifetime prevalence in the United States and worldwide than bulimia nervosa and anorexia nervosa combined [234]. Unlike bulimia nervosa and anorexia nervosa, binge eating disorder is relatively common in both sexes and across ethnic/racial minority groups, and is distributed across broader age groups [234]. As noted, binge eating disorder is significantly associated with elevated rates of medical and psychiatric comorbidity and impairment. It shares some features with, but is distinct from, the other eating disorders and obesity [238; 239; 240].

Binge eating disorder is typically first diagnosed during the early to mid-20s, with symptoms usually persisting well beyond mid-life. The disease course may crossover to and from bulimia nervosa and anorexia nervosa. Binge eating disorder is associated with significant role impairment and relationship dissatisfaction. It is considered a significant public health problem independently as well as for its association with chronic pain, other psychiatric disorders, obesity, and diabetes [241; 242; 243].

PATHOPHYSIOLOGY

Evidence suggests that binge eating disorder overlaps with other behavioral and substance addictions. Persons with binge eating disorder show impairments across a range of compulsivity indices comparable to those with alcohol use disorder [42; 244].

Binge-eating episodes may be triggered by a breakdown in self-regulation induced by sudden increases in negative affect and tension. Impairment in executive function is consistently found in eating disorders. With strong impulses or urges to eat, lack of

inhibitory control may lead to impulsive eating and obesity in some [245]. In addition, dysregulation in dopamine, opioid, acetylcholine, and serotonin neurocircuitry in brain reward regions is implicated in the pathogenesis and maintenance of binge eating disorder [246].

There is growing interest in and debate about whether an addictive process contributes to problematic eating outcomes such as obesity. Craving is a core component of addiction but is little-studied in the context of addictive eating. In a community sample, overall food craving partially mediated the links between addictive eating and elevated body mass index (BMI) and binge-eating episodes. Cravings for sweets and carbohydrates mediated the link between addictive eating and binge episodes. Cravings for fats mediated the link between addictive eating and elevated BMI. Craving was a key contributor to the link between addictive eating and pathologic outcomes [247].

Individuals with obesity vary in their experience of food cravings and reward-driven eating; both can be modulated by the neural reward system rather than physiologic hunger. In a trial that randomized women with obesity to naltrexone or placebo, a subgroup showed that addictive eating behavior and food-craving intensity was linked to reward-driven eating, a trait-like index of three factors (loss of control over eating, lack of satiation, and preoccupation with food). In this subgroup, opioidergic blockade with naltrexone reduced the positive association between reward-driven eating and craving intensity [248].

The stimulant lisdexamfetamine was approved for binge eating disorder treatment by the U.S. Food and Drug Administration (FDA) in 2015. Lisdexamfetamine consistently leads to weight decreases of 5.2% to 6.25%. Psychostimulants are known to facilitate response inhibition by modulating PFC function. Despite successful reduction or remission of binge eating with antidepressants or psychosocial treatment, significant weight loss in these patients has not been reported. Through modulation of prefrontal function, lisdexamfetamine may lead to additional improvements in inhibitory control that account for weight loss [246].

DIAGNOSIS

The Yale Food Addiction Scale, based on DSM-IV criteria for substance dependence, was the first assessment tool for addictive eating behavior. Despite the "food addiction" naming, this scale assesses eating behavior and identifies eating patterns similar to behaviors in substance addiction [231].

Binge eating disorder was reclassified from a provisional diagnosis in the DSM-IV to a formal eating disorder diagnosis in the DSM-5 (***Table 4***) [11; 249; 250]. This disorder is characterized by recurrent episodes of binge eating, defined as eating patterns during discrete periods (i.e., two or more hours) that involve excessive, rapid food consumption. Other core features include a sense of lacking control over eating during binge episodes, significant psychologic distress (e.g., shame, guilt) about binge eating, and the absence of recurrent compensatory behaviors (e.g., purging) [11].

A sense of loss of control during binge episodes is a core feature of binge eating disorder. The term "loss-of-control eating" is used to describe these episodes, but it is also used more broadly throughout the literature to describe binge-like eating behavior accompanied by a sense of loss of control that occurs across a wide spectrum of individuals. That spectrum includes, among others, individuals who exhibit some features of binge eating disorder but do not meet full diagnostic criteria for the disorder (i.e., subthreshold binge eating disorder) and individuals with other eating disorders [80].

The spectrum of loss-of-control eating also includes individuals for whom meeting the diagnosis threshold of binge eating disorder is uniquely challenging, such as children and those who have undergone bariatric surgery. Bariatric surgery significantly reduces stomach size and capacity, rendering it physically impossible to meet the binge eating disorder criterion specifying that a larger than usual amount of food is ingested. In the bariatric surgery literature, loss-of-control eating is used to describe binge-like behavior that falls short of meeting this criterion and to describe eating behavior that is contraindicated based on meal size and meal content. Children may not meet the binge eating disorder criterion for a larger-than-normal amount of food because their

DSM-5 DIAGNOSTIC CRITERIA FOR BINGE EATING DISORDER	
Criteria Set	**Specific Definitions for Each Criterion**
Criterion 1	Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following: • Eating, in a discrete period of time (e.g., within any two-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances • The sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating)
Criterion 2	Binge-eating episodes are associated with three or more of the following: • Eating much more rapidly than normal • Eating until feeling uncomfortably full • Eating large amounts of food when not feeling physically hungry • Eating alone because of being embarrassed by how much one is eating • Feeling disgusted with oneself, depressed, or very guilty after overeating
Criterion 3	Marked distress regarding binge eating is present
Criterion 4	The binge eating occurs, on average, at least one day per week for three months
Criterion 5	The binge eating is not associated with the regular use of inappropriate compensatory behavior (e.g., purging, fasting, excessive exercise) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa
Severity grading	Mild: 1 to 3 episodes per week Moderate: 4 to 7 episodes per week Severe: 8 to 13 episodes per week Extreme: 14 or more episodes per week
Source: [11]	*Table 4*

parents or others limit the quantity of food they consume or because they are unable to accurately quantify the amount they eat [80]. Nevertheless, loss-of-control eating is one of the most commonly reported disordered eating behaviors in children and adolescents and is particularly common in youth who have overweight or obesity [251; 252].

TREATMENT

Binge eating disorder treatment addresses the core behavioral (binge eating) and psychologic (distress and eating, weight, and shape concerns) features of the condition. Other important targets include metabolic health (in patients with obesity, diabetes, or both) and mood regulation (in patients with comorbid depression or anxiety) [80; 253; 254].

Although binge eating disorder is found across all weight categories and overweight/obesity is not a criterion of binge eating disorder, weight loss has been considered an important treatment outcome, due to its strong association with the disorder and the heightened risk for obesity and associated medical comorbidities [255].

Psychologic Interventions

Cognitive-Behavioral Therapy

Therapy variants in binge eating disorder include CBT combined with body image exposure or cognitive restructuring components, and ecologic momentary assessment to increase self-monitoring adherence [80; 253; 256]. A meta-analysis of studies that randomized patients to CBT or non-treatment (control) found improved binge frequency and abstinence outcomes with therapist-led, partially therapist-led, structured self-help, and guided self-help CBT. Therapist-led CBT outcomes were compelling, with a higher rate of abstinence (59% CBT vs. 11% control), reduction of binge episodes, reduced patient hunger and eating concerns, and improved sense of control over eating. Guided self-

help CBT reduced global eating-related psychopathology. When all study results were pooled, CBT and control groups were similar in weight lost and depressive symptom reductions. Therapist-led CBT was much more effective in improving key behavioral and eating-specific psychologic domains [80].

In a separate study, patients with clinical or subclinical binge eating disorder were randomized to individual or group CBT. Significant reductions in binge eating frequency and mild weight reduction were shown in both CBT formats at three-year follow-up. Predictors of full, long-term recovery were absence of treatment history with amphetamine derivatives, lower emotional eating, and lower pretreatment binge eating severity. Low emotional eating was the only predictor of weight reduction. Predictors of treatment resistance included overweight in childhood, full binge eating disorder diagnosis, and high prevalence of emotional eating [257].

Interpersonal Psychotherapy

Patients with binge eating disorder receiving therapist-led CBT and IPT showed differing abstinence trajectories at 46-month follow-up. Patients receiving CBT had high initial abstinence (81%) at the end of treatment, declining to 52% at final follow-up. Patients undergoing IPT had lower post-treatment abstinence (64%), but this increased (to 77%) by last follow-up [258].

Group Psychodynamic Interpersonal Psychotherapy

Compared with patients on a wait list, patients receiving 16 weekly group PIPT sessions showed greater binge frequency reductions (-0.5 vs. -3.0 binge days per week) and greater binge-eating abstinence (9.1% vs. 59.5%) [259]. Group PIPT also led to greater improvements in dietary restraint, depression, and interpersonal problems, but it did not differ from controls in BMI, self-esteem, or susceptibility to hunger at the end of treatment [80].

Behavioral Weight Loss

Behavioral weight loss treatment in binge eating disorder incorporates various behavioral strategies to promote weight loss, such as caloric restriction and increased physical activity. A meta-analysis compared CBT and behavioral weight loss on measures of binge frequency, abstinence, weight, and eating-related psychopathology [80]. Both CBT and behavioral weight loss were associated with substantial improvement in binge frequency, but CBT was superior to behavioral weight loss at the end of treatment and at 12-month follow-up. Behavioral weight loss led to larger BMI reductions than CBT at the end of treatment. There were no differences in binge-eating abstinence or eating-related psychopathology at any point in the study [80].

Pharmacotherapy

Overall, strong evidence shows that second-generation antidepressants, lisdexamfetamine, and therapist-led CBT increases the likelihood of achieving abstinence. CBT and second-generation antidepressants reduce binge frequency, and second-generation antidepressants reduce obsessions and compulsions related to binge eating. Lisdexamfetamine reduces binge frequency and obsessions and compulsions related to binge eating [80].

Lisdexamfetamine

The prodrug lisdexamfetamine is the sole FDA-approved drug for the treatment of moderate-to-severe binge eating disorder. The FDA has withdrawn several investigational or approved binge eating disorder pharmacotherapies from the U.S. market over safety concerns and adverse effects (e.g., sibutramine, rimonabant, d-fenfluramine) [80].

Lisdexamfetamine improved binge-eating outcomes in several rigorously designed studies. In aggregate, patients treated with lisdexamfetamine were 2.61 times more likely to achieve binge-eating abstinence than with placebo [80]. Lisdexamfetamine led to greater reduction in binge-eating days than placebo and was associated with superior eating-related psychopathology and weight-reduction outcomes.

The most common side effects reported by patients receiving lisdexamfetamine were gastrointestinal upset, sympathetic nervous system arousal, insomnia, headache, and decreased appetite [80].

Lisdexamfetamine product labeling includes a "limitation of use" that the drug is not indicated for weight loss, its effects on obesity are unknown, and similar medication classes have been associated with cardiovascular adverse events in the past. Lisdexamfetamine is also considered a controlled substance, and the product labeling includes a warning that CNS stimulants have high potential for abuse and dependence. Careful assessment and on-going monitoring for signs of misuse are indicated [255].

Another FDA-approved drug for ADHD treatment, atomoxetine, was significantly superior to placebo in reducing binge eating and achieving binge-eating remission in a small randomized controlled trial [255]. However, its use in the treatment of binge eating disorder is off-label.

Selective Serotonin Reuptake Inhibitors

SSRIs are the most frequently studied binge eating disorder medications, likely due to the efficacy and FDA approval of fluoxetine for bulimia nervosa [255]. Fluoxetine was superior on some measures of binge eating to placebo in a 6-week study, but not in a 16-week trial. In the six-week study, fluoxetine rapidly reduced binge eating versus placebo, but binge eating reduction and abstinence did not differ at study conclusion. Fluoxetine did not differ significantly from sertraline or fluvoxamine, and fluoxetine and fluvoxamine were significantly inferior to CBT alone in another trial [255].

Other SSRIs mostly fail to separate from placebo. Trials of fluvoxamine and escitalopram reported statistically significant differences in weight loss favoring the SSRI. With a mean BMI of 33–40, these patients had a mean weight loss of 2.7–3.52 pounds, which shows reports of statistically significant differences can be clinically irrelevant [255].

Adding fluoxetine or fluvoxamine to CBT or other behavioral treatments did not enhance outcomes compared with either psychologic therapy alone [255]. However, the pooled results of seven second-generation antidepressant trials (five SSRIs, one duloxetine, and one bupropion) found patients receiving second-generation antidepressants were 1.67 times more likely to achieve binge-eating abstinence than those receiving placebo. Second-generation antidepressants reduced weekly binge-eating episodes by roughly two-thirds and binge-eating days per week by roughly one day (vs. placebo) [80].

Other Antidepressants

Duloxetine, a serotonin-norepinephrine reuptake inhibitor, was studied in 40 patients with binge eating disorder and comorbid depressive disorder. After 12 weeks, duloxetine (mean dose: 78.7 mg) significantly reduced weekly binge-eating days, binge-eating episodes, and severity ratings for binge eating disorder and depressive disorders compared with placebo. However, it did not differ from placebo in BMI, eating pathology, depression, or anxiety [260].

Antiepileptic Medications

In several randomized controlled trials, topiramate was significantly superior to placebo in more rapid reductions in binge eating, binge-eating remission, and weight loss. Binge-eating remissions in two topiramate studies were 64% and 58%. Adding topiramate (vs. placebo) to CBT significantly enhanced binge-eating remission rates and led to significantly and meaningfully greater weight loss [255]. Aside from lisdexamfetamine, topiramate is the only medication to consistently demonstrate substantial weight loss in patients with binge eating disorder [80].

In one randomized, placebo-controlled trial, 18 patients were randomized to 12 weeks of either phentermine/topiramate or placebo. Eating disorder behaviors, mood, and side effects were measured.

Outcome measures included objective binge-eating days/week and binge abstinence. Baseline objective binge-eating days/week decreased from 16.2 to 4.2 with phentermine/topiramate versus 13.2 with placebo. Abstinence rates were 63.6% with phentermine/topiramate versus 9.1% with placebo. Other measured effects with the combination were minimal and similar to placebo [261].

The promising outcomes with topiramate are tempered by high rates of adverse events resulting in study dropout, including sympathetic nervous system arousal, headaches, sleep disturbances, and a collection of other symptoms including rash, high blood pressure, confusion, and taste aversion [80]. Dropout from topiramate is substantially reduced by adding CBT [255].

Other antiepileptics show less robust efficacy than topiramate [255]. Lamotrigine was not superior to placebo, and zonisamide was superior to placebo in more rapid binge-eating reduction and greater weight loss, but binge-eating remissions did not differ [255].

Anti-Obesity Medications

While not a binge eating disorder criterion, obesity is strongly associated with binge eating disorder in epidemiologic studies and is nearly universal in participants in binge eating disorder studies. Orlistat is a peripherally acting drug that blocks fat absorption in the gut without CNS effects. Several trials found orlistat significantly superior to placebo in enhancing weight loss, with no difference in binge eating reductions. Orlistat added to CBT significantly enhanced weight-loss outcomes but not binge eating reductions (compared with placebo). Orlistat or placebo added to behavioral weight loss therapy enhanced weight losses in obese patients without binge eating disorder, but not among obese patients with binge eating disorder [255].

Disulfiram

A small 12-week trial of disulfiram 250 mg/day significantly decreased binge-eating episodes per week (7.9 pretreatment vs. 0.9 post-treatment), and 58.3% achieved binge-eating remission. However, 25% dropped out from side effects, and the remaining subjects reported drowsiness, headache, dysgeusia, tachycardia, dizziness, and nausea [262].

Non-Invasive Brain Stimulation Therapy

A small pilot study evaluated single-session transcranial direct current stimulation of the right dorsolateral PFC compared with sham therapy. Transcranial stimulation decreased craving for sweets, savory proteins, and food overall (with the strongest reductions in men), and significantly reduced desire to binge eat in men the day of active treatment. Greater reductions in craving and food intake correlated with less eating for reward motives and greater intent to restrict calories, respectively. Stimulation of the brain may recruit mechanisms that enhanced cognitive control and/or reduced the need for reward. Transcranial direct current stimulation in binge eating disorder was safe, and the results support evaluation of multi-session therapy [263].

Surgical Interventions

Bariatric surgery is a treatment option for binge eating disorder with severe obesity (BMI ≥40 or BMI ≥35 with comorbid conditions). In the past, an eating disorder diagnosis was a contraindication to bariatric surgery, but this restriction has been relaxed. The disability and difficulty achieving stable weight loss with standard behavioral interventions has increasingly led to surgery candidates with binge eating disorder [240; 264]. Bariatric surgery can be suitable for selected patients with binge eating disorder, but the extent of weight loss depends on post-surgery binge episode reduction, and 20% to 40% fail to lose sufficient weight or regain significant weight post-surgery [265]. Patients may experience emergent post-bariatric surgery psychiatric comorbidities. Considering the demonstrable and durable behavioral and neuroplastic changes associated with addictive eating behavior, obesity treatment is not realistic for some of these patients [266].

Overeaters Anonymous

Overeaters Anonymous (OA) is a 12-step program for compulsive overeaters. Binge eating is viewed as a physical, spiritual, and emotional disorder, and OA proposes recovery through change that promotes physical, spiritual, and emotional well-being. Members have stated the tools for spiritual and emotional work provided in OA were essential to recovery. Secure attachment experiences are very likely to occur within OA, in which safe ground is provided and positive attachment figures are accessible. These factors facilitate a corrective emotional experience that compensates for childhood rejection and time spent with a caregiver who lacked the emotional availability required for the creation of a secure attachment [267].

The contribution of secure attachment figures to binge-eating recovery is consistent with other research that connects emotional overeating in women to attachment-related themes of relationship history, addiction as coping mechanism for insecure attachment, emotional eating as reminiscent of ambivalent attachment, and emotional hunger [268].

COMPULSIVE BUYING DISORDER

Compulsive buying disorder, also termed shopping addiction, pathologic buying, or compulsive buying, is characterized by excessive or poorly controlled preoccupations and urges to spend. The excessive, impulsive, and uncontrollable purchase of products persists despite severe psychologic, social, occupational, or financial consequences [269; 270].

EPIDEMIOLOGY AND RISK FACTORS

The prevalence of compulsive buying disorder in the United States is 5.8% to 8.9% [271; 272]. However, estimating compulsive buying disorder prevalence is difficult because standardized diagnostic criteria are lacking. Current definitions are based on similarities between compulsive buying disorder and substance/behavioral addictions, OCD, and eating disorders [270].

The prevalence of compulsive buying has increased worldwide in the past two last decades. The pooled results of 40 compulsive buying prevalence studies from 16 countries found rates of 4.9% in adults, 8.3% in university students, and 12.3% in shopping-specific samples (e.g., shopping mall visitors). Younger age and female sex were associated with increased risk, but prevalence rates in the United States and other studied countries did not differ [273].

In one study, among 1,441 shopping mall visitors, the rate of compulsive buying disorder was 8.7% [273]. Compared with non-compulsive buyers, compulsive buyers were younger, less educated, more likely female, and more likely to have used licit and illicit substances. They also showed higher levels of impulsivity and obsessive-compulsive symptoms, lower levels of well-being and self-esteem, and greater psychologic distress. Patients identified as compulsive buyers were five times more likely to meet diagnostic criteria for borderline personality disorder. These findings suggest that, among shopping mall visitors, compulsive buyers are prevalent and have high rates of important indicators for psychopathology [273].

COMORBIDITY

Comorbidities associated with compulsive buying disorder include [271; 274]:

- Mood disorders (21% to 100%)
- Personality disorders (60%)
- Anxiety disorders (40%)
- Substance use disorders (24% to 46%)
- Eating disorders (8% to 85%)
- Impulse control disorders (35%)

Patient samples show positive associations between compulsive buying disorder and bulimia nervosa, substance use disorders, gambling disorder, and impulse control disorders (e.g., intermittent explosive disorder). Compulsive buying disorder and ADHD are highly comorbid in both community and clinical samples. Patients with compulsive buying disorder also suffer high comorbidity with depressive and anxiety disorders, OCD, and hoarding disorder [275].

In a comparison of outpatients with compulsive buying disorder, inpatients without compulsive buying disorder, and healthy controls, the patients with and without compulsive buying disorder did not differ in borderline personality disorder symptoms, ADHD symptoms, or self-harming behaviors. Current impulse control disorder diagnosis was three times greater in patients with compulsive buying disorder than those without or control groups, despite patients with compulsive buying disorder perceiving themselves as being no more impulsive than either comparison groups [275].

CLINICAL CHARACTERISTICS

Compulsive buying is a chronic, repetitive behavior that becomes a primary response to negative events and feelings [276]. Four distinct phases in compulsive buying events are described: anticipation, preparation, shopping, and spending. Negative emotions, such as anger, anxiety, boredom, and self-critical thought, were the most common antecedents to shopping binges, with euphoria or relief from negative emotions the most common immediate effects [271].

Unlike ordinary consumers with primary buying motives of value and usefulness, compulsive buyers make purchases to elevate their mood, cope with stress, gain social approval/recognition, and/or improve their self-image [277; 278]. Compulsive buying through the Internet is motivated by immediate gratification, ability to buy unobserved and avoid social interactions, and the vast number of products [22]. Compulsive buying disorder is driven by mood-modifying motivation—not excessive spending or materialism alone. It can lead to serious adverse outcomes, including substantial debt, relationship problems, elevated risks of criminal behavior, and suicide attempts [279]. Emotional consequences from protracted compulsive buying include shame, guilt, and regret/remorse over purchases and failed efforts to stop [213; 270].

The role of impulsivity in compulsive buying disorder is demonstrated, characterized by novelty-seeking, acting rashly without considering future consequences, and approach behavior triggered by high sensitivity to reward stimuli. Other data suggest correlations between compulsive buying disorder and positive/negative urgency (i.e., the tendency to act rashly while in positive/negative mood) [275].

The purchases of compulsive buyers are seldom used, and patient interest is quickly lost. However, the urge to shop and buy recurs, along with excessive, inappropriate buying sprees the patient is unable to control. Patients with compulsive buying disorder usually spend far beyond their means, leading to distress, debt, interference with employment and social functioning, relationship difficulty, and occasionally, criminal behavior such as fraud or embezzlement [275].

Sociodemographic and personality characteristics may help determine different clinical subtypes of compulsive buying disorder to consider during assessment and treatment planning. Three clusters of compulsive buying disorder were identified among patients attending a specialized treatment program [280]:

- Male compulsive buyers: The highest prevalence of comorbid gambling disorder and the lowest levels of reward dependence
- Female, low dysfunctional: Mainly employed women, with the highest level of education, the oldest age of onset, the lowest scores in harm avoidance, and the highest levels of persistence, self-directedness, and cooperativeness
- Female, highly dysfunctional: The youngest age of onset, the highest levels of comorbid psychopathology and harm avoidance, and the lowest scores in self-directedness

Comparisons with Other Behavioral Addictions

A study compared characteristics of patients seeking treatment for compulsive buying disorder, gambling disorder, Internet gaming disorder, Internet addiction, or sexual addiction. The results show that although compulsive buying disorder could likely be related to other addictive behaviors, significant differences in its phenomenology exist. Compulsive

buying disorder is characterized by a higher proportion of women, older age of onset, poorer general psychopathologic state, higher levels of novelty seeking and harm avoidance, and moderate levels of reward dependence, persistence, and cooperativeness. In this sense, patients with compulsive buying disorder could be described as curious, easily bored, impulsive, and active seekers of new stimuli and reward. At the same time, these patients show pessimism and worry in anticipation of upcoming challenges [270].

Clinical differences were lower compared with sex addiction and higher compared with gambling disorder and Internet gaming disorder. Compulsive buying disorder and sexual addiction showed similar profiles, with their psychopathologic symptoms and personality scores being clearly worse than for gambling and Internet gaming disorder. In behavioral addictions, impulsivity is a core feature, but multiple studies also show high levels of compulsivity [281; 282]. This study showed the combination of impulsive/compulsive symptoms is especially prominent in compulsive buying disorder and sex addiction [270]. In hoarding, one of the most commonly reported symptoms is acquiring behavior, and other studies have identified numerous similarities between hoarding and compulsive buying disorder [283].

Gambling disorder and compulsive buying disorder share features, including the urge to achieve immediate gratification or relieve negative emotion through impulsive/compulsive behavior, early onset of problematic addictive behavior, and impaired money management skills [270]. The inability to resist an impulse, drive, or temptation to perform a harmful act is an essential feature of most behavioral addictions [45].

Online compulsive buying disorder was studied in 240 female subjects. The relationship between excitability from shopping and pathologic online buying was partially mediated by expectancies for online shopping (i.e., the expectation that online buying will satisfy urges for excitement, distress relief, or mood elevation). Craving and pathologic online buying were strongly correlated, and increased cue-reactivity (i.e., craving after presentation of online shopping images) was observed solely in individuals scoring high for online compulsive buying disorder. This mediation model explained most of the variance for tendency to develop online compulsive buying disorder [284].

PATHOGENESIS AND PATHOPHYSIOLOGY

A breakthrough in the understanding of the pathogenesis of compulsive buying disorder came from Parkinson disease studies, in which some patients prescribed dopaminergic agents developed compulsive buying behavior. This suggests dopamine plays a role in compulsive buying, similar to substance use disorders. This parallel to substance addiction is supported by the finding that the brain does not distinguish between dopamine release in the nucleus accumbens and ventral tegmental area secondary to drug use or experience (like shopping) [285]. Furthermore, functional neuroimaging studies have shown that shopping activates many of the same brain regions as drugs of abuse, specifically the mesocorticolimbic system and the amygdala [286]. Evidence suggests that behavioral addictions may lead to neuroadaptations similar to those reported with substance use disorder, including a hijacking of the neural circuits responsible for the natural reward pathways. If a drug or experience is hyperstimulating, this neuroadaptation (namely, down-regulation of dopamine receptors) occurs with repeated exposures [272].

Different emotional (e.g., pleasure seeking, escape of negative emotions) and cognitive (e.g., impulsivity, self-regulation, decision-making deficits) mechanisms contribute to the development and maintenance of compulsive buying disorder. A growing number of researchers emphasize that compulsive buying disorder shares several key characteristics with other behavioral addictions, including preoccupation with the behavior, diminished control over the behavior, repeated unsuccessful attempts to cut down or stop the behavior, tolerance, withdrawal, and adverse psychosocial consequences.

Importantly, cue-reactivity and craving has been demonstrated in individuals with compulsive buying disorder [284]. As discussed, Internet characteristics seem to encourage compulsive buying, with greater and more rapid excitation, gratification and reward response, buying opportunity 24 hours per day, buying from the convenience of home, and easy payment that can lead to unplanned overspending [284].

Brain imaging studies of compulsive buying disorder and other behavioral addictions have consistently found frontoparietal region, reward processing, and limbic system abnormalities, also commonly observed in substance use disorders [270]. Neurocognitive data suggest that subjects with compulsive buying disorder have impairments in response inhibition, risk adjustment during decision-making, and spatial working memory. Problems in these distinct cognitive domains support a neurobiologic overlap between compulsive buying and other behavioral and substance addictions [84].

As discussed, Gray's reinforcement sensitivity theory states that a hyper-responsive behavioral system predisposes individuals to engage in impulsive behaviors [287]. It has also been used to explain the addictive processes underlying compulsive buying disorder; both reinforcement and punishment systems seem to participate in the onset and development of this disorder [288]. Dysfunctional emotional regulation is also implied in the phenotype of behavioral addictions, particularly in aspects such as managing cravings and withdrawal symptoms [270].

Research indicates that individuals with high reward sensitivity are motivated to engage in behaviors that provide internal reinforcement (enhancement/winning motives) and external reinforcement (social motives). Similarly, greater compulsive buying tendencies have been positively associated with reward sensitivity, which has been reported to be a powerful predictor of disorder severity [45; 289].

College students meeting the criteria for compulsive buying have shown greater psychiatric comorbidity, increased stress, and worse physical health [45]. Individuals with greater punishment sensitivity are vulnerable to negative emotions (e.g., frustration, anxiety, fear, sadness), and compulsive buying may serve as a form of negative reinforcement [45].

DIAGNOSIS

Compulsive buying disorder is characterized by a maladaptive preoccupation with buying and shopping and by repetitive purchasing of consumer goods to relieve stress, to escape from negative feelings, and to enhance a poor sense of self [275]. These patients engage in persistent, excessive, impulsive, and uncontrollable purchase of products despite severe psychologic, interpersonal, social, occupational, and financial consequences [45].

Compulsive buying disorder was classified in the DSM-III-R as an impulse control disorder not elsewhere specified, but it was omitted from the DSM-5 [11; 290]. Since 2013, research demonstrates that compulsive buying disorder has several features that characterize addictions, including cue reactivity and cravings [279].

Although not recognized in the DSM-5 as a diagnostic entity, the following diagnostic criteria have been proposed for compulsive buying [84; 272; 274]:

- Frequent, maladaptive preoccupation with, or engagement in, buying; intrusive, irresistible, "senseless" buying impulses
- Clearly buying more than needed or can be afforded
- Distress related to buying behavior
- Significant interference with work or social functioning
- The buying behavior does not occur exclusively during hypomanic or manic episodes

The YBOCS-Shopping Version provides a robust, sensitive measure of compulsive buying disorder treatment response and can be used in the clinical screening of compulsive buying disorder [291].

TREATMENT

Psychologic Interventions

Studies suggest that CBT, dialectical behavior therapy, and psychodynamic psychotherapy may have promising results in treating compulsive buying disorder [272]. CBT focuses on identifying factors that maintain and reinforce the problematic buying behavior and delineates strategies for controlling impulsive spending. Maintaining a diary to identify triggers, describe the shopping behavior itself, and record the consequences of compulsive shopping assists patients in understanding behaviors from a different perspective. Additional options include 12-step programs such as Debtors Anonymous, debt consolidation, and credit counseling [272; 292].

Group Psychotherapy

Group psychotherapy that uses a CBT approach or cognitive-behavioral methods within an eclectic approach appears beneficial, with durable improvements in reducing distress associated with compulsive buying disorder and maladaptive buying behavior. Research suggests that impulse control training should be a core component of compulsive buying disorder treatment [279]. Attrition rates show that group psychotherapy is not acceptable to all patients with compulsive buying disorder; patient choice and suitability are important considerations [279].

Stepped Care Delivery Model

Evidence suggests that a low-intensity, guided self-help approach to treating compulsive buying disorder was comparable to high-intensity group therapy. If patients can be treated with effective, brief, and less intensive psychologic interventions first, this may increase service throughput and efficiency [293]. Studies of Internet-based, therapist-assisted self-help programs also usefully mimic the shift of behavior in online shopping [294]. Research indicates that excitability regarding online shopping and compulsive buying disorder are mediated by Internet use expectancies [284]. Treatments clearly need to reflect the context within which compulsive buying occurs [279].

Effect of Reward and Punishment Sensitivity on Treatment Response

Reward and punishment sensitivity was studied for impact on treatment outcome (12 weekly CBT sessions) in female patients with compulsive buying disorder or gambling disorder [45]. In compulsive buying disorder, higher reward sensitivity was related to poorer treatment adherence but reduced risk of dropout. Patients were likely to have stronger intrusive urges to buy, interfering with ability to curb buying behavior and carry out practice homework for CBT. The lower dropout risk may reflect patient motivation by social factors, with patients more likely to form a therapeutic alliance and not abandon treatment. High punishment sensitivity correlated with harm avoidance; these patients had increased risk of treatment drop-out.

Among all patients, high reward and punishment sensitivity was associated with greater psychopathology; worse psychologic adjustment and treatment outcomes; higher novelty-seeking, persistence, and self-transcendence; and low self-directedness and cooperativeness. These results suggest patient response to CBT is conditioned by sensitivity to reward and punishment; compulsive buying disorder and gambling disorder shared this phenotype. This information could be incorporated into targeted interventions to strengthen CBT efficacy in compulsive buying disorder [45].

Pharmacologic Interventions

A review of compulsive buying disorder treatment found few published pharmacotherapy studies. SSRI antidepressants are the most-studied pharmacotherapy, based on the same premise as their use in other behavioral addictions [84]. Evaluations of citalopram, escitalopram, and fluvoxamine have reported mostly negative results, and clinicians should consider psychotherapeutic options before pharmacotherapy [279].

TRICHOTILLOMANIA (HAIR PULLING DISORDER)

Trichotillomania is the repetitive pulling out of one's own hair, leading to hair loss and functional impairment. Although documented in the medical literature since the 19th century, trichotillomania has received little research attention [295]. In clinical settings, the preferred term for this condition is "hair pulling disorder" [296].

EPIDEMIOLOGY

Community prevalence studies suggest that trichotillomania occurs in 0.5% to 2.0% of the population [297; 298]. In adults, trichotillomania predominately affects women (4:1 female to male), but childhood trichotillomania shows equal sex distribution [297; 298]. As a behavior, hair pulling appears quite common and often presents along a continuum from mild to severe. When hair pulling meets the criteria for trichotillomania, interventions should be considered [299].

CLINICAL FEATURES

The typical age of onset for trichotillomania is consistently found to be 10 to 13 years of age. The natural history may vary, but untreated trichotillomania is usually a chronic disorder with fluctuating intensity; studies show an average illness duration of 22 years. Symptom intensity usually waxes and wanes, but persists without treatment [299; 300].

With hair pulling in trichotillomania, the scalp is the most common site (72.8% of patients) followed by the eyebrows (56.4%) and the pubic region (50.7%) [299]. Triggers to pull can be sensory (e.g., physical sensations on the scalp, hair thickness, length, and location), emotional (e.g., feeling anxious, bored, tense, angry), or cognitive (e.g., thoughts about hair and appearance, rigid thinking, cognitive errors). Many patients report not being fully aware of their pulling behaviors at least some of the time—a phenomenon termed "automatic" pulling. "Focused" pulling, in contrast, generally occurs when the patient sees or feels a hair that is "not right" or if the hair feels coarse, irregular, or "out of place" [299; 301].

The inability to stop hair pulling and the resultant alopecia can lead to low self-esteem, psychosocial dysfunction, and social anxiety. Individuals commonly report failing to pursue job advancements or avoiding job interviews because of the pulling and/or their appearance. A low or very low quality of life is reported by close to 33% of adults with trichotillomania [302]. In patients with trichotillomania, greater functional impairment is associated with later symptom onset, lower quality of life, and worse disease severity [303].

Unwanted medical consequences from trichotillomania include skin damage or infection that results from using sharp instruments (e.g., tweezers, needles) to pull hair. More than 20% of patients eat hair after pulling it out (trichophagia); gastrointestinal obstruction and formation of intestinal hairballs (trichobezoars) can ensue, requiring surgical intervention in extreme cases [304].

COMORBIDITY

Patients with trichotillomania have high rates of comorbidity, including major depressive disorder (39% to 65%), anxiety disorders (27% to 32%), and substance use disorders (15% to 19%). Trichotillomania is often misdiagnosed as OCD. Rates of comorbid OCD are significantly higher in clinical (13% to 27%) than community (1% to 3%) populations [299; 305].

The age of trichotillomania onset is generally earlier than its common comorbidities. In a large trichotillomania survey, patients sought to alleviate negative feelings associated with hair pulling through use of tobacco products (17.7%), alcohol (14.1%), or illicit drugs (6.0%). Also, 83% reported anxiety and 70% reported depression due to pulling, indicating that clinicians should screen for trichotillomania and the secondary manifestations of the behavior to better ensure successful treatment outcomes [305; 306].

PATHOPHYSIOLOGY

The similarity between repetitive motor symptoms of hair pulling and repetitive compulsive rituals in OCD led to proposals that both disorders shared common neurobiologic pathways. However, evidence indicates that trichotillomania and OCD

are distinct. In contrast to OCD, patients with trichotillomania are more commonly female, and body-focused repetitive behavior disorders (e.g., skin picking, compulsive nail biting) more frequently occur in these patients and their first-degree relatives. In OCD, compulsions are often driven by intrusive and obsessional thoughts, which are seldom found in patients with trichotillomania and are not listed in diagnostic criteria. The typical age of onset is early adolescence for trichotillomania and late adolescence for OCD. Treatment response also differs, with SSRIs effective in OCD but not trichotillomania [307; 308].

Hair pulling is considered a means of escaping or avoiding aversive experiences, and the temporary relief from negative emotions can maintain the behavior through a cycle of negative reinforcement. Patients with trichotillomania have demonstrated greater difficulty regulating negative affective states than controls. In some individuals, boredom may trigger hair pulling. Some have hypothesized that in a subgroup of patients, hair pulling alleviates negative emotions resulting from perfectionism and an inability to relax, with pulling serving the function of releasing tension [309].

In the few trichotillomania neuroimaging studies conducted, patients with trichotillomania showed volume deficits and disorganization in neurocircuits that mediate affective regulation, motor habit generation, and suppression (compared with healthy controls) [310]. Another study of subjects with trichotillomania failed to identify abnormalities in implicit learning or striatal/hippocampal activation, characteristics of OCD [311].

DIAGNOSIS

Trichotillomania was not officially endorsed as a mental health disorder by the APA until 1987, when it was included in the DSM-III-R as an impulse control disorder not elsewhere classified [290]. Trichotillomania was moved to the chapter on Obsessive-Compulsive and Related Disorders in the DSM-5, alongside OCD, excoriation disorder, body dysmorphic disorder, and hoarding disorder [11]. As discussed, this designation of trichotillomania as an OCD-related disorder can be misleading.

One trichotillomania criterion in the DSM-IV was "an increasing sense of tension prior to hair pulling or when resisting the urge; pleasure, gratification, or relief when pulling out hair" [250]. The DSM-5 omitted this criterion because not all trichotillomania sufferers had this experience [296]. The DSM-5 criteria are [11]:

- Recurrent pulling out of one's hair, resulting in hair loss
- Repeated attempts to decrease or stop hair pulling
- The hair pulling causes clinically significant distress or impairment in social, occupational, or other important areas of functioning
- The hair pulling or hair loss is not attributable to another medical condition (e.g., a dermatologic condition)
- The hair pulling is not better explained by the symptoms of another mental disorder (e.g., attempts to improve a perceived defect or flaw in appearance in body dysmorphic disorder)

TREATMENT

Persons with trichotillomania rarely seek professional mental health treatment, largely due to concerns that providers do not understand the disorder or fears over the reactions of professionals. Others avoid seeking help from social embarrassment, believing their condition is just a bad habit or that it is untreatable. With early diagnosis and appropriate treatment, up to 50% of patients may attain at least short-term symptom reduction [295].

As noted, trichotillomania often co-occurs with anxiety disorders, and persons with trichotillomania frequently report their hair-pulling worsens during increased anxiety. Adult patients with trichotillomania and comorbid anxiety disorders show significantly worse hair pulling symptoms, are likely to have co-occurring depression and first-degree relatives with OCD, and show significantly worse motor inhibition. Male patients with trichotillomania are more likely to have substance use disorder comorbidity, older age, and marriage status than female patients [300; 312].

Psychologic Interventions

The evidence base for psychotherapy in trichotillomania is small, and habit reversal therapy has the greatest empirical support. Habit reversal therapy is a behavioral therapy initially introduced for the treatment of nervous habits and tics. The core components of habit reversal therapy include self-monitoring (e.g., asking the patient to track hair-pulling or skin-picking events), awareness training, competing response training, and stimulus control procedures (e.g., removing hair-pulling or skin-picking cues from the patient's environment) [309].

Habit reversal therapy is generally delivered in weekly 60-minute sessions for 4 to 22 weeks, with more frequent sessions for patients with greater symptom severity. It can be delivered individually, in a group format, or online using a self-help manual [313]. The clinical benefits of trichotillomania symptom reduction have been augmented by adding components of acceptance and commitment therapy or dialectical behavior therapy [314; 315]. Patient gains from habit reversal therapy are usually maintained for three to six months. Many clinicians combine habit reversal therapy and CBT, but published data support habit reversal therapy use as a stand-alone, first-line psychotherapy approach in trichotillomania [295]. One small study reported success combining metacognitive therapy with habit-reversal techniques for treatment of trichotillomania [316].

Pharmacologic Interventions

The classification of trichotillomania as an OCD-spectrum disorder has interfered with identifying effective drug treatment. SSRIs have been repeatedly evaluated in trichotillomania, without benefit [296]. The only antidepressant with partial efficacy in trichotillomania is clomipramine, a tricyclic agent [317].

Clinical experience and results from a single clinical trial indicate that NAC (1,200 mg twice daily) can reduce hair-pulling urges and may be considered as initial pharmacotherapy. Importantly, NAC may require daily adherence for up to nine weeks for benefits to appear. NAC is well-tolerated, with generally mild side effects of abdominal bloating and flatulence [318].

The atypical antipsychotic olanzapine was evaluated over 12 weeks (mean dose: 10.8 mg/day) and showed significant reductions in trichotillomania symptoms compared with placebo [319]. The propensity to cause metabolic syndrome requires that benefits be carefully weighed against this drug's side effect profile [295].

Dronabinol is synthetic delta-9 tetrahydrocannabinol (THC), the primary psychoactive constituent of cannabis. THC binds to cannabinoid receptors (CB); CB1 receptor activation inhibits glutamate overproduction and resultant excitotoxicity implicated in trichotillomania [296]. A 12-week uncontrolled trial of dronabinol (mean dose: 11.6 mg/day) led to marked reductions in trichotillomania symptoms. Side effects of mild sedation were reported, and no significant effects were found on memory, attention, executive function, or psychomotor speed [320]. These promising results are tentative until confirmed by placebo-controlled trials.

EVIDENCE-BASED PRACTICE RECOMMENDATION

A Cochrane Review found that no particular medication class definitively demonstrates efficacy in the treatment of trichotillomania. Preliminary evidence suggests treatment effects of clomipramine, NAC, and olanzapine based on three individual trials, albeit with very small sample sizes.

(https://www.cochranelibrary.com/cdsr/doi/10.1002/14651858.CD007662.pub2/full. Last accessed April 12, 2021.)

Level of Evidence: Meta-Analysis

KLEPTOMANIA (COMPULSIVE STEALING)

Kleptomania is characterized by repetitive stealing behavior and is associated with severe adverse outcomes, including incarceration and increased rates of suicidal behavior [321]. Persons with kleptomania differ from ordinary shoplifters because their stealing is motivated by symptom relief and not personal gain [322].

Persons with kleptomania mostly steal from stores, usually items of little value, and then discard, hoard, secretly return, or give the items away. The urge to steal may be resisted when immediate arrest is likely, but the odds of apprehension are usually not fully considered. The exhilaration, gratification, or relief experienced during the theft is short-lived, followed by feelings of guilt, remorse, or depression [322].

EPIDEMIOLOGY, RISK FACTORS, AND COMORBIDITY

The commonly quoted kleptomania prevalence of 0.6% in the general population was extrapolated from study data of patients with eating disorders [323]. A 2010 college student survey found a lower kleptomania prevalence rate of 0.38% [324]. This latter figure is likely more accurate because it was not derived from a specific subpopulation [323]. Among those arrested for shoplifting, kleptomania rates have ranged from 0% to 8%. This may be falsely low due to incomplete psychiatric evaluation, lack of strict diagnostic criteria for kleptomania, and/or selection bias [322].

In contrast to community prevalence rates, studies of patients with psychiatric disorders suggest prevalence rates of kleptomania sufficient to potentially represent a public health concern. Among 203 inpatients with diverse psychiatric disorders, 7.8% met current and 9.3% met lifetime criteria for a diagnosis for kleptomania. The similar current and lifetime prevalence suggests that untreated kleptomania is chronic. Other studies show increased rates of kleptomania in patients with depression (3.7%), alcohol use disorder (3.8%), and gambling disorder (2.1% to 5%) [322; 325].

Most kleptomania studies report female predominance. However, women are more likely to seek professional help, and the legal system is more likely to send female shoplifters for psychiatric evaluation and male shoplifters to jail [322]. So, the true prevalences in men and women may be more similar than reported. Among 101 adults meeting DSM-IV kleptomania criteria (73.3% female), the mean age of shoplifting onset was 19.4 years, persisting an average 8.2 years before full diagnostic criteria were met [326].

CLINICAL CHARACTERISTICS AND ILLNESS COURSE

The average age for stealing behavior onset is adolescence, with the first professional evaluation in the mid- to late-30s. Women tend to present for evaluation at a younger age than men. The extended length of time between onset and mental health evaluation reinforces the guilt, shame, and secrecy involved in this disorder [322].

Little data are available on the natural history of kleptomania, but three patterns have been described [11]:

- Sporadic: Brief episodes of kleptomanic behavior punctuating long periods of remission
- Episodic: Relatively equal, protracted periods of kleptomanic behavior alternating with remission
- Chronic: Long-term kleptomania with some degree of fluctuation in the behavior

Up to 68.2% of individuals report the value of stolen items increases over time, suggesting tolerance. Most persons with kleptomania unsuccessfully attempt to stop stealing; these failed efforts contribute to feelings of shame and guilt [322].

A study of persons interviewed after apprehension for theft found that shoplifters and kleptomaniacs differed by mean age (27 years vs. 41 years) and male proportion (58% vs. 32.4%). Approximately 20% had not stolen for personal use and had discarded the object. Both groups reported the same level of impulsivity and "a feeling of not being oneself," but

those with kleptomania reported a greater number of thefts than shoplifters, reflecting the compulsive aspect of the disorder [323; 327].

Consequences

One study found 24% of persons with kleptomania had attempted suicide, of which 92% attributed the attempt to kleptomania itself [323]. Suicide attempts were associated with comorbid bipolar disorder, other impulse control disorders, and personality disorders (especially borderline and narcissistic). These syndromes share neurobiologic deficits in executive function that also typify kleptomania. Unlike earlier studies, anxiety, depression, and substance use disorders did not correlate with increased risk of suicide attempt. Data on suicides were not included, and this group may have been under-represented. Legal problems did not correlate with suicide risk, but patients who complete suicide after arrest or conviction have been reported. Other suicide risk factors in patients with kleptomania include shame, poor self-esteem, high levels of impulsivity and disinhibition, and social isolation [323; 328].

Kleptomania is associated with significant legal consequences. In one study, common legal consequences included being arrested (68.3%), arrested but not convicted (36.6%), convicted and incarcerated after conviction (20.8%), and convicted but not incarcerated after conviction (10.9%) [326].

PATHOPHYSIOLOGY

Evidence increasingly supports kleptomania as a behavioral addiction, and kleptomania shares several features with substance use disorders [322; 323]:

- Urges to engage in behaviors with negative consequences
- Mounting tension unless the behavior is completed
- Rapid but temporary urge reduction following the behavior
- Return of the urge
- External and unique behavioral cues
- Secondary conditioning by external and internal cues (dysphoria, boredom)
- A hedonic quality earlier in the natural history

Substance use disorders are highly comorbid in patients with kleptomania (29% to 50%), and patients with kleptomania often have first-degree relatives with substance use disorders. Kleptomania responds to the anti-craving drug naltrexone, an opioid antagonist used in opioid and alcohol use disorders. In contrast, opioid agonists (tramadol, morphine), but not antagonists, have efficacy in OCD [322; 329].

Clinical subtypes of kleptomania have been identified, based on the underlying drive of the behavior [323; 330]:

- Urges often triggered by external cues (around 50% of patients), closely resembles substance use disorders
- Emotional states of feeling bored, lonely, anxious, or depressed (10% of patients) that resembles affective spectrum disorder. However, patients with this subtype describe their behavior driven by a need to "escape" similar to patients with substance use disorder who experience relief from craving.
- A combination of urges and emotion states (40% of patients), with urges to steal occurring during specific emotional states only

DIAGNOSIS

Kleptomania is a secretive disorder with a hidden nature. Patients often present for treatment from legal coercion after being arrested for shoplifting. The diagnosis is often missed in routine psychiatric evaluations, and clinicians do not usually inquire about it. Patients with kleptomania often do not initiate disclosure of the behavior, even when they are already in psychiatric treatment for another disorder, because of shame about their behavior [323]. One study found only 42% of married individuals with kleptomania had told their spouses about the behavior [322].

The 2000 DSM-IV-TR diagnostic category of impulse-control disorders not elsewhere classified was renamed disruptive, impulse-control, and conduct disorders in the DSM-5 and expanded to include oppositional defiant disorder, intermittent explosive disorder, conduct disorder, antisocial personality disorder, pyromania, and kleptomania [11; 250]. Instead of assigning kleptomania to a work group, the DSM-5 Study Group placed it in this ill-fitting diagnostic category. Data that show similarity between kleptomania and conduct disorder or intermittent explosive disorder are virtually non-existent [181].

The DSM-5 diagnostic criteria for kleptomania are [11]:

- Recurrent failure to resist impulses to steal objects that are not needed for personal use or for their monetary value
- Increasing sense of tension immediately before committing the theft
- Pleasure, gratification, or relief at the time of committing the theft
- Stealing not committed to express anger or vengeance and is not in response to a delusion or a hallucination
- Stealing not better explained by conduct disorder, a manic episode, or antisocial personality disorder

Differential Diagnoses

Kleptomania should be distinguished from ordinary theft, whereby stealing is motivated by the value of the object regardless of whether it was planned or impulsive. Shoplifting and ordinary theft are common, but kleptomania is rare. Some individuals feign symptoms of kleptomania to avoid criminal prosecution, and malingering should be ruled out. Stealing in the context of conduct disorder or antisocial personality disorder is a pattern of antisocial behavior; stealing in the context of acute psychiatric or cognitive symptoms should be distinguished from kleptomania [11; 323].

Kleptomania is easy to mis- or overdiagnose because most diagnostic criteria are based on self-report, which is difficult to objectively confirm. Determination that stealing is not committed to express anger or vengeance may require several sessions to understand patient motives for the behavior, as patients are usually secretive and may not be consciously aware of their motive at the time of assessment [331].

TREATMENT

Successful treatment of kleptomania involves educating patients that they have an illness that is likely to become lifelong without treatment [330]. Psychotherapy should anticipate that, in some patients, kleptomania has become a part of their identity and gives them self-esteem and temporary relief. This should not be ignored when helping patients develop healthy coping skills to handle distress and should be discussed throughout the course of treatment [323; 330].

Because kleptomania is a relatively rare psychiatric disorder, few studies have evaluated psychosocial and pharmacologic treatments and empirically validated treatment options remain lacking. Most published treatment suggestions for kleptomania are extrapolated from conceptual models for substance use disorders, OCD-related or affective disorders, or ADHD [323].

Preliminary data from treatment-seeking patients show gender differences in clinical features and comorbid disorders that may reflect biologic and sociocultural factors with implication for prevention and treatment. Women and men with kleptomania both show substantial symptom severity and functional impairment. Women are more likely than men to be married (47.1% vs. 25.9%), experience shoplifting onset at a later age (20.9 years vs. 14 years), steal household items, hoard stolen items, and have a comorbid eating disorder. Men are more likely to steal electronic goods and have another impulse-control disorder [332].

Psychologic Interventions

CBT is tailored for use in the treatment of kleptomania by applying the following strategies [322]:

- Overt sensitization: The patient is instructed to imagine stealing and then to imagine a negative outcome, such as being caught or feeling nauseated or short of breath.
- Aversion therapy: Patients practice aversive breath-holding until mildly painful when experiencing a stealing urge or image.
- Systematic desensitization: The patient achieves a relaxed state through progressive muscle relaxation and is asked to imagine the different steps of a stealing episode while suggesting they can better manage the urge to steal by controlling the anxiety.

CBT or variants of cognitive and behavioral therapies seem promising for kleptomania, but large-scale studies with control groups are lacking. Furthermore, there may be a shortage of clinicians skilled in these therapies for kleptomania [322]. Findings also suggest that patients with kleptomania require longer treatment beyond the standard 6- to 12-session structure of CBT [333].

Pharmacologic Interventions

Memantine is an *N*-methyl-d-aspartate (NMDA)-receptor antagonist with promising results in substance and behavioral addictions. A small, uncontrolled trial in patients with kleptomania reported that after eight weeks of memantine (up to 30 mg/day), 91.7% met criteria for clinical response (defined as ≥35% improvement in symptom severity and clinician rating of improved/very much improved). All kleptomania disease severity scores decreased, and mood, anxiety, disability scores, and impulsivity improved. Memantine was generally well-tolerated, reduced shoplifting urges and behavior, and improved psychosocial functioning [321].

Naltrexone efficacy was evaluated after eight weeks and led to significantly greater reductions in overall kleptomania severity, stealing urges, and stealing behavior compared with placebo. The mean effective naltrexone dose was 116.7 mg/day, and 92% of subjects completed the study [334]. Naltrexone may decrease dopamine transmission in the nucleus accumbens, thus reducing the reward and reinforcement experienced with stealing and urges to steal [334].

The SSRI escitalopram has shown high response rates in uncontrolled trials, but failed to separate from placebo under double-blinded conditions [335]. Although SSRIs have been considered first-line pharmacotherapy in kleptomania, evidence indicates naltrexone is more effective, especially in patients with greater stealing urges. Patients with urges/cravings to steal and/or a family history of substance use disorders should receive a trial of naltrexone. If kleptomanic symptoms appear within the context of general impulsivity and inattentiveness in ADHD, psychostimulants can be considered. Consider antidepressants or mood stabilizers for comorbid affective disorders [323].

Twelve-Step Programs

Shoplifters Anonymous defines itself as a 12-step group for compulsive shoplifters, stealers, thieves, and kleptomaniacs. Groups under the names Compulsive Shoplifters Anonymous (CSA) and Cleptomaniacs and Shoplifters Anonymous (CASA) can also be found. Although there is little research about these groups, recommending them in conjunction with medication and individual treatment is reasonable to consider [323].

INDUCED BEHAVIORAL ADDICTIONS

As noted, impulse control disorders and behavioral addiction can emerge during treatment with dopaminergic agents. Most published cases involve patients with Parkinson disease, who at some point require the dopamine precursor levodopa or dopamine agonists.

PATIENTS WITH PARKINSON DISEASE

Patients with Parkinson disease receiving dopaminergic therapy can develop behavioral side effects that include impulse control disorders. A multicenter study in 2010 found that gambling disorder, compulsive shopping, compulsive sexual behaviors, or binge eating developed in 17% of patients receiving dopaminergic medications. Other behaviors associated with impulse control disorders included compulsive medication use (dopamine dysregulation syndrome) and punding (repetitive non-goal-directed simple or complex behaviors, including hobbyism) [336].

Another study of patients with Parkinson disease identified addictive behaviors in 14% of patients receiving any dopaminergic medication and 17% of patients receiving dopamine agonists. Behaviors included compulsive gambling (5%), compulsive sexual behavior (3.5%), compulsive shopping (6%), and binge-eating disorder (4%). Compulsive behaviors were more common with dopamine agonists than levodopa and other medications (17.1% vs. 6.9%). The prevalence was similar with the two most commonly used dopamine agonists, pramipexole (17.7%) and ropinirole (15.5%) [337].

Underlying susceptibility factors are suggested by the relatively small proportion of patients with Parkinson disease exposed to dopamine agonists who develop impulse control disorders. In these patients, risk factors for impulse control disorder during dopamine agonist therapy include levodopa treatment, age younger than 65 years, being unmarried, high caffeine use, family history of gambling problems, and current cigarette smoking. Other associated factors were functional impairment, depression, anxiety, obsessive-compulsive symptoms, impulsivity, and novelty-seeking. These risk factors show similarity to those reported in substance use disorders and gambling disorder and suggest common neurobiologic substrates [337].

When starting dopamine agonist therapy, patients with Parkinson disease and their family members and carers (as appropriate) should be given oral and written information about the increased risk of developing impulse control disorders (e.g., compulsive gambling, hypersexuality, binge eating, obsessive shopping) when taking dopamine agonist therapy and that these may be concealed by the person affected.

(https://www.nice.org.uk/guidance/ng71. Last accessed April 12, 2021.)

Level of Evidence: Expert Opinion/Consensus Statement

OTHER DISORDERS

Restless legs syndrome is a common sensorimotor disorder treated with levodopa or a dopamine agonist. A case study of patients with restless legs syndrome and new-onset addictive behaviors linked to dopamine agonist therapy found that patients developed pathologic gambling, kleptomania, compulsive shopping, and hypersexuality; other adverse behavioral effects included criminality, suicidality, and marital discord. The behaviors developed over a wide range of levodopa/dopamine agonist exposures and latencies and, in some patients, persisted despite discontinuation. Some of these patients suffered devastating financial, social, and marital consequences, and the authors concluded dopamine agonist use in restless legs syndrome requires careful consideration [338].

OTHER MEDICATIONS

Aripiprazole, an atypical antipsychotic drug, has been linked to treatment-emergent pathologic gambling in eight adult male patients with schizophrenia or bipolar disorder [339]. All had histories of substance use disorders and non-pathologic gambling before taking aripiprazole. Patient histories showed aripiprazole initiation coincided with intensified urges to gamble or loss of control over gambling; discontinuation of aripiprazole coincided with cessation of severe gambling urges or loss of control [339].

PATHOPHYSIOLOGY

When titrated to alleviate dorsal striatal motor neuron loss, dopaminergic agonist therapy may overload intact dopamine circuits in ventral cortico-striatal cognitive and limbic pathways, explaining the development of treatment-emergent addictive behaviors with these medications [340; 341].

Patients with Parkinson disease who develop impulse control disorders show impairments in decisional but not motor impulsivity. Dopaminergic medications are thought to enhance learning from reward (positive feedback) and impair learning from consequences (negative feedback); this relative imbalance presents as impulsivity [337].

A 2014 report addressed the role of dopamine-3 receptor (D3R) agonist medications in impulse control disorders. This report emphasized the significantly greater link between addictive behaviors and dopamine agonists with higher D3R selectivity (pramipexole, ropinirole) compared with dopamine agonists with lower D3R selectivity (aripiprazole) [342]. D3R is implicated in motivation, physiologic and behavioral responses to drug cues, and novelty-seeking behaviors [337].

TREATMENT

In patients with Parkinson disease, behavioral addiction symptoms improve after decreasing or discontinuing dopamine agonists; however, many patients do not tolerate replacement with levodopa for treatment of motor symptoms. Compared with patients without impulse control disorders, patients with Parkinson disease and impulse control disorders are at increased risk for dopamine agonist withdrawal syndrome, characterized by craving, autonomic, and psychiatric symptoms. Efforts to manage behavioral addiction symptoms while maintaining dopamine agonist therapy have been evaluated with amantadine and naltrexone. Research shows mixed or incomplete response. Preclinical studies identified possible efficacy with the atypical antidepressant mirtazapine [337].

Continuous levodopa delivery has improved impulse control disorder symptoms and compulsive medication use. Continuous delivery of apomorphine improved pre-existing impulse control disorders, but novel or additional addictive behaviors may develop. Case reports indicate the antipsychotic agent clozapine is potentially useful [337].

CONCLUSION

The concept of non-substance behavioral addiction has grown in public awareness and clinical acceptance. While published research over the past decade has dramatically increased, confusion surrounds many aspects of this emerging field in behavioral health. The best available evidence has clarified several characteristics of behavioral addictions, including clinical management and how behavioral addictions resemble and differ from impulsivity, compulsivity, and substance use disorders in phenomenology and neural and cognitive features. Neuroimaging and neuropsychologic research have shown changes in brain structure and function associated with dysfunctional cognitive, affective, and behavioral processes in behavioral addictions. These findings are now forming the basis of targeted psychologic and pharmacologic therapies in these patients.

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