Journal of Psychoactive Drugs, 44 (1), 5–17, 2012

ISSN: 0279-1072 print / 2159-9777 online
DOI: 10.1080/02791072.2012.662859

Routledge
Taylor & Francis Group

Behavioral Addictions: An Overview

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Abstract — The legitimacy of nonsubstance addictions has received increased attention from clinicians, researchers and the general population as more and more individuals report symptoms consistent with impairment of impulse control. The clinical presentation of these disorders is varied, as compulsive activities may include: gambling, eating, sex, shopping, use of the Internet or videogames or even exercising, working or falling in love. As such, there is great controversy in diagnosing, treating or even naming these conditions, as many of these behaviors are daily rituals instrumental to our ultimate survival. Historically, the phrase "impulse control disorders" described these conditions but many researchers and clinicians also use the term "behavioral addictions," "process addictions" or "impulsive-compulsive behaviors" to report behavioral pathology. This review summarizes the data of each of these behavioral addictions from epidemiology to neurobiology to treatment options. Research suggests similarities between natural and drug reward processing but clinical evidence supports the utilization of treatment modalities for these behavioral conditions that can sometimes differ from traditional drug treatment.

Keywords — behavioral addictions, binge eating, impulse control disorders, pathological gambling, process addictions, sex addiction

Rapid advances in technology, overstimulation and the subsequent diminishing effort towards emotional growth and awareness are making some individuals more susceptible to "out of control behaviors." The concept of self medicating with substances is well-known, but how about self medicating with behaviors? The use of repetitive actions, initiated by an impulse that can't be stopped, causing an individual to escape, numb, soothe, release tension, lessen anxiety or feel euphoric, may redefine the term addiction to include experience and not just substance.

The word *addiction* can be defined in many ways. Traditionally, the dependence on exogenous drugs of abuse causing neuroadaptation has served as a primary definition. But some would argue that specific behaviors in a vulnerable individual can also lead to an addictive state. Critics, however, report that the inclusion of behavioral addictions may "medicalize" bad behaviors and blur the line of demarcation between an excessive bad behavior and a true addiction. There is a distinct possibility that adding many more disorders to the *Diagnostic and Statistical Manual of Mental Disorders*, *Fourth Edition* (DSM-IV; APA 2000) may effectively dilute pathological behavior and pathologize variants of normative behavior, subsequently increasing the general public's suspicion of the validity of psychiatric disorders. If everyone meets criteria for a disorder, is there really an effective diagnostic system?

There is great debate over how to classify nonsubstance addictions within the diagnostic classification of mental disorders. Many have suggested that the constellation of symptoms and impairments in functioning associated with "behavioral disorders" are simply symptoms of other disorders and do not have enough in common to warrant their own category let alone individual disorder status such as "sex addiction," "compulsive shopping" and "pathological gambling." However, recent findings are shedding new light on the shared attributes of this class of impulse control disorders and forging a better understanding of how they develop.

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Historically, both the construction and development of DSM criteria in the field of psychiatry and the boundaries between normative behavior and disordered or abnormal behavior have been riddled with controversy, with significant research findings ultimately defining the criteria for a disorder.

NEUROBIOLOGICAL MECHANISMS OF ADDICTION

When reviewing the neurobiological correlates of addiction, it is usual to start with the brain reward circuitry. This region is significant for understanding the origins of how addictive related behaviors may emerge. Motivation is an ancient and evolutionarily conserved phenomenon. As a species, the genetic drive for survival requires incentivizing the acquisition of vital resources such as food, water, shelter and sex. In an age where resources were scarce and the availability of these assets was the key to life or death, strongly imprinting the location and availability of resources and mates ensured prosperity.

Over time, the brain has developed mechanisms to reinforce these behaviors; this neural circuit has been defined as the mesolimbic reward system (Di Chiara 1998). The neuropharmacological mechanisms that mediate this circuit appear to involve several different neurotransmitter systems collectively; however the dopaminergic and endogenous opioid systems appear to be the most influential in regulating "rewarding behaviors." Addiction has traditionally been defined as dependence on a drug that can pharmacologically "hijack" reward circuitry mediated by its effect on the brain and body (the neuromimetic effect of drug administration). However, it could be suggested that any stimuli (drug or behavior) that transforms basic drives required for survival (natural rewards like feeding, thirst, reproduction) into actions of craving/seeking behaviors or repetitive out-of-control behaviors may make it plausible that addiction can occur even in the absence of drug taking. Thus, behavioral addictions may share many of the same pathways associated with chemical dependence. A growing theory is that if one can alter neurocircuitry with illicit drugs and pharmacology, then one can alter it with behavior as well (Holden 2001).

In addition to similarities in clinical overlap, the common currency of both drug and behavioral addiction is learning and memory (Hyman 2005). Cravings are triggered by memories, affective states and situations associated with both out-of-control behaviors and drug use (Martin & Petry 2005). Cue-induced behaviors likely evolved along side the pleasure system to provide a memory of both rewarding as well as aversive stimuli. These signals would both help drive behaviors that would benefit us and avoid circumstances that would prove detrimental. In the case of addictions, cues can be so strong that they reinforce particular behavioral patterns despite their negative consequences. Repetitive behavioral patterns help establish and maintain the cue-induced behaviors associated with addiction through neuroadaptation.

Neuroadaptation and neural plasticity are the hallmarks of the adaptive brain. In response to a drug or behavior, neuroadaptations occur in centers of the brain associated with reward, emotion, and decision-making through plasticity changes and relearning, which elicits behavioral reinforcement and habit formation during addiction. Sensitization, a neuroadaptive response, greatly dependent on context and learning, alters neuronal circuitry involved in the normal processes of incentive, motivation and reward, and thus is equally applicable to "out of control drug use" or "non drug" problematic behaviors (Martin & Petry 2005). These types of neurochemical adaptations also occur in areas of the brain critical to higher order decision-making.

The reward circuit is closely tied with the executive function/decision-making centers of the brain, the prefrontal cortex and orbitofrontal gyrus. Studies suggest that impulse control disorders, like addiction, lead to dysregulation of the prefrontal cortex circuitry (Jentsch & Taylor 1999). Impulsivity is often defined as something that has a sense of urgency or lack of premeditation, an act that restricts evaluation and decision-making. Indeed all of these features tend to define the manner in which drug use manifests itself as one transitions into an "addictive state." An important function of this brain region is that it acts as the "brake system" for the brain by sending stop signals to inhibit the execution of distinct behaviors or actions.

Drugs of abuse have been shown to alter glutamate and dopamine functioning in the prefrontal cortex which may compromise its ability to direct inhibitory regulation (Kalivas & O'Brien 2008); the same may be true for behavioral disorders. It is of note that the prefrontal cortex receives and sends projections to reward, memory, emotion, and stress centers of the brain, all regions that play a substantive role in the addiction process. Thus the impulsive aspects of addiction mediated by alterations in the prefrontal cortex appear to alter the brain's behavioral inhibitory system, opening the door for repetitive maladaptive behaviors.

A SHARED APPROACH TO ADDICTION

There has been a trend toward thinking about non-drug addictions as sharing neurobiological mechanisms with substance abuse and dependence (Deadwhyler 2010; Petry 2006; Volkow & Wise 2005). Drugs of abuse are thought to hijack neural circuits that underlie encoding of natural rewards and plasticity in this circuitry. It has been suggested that these changes may be responsible for the behavioral plasticity associated with increased craving and drug seeking seen in addictive states (Kalivas & O'Brien 2008). Evidence of hijacking is seen in several brain regions known to affect executive function, reward

processing and motivation. (Koob & Volkow 2010). It is widely thought that this plasticity underlies the maladaptive changes in behavior associated with addiction (Olsen 2011). In humans, some of these changes include impaired decision-making, anhedonia, craving tolerance, withdrawal and high rates of relapse (Potenza 2006; Bechara 2005).

Similarities between substance and non substance rewards can be seen in imaging studies as well. Functional neuroimaging studies in humans have shown that seeing appetizing food (Wang et al. 2004b), the act of gambling (Breiter et al. 2001), shopping (Knutson et al. 2007) and playing video games (Hoeft et al. 2008) activate similar brain regions, including the mesocorticolimbic system and extended amygdala, as do drugs of abuse (Volkow & Fowler 2000). Looking at a traditional description of "addicted states," we may find substance induced and behavioral conditions both meet criteria. Addictive states are characterized by changed reinforcement contingencies, significant anhedonia, the incapacity to experience day-to-day pleasures due to reduced sensitivity to endogenous brain dopamine, and a striking responsiveness to cues that are both internal to the individual and within the environment associated with the behavior or drug use (Volkow & Fowler 2000; Childress et al. 1999).

These behavioral correlates suggest that nonsubstance addictions share similar neuroadaptations. Further support for this concept comes from studies showing medication-induced increases in nondrug rewards for activities including gambling, shopping or sex in patients taking drugs that activate the dopaminergic system (Evans et al. 2006). Thus it appears that dopamine dysregulation is a common thread in both chemical and behavioral addictions.

In looking at the numbers, epidemiological reports estimate prevalence rates in the United States at 1% to 2% for pathological gambling (Potenza et al. 2003; Welte et al. 2001), 5% to 6% for compulsive shopping, (Black 2007; Koran et al. 2006), 3% to 6% for compulsive sexual behavior (Black 2000), 2.8% for binge eating disorder (Hudson et al. 2007) and .5 to 1% for kleptomania (McElroy et al. 1991). Currently, the impulse control disorders have a small section in the *DSM IV-TR* (intermittent explosive disorder, kleptomania, pyromania, trichotillomania, pathological gambling) with some behaviors simply classified under impulse control disorder NOS. Although the term "addiction" is not utilized in the *DSM-IV*, substance use disorders are categorized according to the substance causing the problems and then grouped by abuse, dependence, withdrawal and intoxication. Within the *DSM-IV*, behavioral addictions have been grouped under categories including: "impulse control disorders not otherwise specified," "eating disorders" and "substance-related disorders," (Potenza 2006; Holden 2001). As understanding of these disorders expands, a better grasp of the etiology, prevalence, and neurobiological underpinnings will likely emerge around these "behavioral addictions."

BINGE EATING

Food is an essential component to every organism on the planet. From single celled bacteria to multicelled organisms such as ourselves, almost every living thing has some means of consuming and metabolizing nutrients to get energy for survival. However, the modern era has ushered in a growing population with an unhealthy relationship to food. Within this population exists a growing subgroup of compulsive eaters whose relationship with food in many ways mimics the criteria currently reserved for addictive disorders. These individuals display both compulsive consumption and preoccupation with certain foods, leading some to categorize them as "food addicts."

Compulsive overeating, also referred to as food addiction, is characterized by an obsessive-compulsive relationship to food. An individual suffering from compulsive overeating disorder engages in frequent episodes of uncontrolled eating, during which they may feel frenzied or out of control, often consuming food past the point of being comfortably full. Unlike individuals with bulimia, compulsive overeaters do not attempt to compensate for their binging with purging behaviors such as fasting, laxative use or vomiting. Compulsive overeaters will typically eat when they are not hungry. Their obsession is demonstrated in that they spend excessive amounts of time and thought devoted to food, and secretly plan or fantasize about eating alone. Binge Eating Disorder (BED) is the most common eating disorder in the United States, affecting 3.5% of females and 2% of males, and is prevalent in up to 30% of those seeking weight loss treatment (Smith et al. 1998). The *DSM-IV* (APA 2000) defines Binge Eating Disorder as a type of eating disorder not otherwise specified, that is characterized by recurrent binge eating without the regular use of compensatory measures to counter the binge eating and a minimum of two binge eating episodes a week for at least six months.

The neurobiological mechanisms underlying the behaviors that result in pathological overeating are multifaceted. "The regulation of food intake is a complex balance between excitatory and inhibitory processes. The excitatory processes arise from the body's needs for nutrients and calories. The inhibitory processes arise from satiety signals after food consumption" (Bassareo & Di Chiara 1999). From an evolutionary standpoint the drive for food acquisition is incredibly powerful for humans and animals. The consumption of food is a vital component of our every day lives. Motivation and cue-induced behaviors directed toward food sources ensured that early man would succeed in the race for survival. However, with the advent of the industrial revolution, resources like food have become more easily accessible to the masses in a manner never before seen. For some, it may be that caloric-based resources strongly activate reward and cue based brain centers in a similar fashion to drugs of abuse.

This compounded with the abundant availability of food to many may prove a downward spiral into an addiction-like disorder: compulsive eating. Indeed, neurobiological studies suggest correlates between the neurocircuitry recruited in substance abuse and compulsive food consumption. It has been shown that palatable foods have the potential to increase neuropeptides associated with regulating the brain's pleasure system (Kelley et al. 2005). Brain imaging studies in humans implicate the involvement of dopamine-modulated circuits in pathological eating behavior (Wang et al. 2004a). Further, food cues increase striatal extracellular dopamine as well as metabolism in the orbitofrontal cortex, a brain region associated with executive functions, suggesting activation of both motivational and decision-making centers of the brain (Wang, Volkow & Thanos 2009).

Just as various drugs promote different degrees of dependence, foods also differ in their capacity to promote abuse (Volkow & Wise 2005). Highly palatable foods such as those high in fats and sugars have been shown to strongly activate mesolimbic dopaminergic circuits within the brain (Sharf, Lee & Ranaldi 2005). Similar to drug-addicted subjects, striatal dopamine D2 receptor availability is reduced in obese subjects, which could explain how food could temporarily compensate for understimulated reward circuits in these individuals. Decreased DA D2 receptors in obese subjects are also associated with decreased metabolism in prefrontal cortical regions involved in inhibitory control, which may underlie their inability to control food intake (Volkow, Wang & Telang 2008); Volkow suggests that, "Dopamine deficiency in obese individuals may perpetuate pathological eating as a means to compensate for decreased activation of these circuits." In conjunction with dopamine, the serotonin system been shown to play a distinctive role in modulating appetitive behaviors (Blundell 1984). Evidence suggests that serotonin is a key regulator of the satiety or "stop eating" signal in the brain (Halford et al. 1998). Serotonergic agonists and reuptake inhibitors have been shown to significantly reduce binge-eating frequency and suppress excess food consumption in human populations (Appolinario & McElroy 2004; Halford & Blundell 2000). Congruently, the serotonergic system appears to play a significant role in several drugs of abuse including cocaine, alcohol, and methamphetamine (Kenna et al. 2009; Filip et al. 2005), indicating that there may be shared pathways between substance abuse and binge eating disorders. Although the *DSM-IV* does not classify food as a substance of abuse, the neurobiological, clinical and behavioral findings suggest that binge eating fits into the framework of addictive disorders.

Treatment options for compulsive eating disorder include pharmacological and behavioral interventions. Randomized controlled trials using cognitive behavioral therapy and brief psychoeducation have led to improved outcomes with binge eating symptoms (Carter et al. 2003). Some success has been seen with antidepressants such as serotonergic reuptake inhibitors such as fluoxetine, fluvoxamine, sertraline and citalopram. Other options that have also shown promise are anticonvulsants like topiramate that modulate voltage-gated ion channels and glutamatergic receptors (Marazziti et al. 2011; Appolinario & McElroy 2004), suggesting a role for these transmitter systems in regulating this behavior. Given the known risks associated with compulsive overeating, such as obesity and increased morbidity and mortality, further investigation is warranted to better understand treatment options and factors that have contributed to this epidemic.

DISORDERED GAMBLING

The Substance Use Disorders Workgroup of the American Psychiatric Association DSM committee has proposed several changes to the current *DSM-IV* classification of pathological gambling. The workgroup has proposed to rename the *pathological gambling* disorder as *disordered gambling* and to reclassify the disorder from the section on impulse control disorders not elsewhere classified to the substance related disorders (which is to be renamed as addiction and related disorders) (Hodgins, Stea & Grant 2011).

The access and availability of gambling opportunity is the highest it has ever been worldwide. Online gaming environments, casinos, destination resorts, sports betting, spread betting, bingo, slot machines, private betting, horse races, card games, and lottery tickets are collectively receiving increased attention from the general public throughout the world. The desire and willingness to wager money or other items of value on randomly established outcomes seems universal. Although most individuals participate in gambling as an enjoyable social activity, a small group of people become too seriously involved in terms of time invested and money wagered and they continue to gamble despite substantial and negative personal, social, family, and financial effects. (Hodgins, Stea & Grant 2011)

Epidemiological research, along with studies in treatment-seeking samples, finds high rates of comorbidity (Petry 2009). In data from the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC) study, pathological gamblers had an increased risk of having a diagnosis of alcohol misuse in their lifetimes by a factor of six and an increased risk of having a substance use disorder by a factor of four compared to nongamblers. Also, rates of manic episodes were eight times higher in pathological gamblers, major depression and dysthymia were three times higher in pathological gamblers and generalized anxiety disorder, panic disorder and specific phobias were each more than three times higher (Petry, Stinson & Grant 2005). Also, most studies of treatment-seeking samples find that individuals with both substance

abuse and disordered gambling have more severe problems than individuals with either disorder alone. (Langenbucher et al. 2001)

The research base on pathological gambling is not substantial but there are comparative studies looking at drug addiction and pathological gambling (PG). From a clinical perspective, gamblers report subjective cravings as powerful as drug abusers, they report "highs" similar to drug highs, they show withdrawal symptoms and autonomic instability when not gambling, and they may throw away everything in their life to gamble.

The behaviors that characterize problematic gambling (chasing losses, preoccupation with gambling, inability to stop) are impulsive in that they are often premature, poorly thought out, risky, and result in deleterious long-term outcomes (Chamberlain & Sahakian 2007). Deficits in aspects of inhibition, working memory, planning, cognitive flexibility and time management or estimation are more common in individuals with pathological gambling problems than healthy volunteers (Hodgins, Stea & Grant 2011). Distorted cognitions in gambling disorders may include: magnification of gambling skill, superstitious beliefs, interpretative biases, temporal telescoping, selective memory, predictive skill, illusions of control over luck, and illusory associations. (Hodgins, Stea & Grant 2011)

Research studies looking at the relationship between gambling and substance use disorders reveal similar performance on personality and neurocognitive assessments of impulsivity, with both groups having high scores on self-reported measures of impulsiveness and sensation seeking (Petry 2001). Both show similar clinical courses and similar clinical characteristics including things like tolerance, withdrawal, craving states and repeated attempts to cut back or quit. Thus there appears to be substantive similarities between the systems and circuits associated with chemical and gambling addictions.

Among those who do seek treatment, Gamblers Anonymous (GA) is the most commonly utilized approach. GA is a 12-Step support group based on the principles of Alcoholics Anonymous (Petry 2009). In many epidemiological studies, an estimated 36% to 46% of pathological gamblers are in recovery (Hodgins, Wynne & Makarchuk 1999). Treatment for pathological gambling and problem gambling is varied and may include: GA, cognitive behavioral therapy, pharmacotherapy, motivational enhancement therapy, family therapy, brief therapy, residential treatment and for some, natural recovery.

Neuroimaging studies reveal decreased activation of the ventro-medial prefrontal cortex (vmPFC) in pathological gambling subjects during presentation of gambling cues (videos), which resembles cocaine addicts watching a cocaine video, with relatively less activation in regions implicated in judgment and motivation (Potenza et al. 2003). This suggests that the decision-making faculties are inhibited in these individuals. Neuroimaging studies in pathological gamblers have indicated diminished ventral striatum, ventromedial prefrontal cortex and ventrolateral prefrontal cortex activity during rewarding events, suggestive of a blunted neurophysiological response to rewards and losses (Reuter et al. 2005). The work of Slutske and colleagues (2000) strongly suggests that pathological gambling is genetically related to substance addictions. Low 5-HIAA levels have been found to correlate with high levels of impulsivity and sensation seeking and have been found in pathological gambling and substance use disorders (Potenza, Kosten & Rounsaville 2001). Baseline decreases in serotonergic tone have been observed in comparison to nongambling controls (Linnoila et al. 1983) and a euphoric "high" in gamblers is seen after administration of $5HT_{2C}$ agonists (Potenza 2008). Also, PG has been shown to lead to elevations in noradrenaline and comparatively elevated heart rates (Potenza 2008).

Currently, there are no FDA-approved medications to treat pathological gamblers. It appears that three types of medications have some efficacy in treating PG: opiate antagonists, mood stabilizers and antidepressants. Results from two double-blind, placebo controlled studies of naltrexone and two multicenter double-blind, placebo-controlled trials of nalmefene suggest efficacy of opioid antagonists in reducing the intensity of urges to gamble, gambling thoughts, and gambling behavior (Hodgins, Stea & Grant 2011). Opiate antagonists have been shown to decrease the craving for gambling in a similar fashion to craving in alcoholics, and elevated rates dopamine in individuals with PG and alterations in the A1 allele of the dopamine D2 receptor gene suggest that the reward associated neurotransmitter systems are playing a significant role in driving the addiction process in this disorder (Goodman 2008; Potenza 2008). The use of paroxetine and other SSRIs, lithium and other mood stabilizers for pathologic gamblers with bipolar symptoms, and the glutamate modulator N-acetyl cysteine have shown some positive effects. Because improvement in glutamatergic tone in the nucleus accumbens has been implicated in reducing the reward-seeking behavior in addictions (Kalivas, Peters & Knackstedt 2006), N-acetyl cysteine has been studied in the treatment of pathological gambling and has had positive effects on urges and gambling behavior (Grant, Kim & Odlaug 2007).

HYPERSEXUAL DISORDER

Sex addiction (also known as compulsive sexual behavior or hypersexual disorder) is a controversial topic in both science and media. There is a lot of press but not much scientific evidence. Sex addiction could be described as a debilitating problem which may include impairment in physical health function, cognition, impulse control, attachment, intimacy and mood or it could simply be a convenient excuse for an individual's indiscretions.

There will always be controversy when any class of behaviors, including sexual behaviors, that are considered

to be intrinsically "normal" are medically "pathologized." (Money 1994) The primary criticism of compulsive sexual behavior or hypersexual disorder is that it may simply be a symptom of an underlying Axis I disorder and not a true disorder itself. In one study of compulsive sexuality, 88% of the sample met diagnostic criteria for an Axis I disorder at the time of the interview and 100% met criteria for an Axis I disorder at some time in their lives, with the most common diagnoses being mood and anxiety disorders (Raymond, Coleman & Miner 2003). Compulsive sexual behavior has been estimated to have a prevalence of between 3% and 6% in the United States (Black 2000). Most individuals with hypersexuality are male but studies that have examined both sexes report a proportion of 8% to 40% female (Kaplan & Krueger 2010).

Sexuality is dependent on many factors, including individual and relationship variables, societal values, cultural mores, and ethnic and religious beliefs. In discussing hypersexuality, these contexts need to be considered (Kaplan & Krueger 2010). The challenge is in defining abnormal and pathological sexual practices. For example, a Swedish study found that simple frequency of sexual activity alone was insufficient to establish pathology; high frequency of sexual behavior with a stable partner was associated with better psychological functioning, whereas solitary or impersonal sexual behavior was associated with psychiatric disorders and psychosocial dysfunction (Langstrom & Hanson 2006)

In defining aberrant sexual behavior, Carnes and Wilson (2002) proposed that sexually addictive behaviors include compulsive masturbation, affairs, use of prostitutes, pornography, cybersex, prostitution, voyeurism, exhibitionism, sexual harassment and sexual offending. Coleman, Raymond and McBean (2003) defined compulsive sexual disorders as compulsive cruising and multiple partners, compulsive fixation on an unattainable partner, compulsive autoeroticism, compulsive use of erotica, compulsive use of the Internet, compulsive multiple love relationships, and compulsive sexuality in a relationship.

Hypersexual Disorder has been proposed as a new psychiatric disorder for consideration in the Sexual Disorders section for DSM-V. Hypersexual Disorder is conceptualized as primarily a nonparaphilic sexual desire disorder with an impulsivity component (Kafka 2010). Proposed diagnostic criteria for Hypersexual Disorder (American Psychiatric Association DSM-5 Development 2010) include:

A. Over a period of at least six months, recurrent and intense sexual fantasies, sexual urges, and sexual behavior in association with four or more of the following five criteria:
   1. Excessive time is consumed by sexual fantasies and urges, and by planning for and engaging in sexual behavior.
   2. Repetitively engaging in these sexual fantasies, urges, and behavior in response to dysphoric mood states (e.g., anxiety, depression, boredom, irritability).
   3. Repetitively engaging in sexual fantasies, urges, and behavior in response to stressful life events.
   4. Repetitive but unsuccessful efforts to control or significantly reduce these sexual fantasies, urges, and behavior.
   5. Repetitively engaging in sexual behavior while disregarding the risk for physical or emotional harm to self or others.

B. There is clinically significant personal distress or impairment in social, occupational or other important areas of functioning associated with the frequency and intensity of these sexual fantasies, urges, and behavior.

C. These sexual fantasies, urges, and behavior are not due to direct physiological effects of exogenous substances (e.g., drugs of abuse or medications) or to Manic Episodes.

D. The person is at least 18 years of age.

Specify if: Masturbation, Pornography, Sexual Behavior With Consenting Adults, Cybersex, Telephone Sex, Strip Clubs, Other.

There is a paucity of literature on brain imaging during conventional or pathological sexual functioning. Research utilizing neuropsychological testing with self-reported behavior has shown a positive correlation between hypersexual behavior and global indices of executive dysfunction including features of impulsivity, cognitive rigidity, poor judgment, and deficits in emotional regulation (Reid et al. 2009). Also, diffusion tensor imaging, psychometric testing and the Go-No-Go procedure revealed higher impulsivity scoring in compulsive sexual behavior patients than controls, with hypersexual patients having higher superior frontal region mean diffusivity than controls (Miner et al. 2009). Patients with hypersexual disorder do report feeling out of control and anxious, with obsessional thinking, mood instability and significant impairment in their daily lives.

Reward circuits such as dopaminergic and endogenous opiate systems have been implicated in the process of sexual behavior in much the same way as substance abuse (Goodman 2008). An interesting piece of evidence around the role of the reward system in these disorders comes out of the Parkinson's field, where treatment with dopamine agonists leads to increased vulnerability to impulse control disorders such as pathological gambling, hypersexuality, compulsive shopping and compulsive eating (Vilas, Pont-Sunyer & Tolosa 2012).

A case study of Internet-based sex addiction involving preoccupation with Internet pornography, extended and frequent masturbation and unprotected sex with cyber contacts revealed interesting diagnostic and treatment-based findings. The patient was initially prescribed an antidepressant (sertraline) with both individual and group therapy and 12-Step work with Sex Addicts Anonymous with little improvement. After the addition of naltrexone (an opiate antagonist), the patient reported significant improvement in his cravings. When the naltrexone was discontinued, the patient's cravings returned and when he was put back on the medication, the urges diminished (Bostwick &

Bucci 2008). Two double-blind, placebo-controlled studies reveal decreased symptoms using medication compared to baseline. The first, by Kruesi and colleagues (1992), compared clomipramine versus desipramine, with a two-week, single-blind placebo lead in. Both drugs decreased paraphilic symptoms. The second study by Wainberg (2006) compared citalopram with a placebo for the treatment of compulsive sexual behaviors in gay and bisexual men. In the study, results included a significant decrease in sexual desire and drive as well as frequency of masturbation and pornography use. Additional treatment includes: cognitive behavioral therapy psychodynamic psychotherapy (exploring family of origin, trauma and underlying factors) and 12-Step groups with a focus on sexual behavior, including Sex and Love Addicts Anonymous, Sex Addicts Anonymous and Sexaholics Anonymous (Kaplan & Krueger 2010).

COMPULSIVE BUYING DISORDER

Like other behavioral addictions, shopping addiction is a controversial idea. Many experts recoil at the idea that excessive spending can constitute an addiction, believing there has to be physical tolerance and withdrawal to be diagnostically classified as such. One of the unifying components of all addictions lies in the reinforcing properties of these behaviors and substances. Although there is variability in the definition of pathological spending, experts define compulsive buying disorder (CBD) as a disorder associated with compulsive thoughts or impulses to purchase unnecessary or large amounts of items despite its negative consequences. The classification of compulsive buying disorder remains unclear; however, McElroy and colleagues (1995) have developed diagnostic criteria for compulsive shopping in research settings, which include: (1) frequent preoccupation with shopping or intrusive, irresistible, "senseless" buying impulses; (2) clearly buying more than is needed or can be afforded; (3) distress related to buying behavior; and (4) significant interference with work or social functioning.

Epidemiological reports suggest that there is a 2% to 8% prevalence of compulsive shopping in the U.S. based on results of a survey in which the Compulsive Buying Scale (CBS) was administered to 292 individuals in Illinois (Claes et al. 2011; Black at el. 2001). The data on gender differences with compulsive buying disorder is mixed; however, some estimate that the gender ratio is nine to one (female to male) (Claes et al. 2011; Black at el. 2001). However, Koran and colleagues (2006) report that compulsive buying disorder is nearly equal in men and women (5.5% and 6.0%), respectively. This finding implies that the gender disparity may be smaller than previous reports suggest and that men may be underrepresented in samples.

Compulsive buying is typically chronic or intermittent, with an age of onset that ranges from 18 to 30 years and a greater proportion of these individuals reporting incomes under $50,000 (Black 2007). Psychiatric comorbidities often include mood disorders (21% to 100%), eating disorders (8% to 85%), substance abuse disorders (24% to 46%) and other impulse control disorders. Furthermore, some studies suggest that nearly 60% of compulsive buyers meet criteria for at least one personality disorder (Black 2007).

Although widespread consumerism has escalated in recent years, compulsive shopping is not a new disorder but rather was identified over a century ago. Kraepelin gave it the name oniomania, which is roughly translated as "buying mania." As such, it has been a long-known phenomenon but only recently suggested to fit into the behavioral addiction spectrum (Brewer & Potenza 2008). Although this concept has historical recognition, there is no clear consensus on the difference between normal shopping, occasional splurges and shopping addiction. Black and colleagues (2001) report that individuals with compulsive buying disorder are preoccupied with shopping and spending and typically spend hours each week engaged in these behaviors. They identified four distinct phases of compulsive buying disorder, including anticipation, preparation, shopping, and spending. Many compulsive buyers describe an escalating level of anxiety that can only be relieved when they engage in the act of spending. Lee and Miltenberger (1997) reported that negative emotions, such as anger, anxiety, boredom and self-critical thoughts, were the most common antecedents to shopping binges, while euphoria or relief of the negative emotions were the most common consequences. They reported that there are several characteristics that compulsive buying shares with other addictions. For instance, shopping addicts become preoccupied with spending, and devote significant time and money to the activity. Similar to drug abuse, shopping addiction is highly ritualized and follows an addictive course where the individual is consumed by thinking and planning the next shopping trip, and engaging in the act of buying itself or returning purchases leads to pleasure and relief of negative feelings. The frequency of pathological shopping episodes can range from once a month to once a day, depending on available funds. Similar to substance abuse, after the act of compulsive shopping, the individual may experience exhaustion or a let down. Once the purchase is complete, it often leads to feelings of guilt, disappointment and shame.

The etiology and mechanisms of action behind compulsive spending are poorly understood; however, new research is shedding light on shared addiction associated circuitry that may mediate this behavior. There is a distinction to be made between window-shopping and compulsive spending; the actual addictive process in this disorder is

driven by the process of spending money. The act of compulsive spending subsequently requires recruitment and possible dysregulation of distinct decision-making circuits in the brain.

The role of opiate, serotonergic and dopaminergic systems have all been suggested in compulsive buying disorder (Mueller et al. 2010), however at present no definitive evidence has strongly linked these systems with it. Although clinical studies suggest that citalopram, a selective serotonin reuptake inhibitor (SSRI), may have some beneficial effects in preventing relapse to compulsive buying disorder patients, use of other SSRIs like fluvoxamine has proven inconclusive (Koran et al. 2006). A key indicator seems to stem from the field of Parkinson's disease, where patients maintained on a dopamine precursor L-DOPA or dopamine agonists tend to have higher rates of compulsive shopping, as well as other behavioral addictions (Djamshidian et al. 2010; Nirenberg & Waters 2006). In fact it has been shown that L-DOPA increased reward learning and risk taking in human imaging data (Pessiglione et al. 2006). This suggests that dopamine may play a distinctive role in driving craving and seeking, reward prediction, and decision-making aspects of behavioral addictions in a similar manner to drugs of abuse (Berridge 2007; Volkow & Wise 2005). As shown in previous sections, these systems play a significant role in regulating emotional affect as well as reward systems in the brain and thus represent key components in the addiction process. Compulsive buying disorder shares behavioral features such as escalation and tolerance, in the form of needing to spend more money in order to receive fulfillment from a shopping binge—both hallmarks of addiction. It is clear that the behavioral traits associated with these maladaptive behaviors share a substantial homology with substance abuse and it stands to reason that similar brain systems are recruited and altered during the etiology of the disorder. However, a more rigorous approach is needed to understand the neurobiological mechanisms underlying compulsive buying disorder.

The social, psychological and biological factors surrounding compulsive spending make it an interesting and complex condition. Additional studies are needed to better understand the etiology, differential diagnosis and treatment of this disorder. There are no published reports describing psychotherapy-focused trials for compulsive buying disorder. However, some preliminary findings suggest that cognitive behavioral therapy and dialectical behavioral therapy may have promising effects. Treatment outcome studies using SSRIs such as citalopram and fluvoxamine also seem to show a therapeutic benefit for individuals with compulsive buying disorder. However, further research is needed to identify the mechanisms that drive this behavior in order to create more efficacious treatment options.

INTERNET ADDICTION DISORDER

There is increasing attention on cyberspace social pathologies, which some would call technical addictions. As with other behavioral addictions, Internet abuse has been a controversial idea and one of the most challenging tasks has been to arrive at a comprehensive definition of the concept. Experts have not been able to come to a consensus on a name, however, there are as many as six different terms associated with Internet addiction, including "Internet Addiction Disorder (IAD)," "Pathological Internet Use," "Excessive Internet Use," and "Compulsive Internet Use" (Widyanto, Griffiths & Brunsden 2011).

Internet addiction is a relatively new concept in psychiatry and not yet recognized by the *DSM-IV*. However, some definitions of compulsive Internet use in the literature have been derived from *DSM-IV* criteria for addiction and impulse control disorder. First introduced by Goldberg (1995) and made popular in Young's (1996) pioneering research, the term Internet addiction disorder (IAD) has been defined as "the compulsive overuse of the Internet and the irritable or moody behavior when deprived of it" (Mitchell 2000). Some prefer a more holistic definition that suggests that an individual's psychological state, which includes both mental and emotional states, as well as scholastic, occupational and social interactions, is impaired by the overuse of the Internet (Beard 2005). Shapira and colleagues (2003) state that in order to diagnose the presence of Internet addiction disorder, individuals must meet the following criteria: (1) the excessive use of the Internet beyond the time allotted and/or irresistible urge to be preoccupied with the Internet; (2) an impairment, distress or poor functioning in social settings caused from a preoccupation with the Internet; and (3) the excessive use of the Internet is not associated exclusively with periods of hypomania or mania and cannot be entirely accounted for by Axis I clinical disorders. Griffiths (2000) believes that technical addictions are a branch of behavioral addictions that satisfy six criteria for addiction: salience, mood modification, tolerance, withdrawal, conflict, and relapse.

The true prevalence of Internet addiction in the U.S. is unknown; however, Young (1998) estimated the figure to be between 5% and 10% of all online users, which is approximately two and five million Internet addicts. Other estimates vary greatly, from as low as 3% reported by Mitchell (2000) and Whang, Lee, and Chang (2003), to as high as 80% in Young's original study (1998). The demographic on who is more likely to be affected by Internet addiction is mixed and not a homogenous group. However, Mafe and Blas (2006) constructed a profile of Internet-dependent users as young, highly educated individuals having a close connection with the Internet. Other researchers have identified Internet addiction-prone individuals as single, males, college students, gays, middle-aged females and the less

educated (Soule, Shell & Kleen 2003). There is mixed data on gender disparities, although, more recent research suggests that that there is no correlation between gender and length of Internet use (Soule, Shell & Kleen 2003). Common psychiatric comorbidities with Internet addiction include depression, bipolar disorder, substance abuse disorder, pathological gambling and sexual compulsions (Morahan-Martin 2005).

After a decade or more of academic research, the etiology and mechanisms of action behind pathological Internet use are not well developed. Research in this area is limited, with few studies using control groups, randomization, or well-validated measures. The reward-deficiency hypothesis suggests that those who achieve less satisfaction from natural rewards turn to substances to seek an enhanced stimulation of reward pathways (Blum et al. 1996). Internet use provides immediate reward and gratification, similar to substance use. Individuals with certain personality attributes such as impulsivity, low self-esteem and introversion have a greater propensity to Internet addiction. Internet use may be used as a compensatory tool for certain deficiencies with social skills and interpersonal relationships. There has been a range of psychological and behavioral theories that have been proposed to explain Internet addiction. Hammersley (1995) has suggested a number of psychological reasons why the Internet is highly reinforcing for some people: (1) it allows correspondence with people who share mutual interests; (2) it puts people in touch with other people who would otherwise never meet; (3) the costs of communicating is low; (4) there is a substantial "puzzle" element to using the Internet, and many people find puzzling tasks reinforcing; (5) people can download software toys, some of which are reinforcing; (6) people can keep in touch with friends with minimal time and financial costs; (7) it gives people feelings of status and modernity, which may bolster self-esteem; (8) it allows people to be taken seriously and listened to; and (9) it allows people to present a "well-managed" persona, which may deviate in significant ways from one's everyday, face-face persona. Others have described a cognitive behavioral model (Davis 2001) where Internet addiction may result when some psychological factor causes an individual to be vulnerable to dependence on new online content, which is followed by obsessive thoughts and then the perception that the Internet is a "friend." This may be reinforced by the decade-long trend of people spending increasingly more time with technology than with humans. There has been a shift away from family and peers to mass media technology as the primary socialization agents.

Treatment strategies for pathological Internet use are under-researched and there is limited published data on effective therapeutic modalities. Young (1999) points to the usefulness of cognitive behavioral therapy for compulsive Internet use. He suggests that catastrophic thinking might contribute to compulsive Internet use in proving a psychological escape mechanism to avoid real or perceived problems. He also hypothesized that those who suffer from negative core beliefs and cognitive distortions may be more drawn to anonymity of the Internet in order to overcome perceived adequacies. Cognitive behavioral therapy and psychoeducation seem to have promising results for the treatment of Internet addiction (Young 2007).

Unfortunately, there are no published controlled trials to evaluate pharmacological interventions. Some experts believe that a similar pattern of cortical arousal exists in pathological gamblers, substance abusers and Internet abusers, and naltrexone may mitigate problematic impulse control behaviors in some individuals (Yellowless & Marks 2007). Research has shown adding naltrexone to a mediation regimen that already includes an SSRI coincided with a decline in symptoms of Internet addiction (Bostwick & Bucci 2008). More research is needed to clarify the mechanism by which naltrexone and SSRIs extinguish addictive behavior.

There is no doubt that the Internet usage among the general population will continue to increase over the next few years. Future studies are needed to examine the quantitative and qualitative effects of Internet abuse, while also investigating treatment differences among the various types of Internet addictions.

VIDEOGAME ADDICTION

Video games have been a part of American culture since the late 1950s, and their prominent role in the lives of American youth has led to increased public scrutiny of the effects and potential harms of video game usage, including the potential of socially maladaptive behaviors such as increased short-term aggressiveness and overuse syndromes (CSAPH Report 2006). In June of 2007, the American Medical Association Council on Science and Public Health considered whether "videogame addiction" could be a disorder.

In the U.S. alone, the sale of video games and related products reportedly grossed between $7 and $10 billion in 2004. Although 70% to 90% of U.S. youth play video games, in 2005 a national survey identified the prototype gamer as a 30-year-old male who averages between 6.8 and 7.6 hours weekly playing video games (ESA 2006, 2005).

Using World Health Organization criteria, a gaming addiction rate of 12% was found by researchers in the United Kingdom who polled 7,000 gamers (Grusser et al. 2007). Research in the United States has estimated that anywhere from a small minority to as much as 10% to 15% of players may be affected (Chak & Leung 2004).

Psychosocial effects of video games are varied. Some studies have found that exposure to video game violence may promote increased aggressive behaviors and decreased prosocial behaviors in social interactions. (Sheese& Graziano 2005; Vastag 2004) Although overuse

can be associated with any type of video game, it is most commonly seen among those using massively multi-player online role-playing games (MMORPG), who represent approximately 9% of gamers (ESA 2005). The MMORPG are very interactive, social and competitive and primarily focused on fantasy. Researchers have attempted to examine the type of individual most likely to be susceptible to such games, and current data suggest these individuals are somewhat marginalized socially, perhaps experiencing high levels of emotional loneliness and/or difficulty with real life social interactions. (Allison et al. 2006) Current theory is that these individuals achieve more control of their social relationships and more success in social relationships in the virtual reality realm than in real relationships (CSAPH Report 2006). Symptoms of time usage and social dysfunction/disruption appear in patterns similar to that of other addictive disorders (Tejeiro et al. 2002). Additionally, dependence-like behaviors are more likely in children who start playing video games at younger ages (Grusser et al. 2007).

Although there are very few research studies looking at imaging or treatment, evidence for striatal dopamine release during video game playing was detected in a positron emission tomography study (Koepp et al. 1998). Areas of research on potential health effects of video games that are receiving increasing attention include attention deficit/hyperactivity disorders (ADHD) and neurology (Chan & Rabinowitz 2006).

OTHER BEHAVIORAL ADDICTIONS

There are many other potential behaviors that may have addictive properties, but there is little published data on these conditions. The terms "love addiction or pathological attachment," "work addiction," "exercise addiction" and others have been discussed. And of course, the current impulse control disorders listed in the *DSM-IV* classification need more data. There is very little research to support any of these "other" conditions that are not currently in the *DSM* being a true disorder, but clinically there are many individuals who report symptoms that warrant further discussion.

CONCLUSION

We live in an overstimulated society and rapid advances in technology and abundant availability to stimuli and resources may play a role in the increased prevalence of behavioral disorders. The use of repetitive actions, initiated by an impulse that can't be stopped, causing an individual to escape, numb, soothe, release tension, lessen anxiety or feel euphoric, may redefine the term addiction to include experience and not just substance. The core feature of these behaviors as well as substance use disorders appears to be impulsivity. Impulse control disorders primarily involve a hedonic quality—sex, gambling and stealing are all associated with a rush or a high (Grant, Brewer & Potenza 2006).

The difficult part of defining impulse control disorders involves comorbidity and the complex relationship between affect and impulsivity. How do you know if the symptoms originate from the proposed primary disorder? Some critics argue that behavioral conditions are simply secondary manifestations of underlying psychiatric illnesses including mood disorders, anxiety disorders, ADHD, personality disorders and other disorders. The repetitive behavior is simply an adaptation or compulsion to avoid discomfort.

As research in nondrug addiction progresses, knowledge gained from the fields of drug addiction, motivation and obsessive-compulsive disorder will contribute to the development of therapeutic strategies for nondrug addictions (Olsen 2011). There is emerging clinical evidence that medications used to treat chemical dependency may be successful in treating nondrug addictions. For example, naltrexone, nalmefine, N-acetyl-cysteine and modafanil have all been reported to reduce craving in pathological gamblers (Grant et al. 2006). Opiate antagonists have also shown promise in the treatment of pathological gambling and compulsive sexual behavior (Grant & Kim 2001) and topirimate has shows some success in reducing binge episodes (McElroy et al. 2007).

Similarities between nondrug and drug addictions include craving, impaired control over the behavior, tolerance, withdrawal and high rates of relapse (Potenza 2006). It makes sense that natural rewards can cause neuroadaptation since learned associations between things such as food or sexual opportunities and the conditions which maximize availability is beneficial from a survival standpoint and is a natural function of the brain (Alcock 2005). In some individuals, this plasticity may contribute to a state of compulsive engagement in behaviors that resembles drug addiction (Olsen 2011). Similar to chemical addictions, there appears to be a transition period between moderate and compulsive use (Grant, Brewer & Potenza 2006). Extensive data suggests that eating, shopping, gambling, playing video games, and spending time on the Internet are behaviors that can develop into compulsive behaviors that are continued despite devastating consequences (Davis & Carter 2009). Clinically, patients may shift from a normative behavioral set point to a pathological one when influenced by comorbidities or environmental stimuli. These addiction and related disorders appear to work on a spectrum.

It is clear there is a substantial amount of overlap between behavioral addictions and substance abuse. Despite this commonality, there haven't been many studies evaluating shared neurobiology, although the research in binge eating and pathological gambling is slowly growing. At a minimum, we need researchers to better define these

conditions with uniform diagnostic criteria and develop universal, valid screening measures. Awareness is building and research is beginning to coalesce around defining the biological systems that drive these types of disorders. The National Institute on Drug Abuse (NIDA 2002), a research-funding agency in the United States, has cited the importance of studying nondrug behaviors/disorders (obesity, pathological gambling, etc.) in understanding substance dependence. Indeed, in gaining a better understanding of behavioral addictions it may prove that we gain a stronger theory of the overall mechanisms that comprise our perception of "addiction."

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