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The Neurobiology of Behavioral Addictions

Sexual Addiction

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Introduction

Addiction has historically been defined from a behavioral perspective, with specific diagnoses described based on a constellation of behaviors related to pathological drug-seeking and -using behavior. Defining addiction based on behavioral considerations alone is no longer a valid or credible option given our growing appreciation for the biological basis for all disease, including mental illness. As stated by the American Society of Addiction Medicine (ASAM) in their definition of addiction, an "understanding of addiction requires understanding of a broader network of neural connections involving forebrain as well as midbrain structures" (1). Clinicians who have been scripted educationally to define addiction based solely on behavioral parameters and those who are not familiar with complex, emerging neuroanatomical and physiological issues related to the brain's reward systems may be dismissive of this wealth of new information, which has provided a fresh perspective on neurobiological bases for addiction and which is changing this paradigm. We review the neurobiological basis for addiction, including natural or process addiction, and then discuss how this relates to our current understanding of sexuality as a natural reward that can become functionally "unmanageable" in an individual's life.

The Idea of Behavioral Addiction

Controversy over the existence and definition of addictive sexuality has been based largely on historical paradigms related to the definition of addiction. This has been further compounded by a dependence on the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*) as an authoritative source for defining all addiction, including considerations relating to the neurobiological etiology of addiction. The *DSM*, however, was never intended to define or even discuss issues related to the neurobiology

of addiction; rather, it has been specifically atheoretical since its third edition, *DSM-III*. Its utility as a field manual is that it is based on direct observation and interview and thus can be used to diagnose and treat mental illness clinically without depending on extensive testing. Because it is also used as a template for provider reimbursement, and by the popular press as a definitional source, it has unfortunately been enlisted to serve in a role for which it was never designed and for which it is receiving growing criticism (2).

Evolution and the Reward System

To understand the premise of sexual addiction, it is necessary to have a basic understanding of the brain's reward system and a conceptualization of the neurological basis for all natural or process addictions. Olds and Milner first identified the reward circuit in 1954. Electrodes were placed in various brain regions of laboratory rats, and the animals were given the ability to self-administer electrical currents. When the electrodes were placed in the area now widely referred to as the reward center, the rats preferred to repeatedly self-administer pleasure-inducing currents at the expense of choosing food and water (3). Research has indicated that all drugs of abuse affect this reward center, neuroanatomically known as the mesolimbic dopamine (DA) pathway (4). Sexual activity activates this brain region (5). This pathway connects the ventral tegmental area to the nucleus accumbens (NAc). These areas are specifically tied to impulsivity, pleasure, reinforcement learning, and reward. The amygdala (positive and negative emotions, emotional memory); hippocampus (processing and retrieval of long-term memories); and frontal cortex (coordinates and determines behavior) also interact with this region. Taken together, these interconnecting areas modulate pleasure, reward, memory, attention, and motivation (6).

The reward center serves an evolutionary purpose, rewarding and thereby encouraging activities necessary for survival (food, sex, etc.). Engagement in survival-based behavior activates the mesolimbic DA pathway (7). Interestingly, as discussed in Chapter 7, stressful stimuli elicit similar effects, underscoring the role of this system in adaptation to highly salient stimuli related to survival. The past decade has yielded multiple theories of addiction, all involving the mesolimbic DA pathway and surrounding brain regions and substrates (8). Dr. Nora Volkow, the director of the National Institute on Drug Abuse (NIDA), described addiction as the process of change from impulsive to compulsive action, based on a transition from positive reinforcement to negative reinforcement. Pursuant to associated changes in brain circuitry, Volkow described this as a three-stage process: (a) binge/intoxication, (b) withdrawal/negative affect, and (c) preoccupation/anticipation.

Three Biological Stages of Addiction

In the first stage, "binge/intoxication," the release of DA in the NAc results in acute positive reinforcement of the behavior that initiated it. According to reinforcement models, this positive reinforcement results in learning associations attached to the

behavior that began the process. The continued release of DA leads to an increase in dynorphin levels, which results in a decrease of the dopaminergic function of the reward center, eventually resulting in decreased reward thresholds (6,9). This is key to increased tolerance, which is discussed further in this chapter.

The second stage, "withdrawal/negative affect," begins after the DA flood has passed. The extended amygdala, an area associated with fear conditioning and pain processing, becomes activated. A negative emotional state ensues, leading to activation of brain stress systems and dysregulation of antistress systems. The inverse of the outcome of the first stage, the second stage leads to a decrease in sensitivity to rewards and an increase in the reward threshold, resulting in a negative reinforcement state that encourages the reinstatement of the addictive behavior. Initially, impulsive behavior becomes compulsive, leading to chronic taking/seeking behaviors (6,9).

Dr. George Koob, director of the National Institute on Alcohol Abuse and Alcoholism (10), expanded these first two stages by superimposing a biological model onto a psychologically based opponent-process model of motivation (11). The opponent-process model of motivation posits emotional experiences as opposing pairs, operating similarly to the positive-to-negative reinforcement transition, wherein "a-processes" reflect positive hedonic effects and "b-processes" reflect negative hedonic effects. Here, a-processes occur first and reflect tolerance, and the b-processes appear after the a-process have completed, reflecting withdrawal. Koob furthered the scope of withdrawal in addiction with his "antireward" theory, which holds that when the brain reward center is activated, there is a corresponding engagement of the brain stress systems to limit the reward response and maintain the homeostatic balance of the reward center. Both body stress systems (in particular the hypothalamic-pituitary-adrenal axis) and brain-based stress systems (in particular the corticotrophin-releasing factor [CRF] system) are activated (see Chapter 7). The aforementioned increased levels of dynorphin further increase CRF, and the activation of these systems is responsible for many of the negative effects associated with the withdrawal stage. Dysregulation also occurs in the brain's antistress system, as marked by decreases in neuropeptide Y (a natural anxiolytic in the brain). When the reward center can no longer be returned to its homeostatic state, the addicted brain enters an "allostatic" state, wherein the reward center has an altered set point, leaving the individual susceptible to relapse and dependence. Koob referred to this process as the "dark side" of addiction (10, p. 559).

A key point to note here is that withdrawal is not exclusively about the physiological effects from any specific substance. Rather, withdrawal is expressed through a negative affect resulting from the process just discussed. Negative emotions such as anxiety, depression, dysphoria, and irritability are indicators of withdrawal in this neurobiological model of addiction (9). This is a major challenge to the claim made by many opponents of the idea of natural or processes addictions, who erroneously state physiological tolerance and withdrawal are hallmarks and requirements of the existence of an addiction.

The third stage, "preoccupation/anticipation," is commonly referred to as craving. The neuroplastic impairments extend beyond the mesocortical DA pathway into other regions of the brain, such as the dorsolateral prefrontal cortex, responsible for key components of cognition and executive function, and the ventromedial prefrontal cortex is responsible for components of inhibition and emotional response. Here, the aforementioned associations and increased salience of learned drug-related cues

intersect with increasing deficiencies in top-down inhibitory control. This creates cravings, leaving the individual vulnerable to reinstatement of the addictive behavior. Cue-induced cravings and stress-induced cravings have been identified as the primary reasons for reinstatement of the addictive behavior (6,9). Multiple neuroimaging studies supported this model for both natural and chemical addictions (12,13), and these impairments are the force behind the "chronic relapsing disorder" component of the medical definition of the term *addiction*.

Genetics and Learning

Researchers found that carriers of the *DRD2-A1* gene have fewer D_2 receptors, leaving them with a propensity to develop alcoholism (14). Researchers later found such individuals to be more likely to have interruptions in the mesolimbic reward system, resulting in a hypodopaminergic state that yields a predisposition to addictive, compulsive, and impulsive behaviors (15). They coined the term *reward deficiency syndrome* to represent a congenital chemical imbalance that leaves people vulnerable to behavioral disorders (15). They found that carriers of the *DRD2-A1* gene have approximately 30%–40% fewer D_2 receptors, and these individuals are overrepresented in cases of alcoholism, drug addiction, obesity, compulsive sexual behavior, compulsive Internet gaming, obsessive texting, pathological gambling, workaholism, and shopaholism (15).

Robinson and Berridge took the learning model one step further via their "incentive salience" or incentive sensitization theory of addiction (16,17). This neural model of pathology theory follows the model of a hypersensitized mesocorticolimbic DA pathway, focused not on pleasure or reward, but rather on the motivational attributions attached to the behavior (18). This model arguably most closely follows the evolutionary purpose of the reward system, wherein "drugs induce a false signal of a fitness benefit, which bypasses higher-order information processing" (19, p. 12). In light of subsequent knowledge regarding the role of dopaminergic systems in reward incentives, they recently revisited their theory. They concluded that "bolstered by the evidence that has accumulated over recent years, we remain confident in concluding 'that at its heart, addiction is a disorder of aberrant incentive motivation due to drug-induced sensitization of neural systems that attribute salience to particular stimuli' (17). While focusing on drug addiction, they acknowledged that because natural rewards also involve dopaminergic reward systems, 'incentive sensitization can also sometimes spill over in animals or humans to other targets, such as food, sex, gambling, etc.' (17). An example they cited is 'dopamine dysregulation syndrome, which not only involves compulsive drug use but also can include pathological gambling, hypersexuality, and food binging' (20). Neurobiology of behavioral sensitization is discussed in detail in Chapter 6; Chapters 1 and 13 address the manner in which cross-sensitization links different types of survival-related and potentially addictive stimuli, with serious clinical consequences.

In the last decade, a growing realization occurred that naturally evolved salience drives in biological systems are inherently motivationally advantageous to the survival of both individual organisms and species. We can now look at salience/desire/craving through a more biologically nuanced lens and see motivational systems subserving pleasure rewards not only as supporting behavioral patterns, but also as a product of genetic transcriptional templates that are programmed to run in response to specific stimuli.

While the incentive sensitization model of addiction had been described as more than just aberrant learning, a more inclusive definition views "neuronal learning" more in terms of dendritic and synaptic plasticity and thus incorporates addiction. In this context, that of reward-motivated plasticity, Kauer and Malenka defined addiction as a "powerful, yet pathological form of learning and memory" (21, p. 844). This perspective of pathological expression of reward transcripts supports the concept of addiction as a disease. It is remarkable that an early, and perhaps the first, reference to the term *addiction* predicted a disease model a century before synaptic science was considered in the context of addiction (22).

Sexual Addiction as a Natural Process Addiction

The Biology of Desire

Neurobiological evidence for natural addiction has grown substantially over the last decade (23). It is based in a growing understanding of how the brain "learns" at the synaptic level. We now better understand how signaling cascades affect synaptic plasticity and are beginning to appreciate how these cascades affect subsequent reward motivation. Whereas we formerly conceptualized "desire" more subjectively, we now understand how DNA transcripts important in craving for natural rewards are related to craving with drug addiction as well (24). The neuroscience of addiction has thus taken a decidedly objective turn with regard to this increased understanding of the biology of desire. It is also intuitive and consistent with an understanding of the evolutionary journey of the neocortex in that, as neuronal aggregates became ever more complex, the mesencephalic impetus to survive powered these increasingly sophisticated telencephalic neural systems with dopaminergic desire. Sexual addiction can only be conceptualized in the context of a phylogenetic understanding of these systems; it is on a platform of both behavior and biology that such an understanding must be built.

A more sophisticated understanding of these signaling cascades important in learning as applied to salience systems has been an important key to understanding how cellular and subcellular mechanisms are altered in both drug and natural addictions. For instance, DeltaFosB, a protein important as an intermediate transcriptional product in a complex signaling cascade important in reward processing, was first found in laboratory models of drug addiction (25). While other members of the cFos family are rapidly mobilized and degraded, DeltaFosB persists for weeks as an intermediary in this cascade in medium spiny neurons in the NAc and may facilitate epigenetic and other changes in gene expression that characterize addiction (26). In addition, DeltaFosB can be selectively overexpressed in viral-mediated transfer models, and these models exhibit phenotypic behaviors consistent with addiction. When DeltaFosB expression is genetically amplified in this manner, for instance, overconsumption of food (27), wheel running (28), and sex (29) ensues. The sexual effect is a supranormal expression of sexual performance (30), while repression of DeltaFosB decreases performance (31), thus confirming a role in physiologic homeostasis as well as the addictive association. Postmortem studies have confirmed that this DeltaFosB-mediated signaling cascade is operative in human drug addiction as well (32). Other changes consistent with neuromodulation that are facilitated by the DeltaFosB-mediated signaling cascade such as

reward-mediated dendritic arborization (33) and synaptic plasticity (34) are invoked by sexual rewards as well. This signal transduction also occurs during more "modern" addictive behaviors, such as pornography abuse and Internet addiction. This process of synaptic plasticity can be noted in the studies conducted by both Voon (35) and Kuhn (36). Both of these studies are consistent with a neuroadaptive role for pornography, when considered in the context of other studies demonstrating gray matter plasticity with learning. This epigenetic process eventually will have a phylogenetic impact. To give the species an evolutionary advantage, the phylogenetic tree strongly conserves these reward transcripts that cause these brain changes.

In addition to these microscopic and metabolic changes, there is a growing understanding of how these processes alter the brain macroscopically. From the early violin studies, other learning mediums have been shown to produce physical alterations in gyral size. The result of learning is seen not only with microcellular changes, such as with arborization, but also with gyral sculpting macroscopically (37). Numerous studies over the last two decades have established the fact that learning physically changes the brain. Such diverse learning templates as music (38,39), juggling (40), taxi driving (41), and intense studying (42) have all been shown to affect morphologic alterations in gyri, and negative neuroplasticity has been seen with disuse (43). This is consistent with Kauer and Malenka's statement, in their paper on synaptic plasticity and addiction, that "addiction represents a pathologic but powerful form of learning and memory" (44, p. 844). It is therefore not surprising to learn that addiction studies correlate with cortical atresia macroscopically. Virtually every study on addiction has demonstrated atrophy of multiple areas of the brain, particularly those associated with frontal volitional control and the reward salience centers. This is true for drug addictions such as to cocaine (45), methamphetamine (46), or opioids (47) and also for behavioral conditions associated with pathologic overconsumption of natural rewards and behaviors such as food (48), sex (49), and Internet addiction and pornography (50–51). Correspondingly, recovery from addiction has been correlated with positive neuroplastic changes as well, such as the return to more normal gyral volumes with recovery from methamphetamine addiction (52) and enlargement of gray matter after mindfulness therapy (53).

More relevant to the discussion of addictive sexuality, however, is the concept that natural or process addictions are manifestations of the same neuroplastic reward system alterations seen with substance addictions. The definition presented by ASAM recognized this continuum by including natural addictions such as to food, sex, and gambling in its definition. This change was informed by shifting paradigms of understanding regarding how the brain processes and assigns salience.

In the last decade, a growing realization that "common molecular pathways" subserve both drug and natural rewards has grown (54). Dopamine receptor alterations previously seen with drug addiction have been with food addiction (55), and reward supersalience seen with drug cues has been seen with food (56,57) and gambling (58). Cross-sensitization with drugs of abuse and natural rewards has been seen with food (59) and sex (60). These understandings have engendered a shift in understanding regarding not only the definition of addiction but also the scope of behaviors that fall under what is now a broader umbrella inclusive of both natural and substance rewards.

The ASAM redefined addiction in 2011 to incorporate this emerging perspective, with addiction meriting the term *disease* (2) in that it represents a pathological alteration of

reward transcripts that have been "usurped" by substances that compete, all too well, with natural rewards to which the individual is evolutionarily acclimated (61). It is the alteration of expression of these transcripts that produces the defect in reward processing, motivational relevance, and memory that characterizes ASAM's new definition.

For example, in their "short definition," ASAM defines addiction as "a primary, chronic disease of brain reward, motivation, memory and related circuitry ... This is reflected in an individual pathologically pursuing reward and/or relief by substance use and other behaviors" (2). They elaborated on the scope of "other behaviors" in their "long definition," specifically including sex as a reward-based addictive behavior: "Addiction also affects neurotransmission and interactions between cortical and hippocampal circuits and brain reward structures, such that the memory of previous exposures to rewards (such as food, sex, alcohol and other drugs) leads to a biological and behavioral response to external cues, in turn triggering craving and/or engagement in addictive behaviors" (2).

Sex and Pornography

Given this more neurologically informed perspective on addiction and learning, including sexual learning, it is important to understand that the unique physical and emotional experience that human orgasm affords, in the context of a dopaminergic reward on par with morphine (61,62), can become a "powerful yet pathological form of learning and memory" (44, p.844) and thus qualify as an addictive behavior. The addictive effect of Internet pornography, for instance, may be in the heighted arousal state provided by the potent triad of novelty, aggression, and role of pornography as a "supranormal stimulus" (a term coined by Nicholas Tinbergen) (63). The endless novelty provided by Internet pornography is driven by demand and enhanced by high-quality digital streaming. Whereas companies like Vivid and Playboy have garnered most of the attention of the popular press, technologically driven enterprises such as Mindgeek (formerly Manwin) are supporting this demand with state-of-the-art Internet technology. This technology provides an endless stream of human sexual acts and body parts, most of it initially free. The user will quickly find, however, that to access the next, and presumably more desirable, content, they will need to pay. This novelty, with an endless variety and combination of body parts and sexual acts, becomes a powerful cornucopia for the sexual salience of the consumer.

Pornography is a perfect laboratory for this kind of novel learning fused with a powerful pleasure incentive drive. The focused searching and clicking, looking for the perfect masturbatory subject, is an exercise in neuroplastic learning. Indeed, it is illustrative of Tinbergen's concept of the supranormal stimulus (63), with plastic surgery–enhanced breasts presented in limitless novelty in humans serving the same purpose as Tinbergen's and Magnus's artificially enhanced female butterfly models; the males of each species prefer the artificial to the naturally evolved (63,64). In this sense, the enhanced novelty provides, metaphorically speaking, a pheromone-like effect in human males, like moths, which is "inhibiting orientation" and "disrupting pre-mating communication between the sexes by permeating the atmosphere" (65).

Doyle and Pazhoohi found that, from an ethological perspective, augmented breasts are consistent with the concept of Tinbergen's supranormal stimulus (66).

Such enhancement does not necessarily provide a net advantage, given the negative psychological and emotional effects of such surgery, including increased rates of suicide in women who have augmented (67). Female genital shaving and cosmetic surgery appear to be tied to a growing expectation that women must conform to a "porn ideal" to compete. As Jillian Lloyd and colleagues noted, "With the conspicuous availability of pornography in everyday life, women and their sexual partners are increasingly exposed to idealized, highly selective images of the female genital anatomy" (68).

As noted, while sex invokes a DA surge rivaling morphine,[69] it is potentiated when fused with aggression. Aggression is also a potent inducer of dopaminergic reward (69), and when sex and aggression occur simultaneously, the reward is compounded. It is pertinent that a recent study examining the content of the most popular renting and selling pornographic movies reported that 88% of the scenes depicted aggression toward women, represented by spanking, gagging, and slapping, with half of the scenes containing verbal aggression, primarily name-calling (70). The overwhelmingly female targets usually showed either pleasure or a neutral response to the aggression.

Aggression is also portrayed by the sexual acts featured in these films. For example, Cowan and Campbell found that 43% of white women and 28% of black women in interracial pornography were portrayed with males ejaculating on their faces (71). Disturbingly, scenes in the recent paper from Bridges and colleagues found that 41% of the scenes examined portrayed the woman performing oral sex on a man who had just performed anal sex, and thus the penis was contaminated with feces (70). They also found that this penetration sequence was a strong predictor of both verbal and physical aggression (70). Therefore, the model described here incorporates the concepts that sexuality can become a process or behavioral addiction, with pornography addiction invoking a powerful neuroplastic response given its limitless novelty combined with the competitive edge it delivers as a supranormal stimulus, especially when fused with aggressive content (72).

Neuroimaging Studies on Pornography Abuse

The first study on the responses of pornography addicts to addiction-related cues was recently completed (35) demonstrating the aforementioned sensitization process. Voon at the University of Cambridge used functional magnetic resonance imaging (fMRI) scans to demonstrate that heavy pornography users have a heightened response to pornography compared to a group of normal volunteers. This increased sensitization to pornography is the same type of response as seen in substance users and Internet addicts (35). The Voon research also strongly supports the addictive model (2). It was a correlative study that, for the first time, demonstrated the classic wanting/liking dissociation. This, of course, is an accepted characteristic of substance addiction. In addition, this study demonstrated fMRI evidence of incentive sensitization, another hallmark of addiction. When considered in the context of our understanding of the ability of sex to invoke a powerful neuromodulatory response, the addiction label is strengthened (35).

Another example is seen in a recent study in *JAMA Psychiatry*, which highlighted both cue-related sensitization and structural atresia in reward areas for chronic Internet pornography abusers (36). As a nonlongitudinal correlative study, it was supportive of at least some causation when considered in light of the fact that virtually all

longitudinal studies to date have demonstrated a structural causative role for learning (38,40,43,47). While no one really argues against a role for a constitutional component with regard to addiction, with up to 50% of addictive tendencies being influenced by genetic traits (73), the *JAMA* article strongly supports a neuroplastic influence regarding the structural changes associated with pornography when this information is interpreted in light of the abundant longitudinal data establishing a role for learning in structurally altering the brain. While the authors acknowledged the limitations of the study by specifying that they could not conclude that these changes were not inherent, and would need longitudinal data in this regard, they also stated that these changes "could reflect change in neural plasticity as a consequence of an intense stimulation of the reward system, together with a lower top-down modulation of prefrontal cortical areas" (36, p. E1).

To categorically assume otherwise, we are brought to the illogical conclusion that, while the learning process associated with a 12-year-old regularly playing the violin alters the brain structurally, a 12-year-old masturbating regularly to hardcore pornography is immune from any such change. Such a perspective is inconsistent with our current neuroscientific understanding of how learning affects structural changes in the brain.

Conceptualizing Sexual Addiction

To develop a conceptualization of addictions that includes biology, plasticity, and neuromodulation, we must consider the overarching concepts of behavioral addictions as addictions to natural rewards that share common nosology, of which sex addiction is just one. In addition, they must include criteria that illustrate the brain's learning and changes experienced with time, such as sensitization, desensitization, tolerance, and withdrawal.

Although the controversy surrounding the nomenclature, conceptualization of the disease, and etiological underpinnings has been intense, the proposed diagnostic criteria for sex addiction have been strikingly similar across perspectives. The proposed criteria for sexual addiction developed by Carnes (74) and listed in *Kaplan and Sadock's Comprehensive Textbook of Psychiatry* (75) are provided in Table 8.1. Many researchers and authors have argued for different terminology, including hypersexuality (76–78) and compulsivity (79); however, a review of the criteria in the table demonstrates consensus across conceptualizations for many of these criteria (authors with similar criteria are listed by each one) (80). However, what differentiates the addiction model from other conceptualizations are the criteria related to tolerance and withdrawal. Only addiction researchers incorporate these ideas involving the neuroplastic brain changes involved in the addiction process.

Opponents of the concept of sex addiction (or even behavioral/natural/process addictions in general) claim the disorder is impossible due to the lack of an exogenous chemical for the body to develop a tolerance to or experience a withdrawal from. This is an argument that fails for two reasons. First, despite common public and professional misconceptions, these elements are not required components of the disease of addiction. The *DSM-IV-TR* states, "Neither tolerance nor withdrawal is necessary

Table 8.1 **Criteria for Sexual Addiction**

Addiction Criteria	*Supporting Literature*
1. Continuation of behavior despite knowledge of having persistent or recurrent social, financial, psychological, or physical problem that is caused or exacerbated by the behavior	Carnes, 1983, 1991, 2005 (74,75,81) Coleman, 2003 (79) Goodman, 1998 (82,83) Kafka, 2010 (76) Orford, 1978 (77) Stein et al. 2001 (78)
2. Preoccupation with the behavior or preparatory activities	Carnes, 2005 (75) Coleman, 2003 (79) Kafka, 2010 (76) Orford, 1978 (77) Stein et al. 2001 (78)
3. Frequent engaging in the behavior when expected to fulfill occupational, domestic, or social obligations	Carnes, 2005 (75) Coleman, 2003 (79) Goodman, 1998 (82,83) Orford, 1978 (77) Stein et al. 2001 (78)
4. Persistent desire or unsuccessful efforts to stop, to reduce, or to control behaviors	Carnes, 1983, 1991, 2005 (74,75,81) Goodman, 1998 (82,83) Orford, 1978 (77) Stein et al. 2001 (78)
5. Recurrent failure (pattern) to resist sexual impulses to engage in specific sexual behavior	Carnes, 1983, 1991, 2005 (74,75,81) Goodman, 1998 (82,83) Kafka, 2010 (76) Orford, 1978 (77)
6. Frequent engaging in those behaviors to a greater extent	Carnes, 1983, 1991, 2005 (74,75,81) Goodman, 1998 (82,83) Kafka, 2010 (75) Orford, 1978 (76)
7. Giving up or limiting social, occupational, or recreational activities because of their behavior	Carnes, 1983, 1991, 2005 (74,75,81) Goodman, 1998 (82,83) Kafka, 2010 (76)
8. Inordinate amount of time spent in obtaining sex, being sexual, or recovering from sexual experiences	Carnes, 2005 (75) Goodman, 1998 (82,83) Kafka, 2010 (76) Orford, 1978 (77)
9. Need to increase the intensity, frequency, number, or risk of behaviors to achieve the desired effect or diminished effect with continued behaviors at the same level of intensity	Carnes, 1990, 2005 (75,84) Goodman, 1998 (82,83)
10. Distress, anxiety, restlessness, or irritability if unable to engage in the behavior	Goodman, 1998 (82,83) Carnes, 2005 (75)

Sources: Carnes PJ. Out of the Shadows. Center City, MN: Hazelden; 1983; and Carnes PJ. Sexual addiction. In: Sadock BJ, Sadock VA, editors. Kaplan & Sadock's Comprehensive Textbook of Psychiatry. Vol. 1, 8th ed. Philadelphia: Lippincott, Williams, & Wilkins; 2005, pp. 1991–2001.

or sufficient for a diagnosis of substance dependence" (85, p. 194). The *DSM-5* states, "Neither tolerance nor withdrawal is necessary for a diagnosis of a substance use disorder" (86, p. 484). Second, both of these components *do* manifest in individuals suffering from the disorder. As shown by the neurobiological models of addiction, the element of tolerance in addiction is the result of a homeostatic adaptation to whatever has been causing chronic levels of DA increase in the reward center, be it drugs or compulsive gambling, video-game playing, or the chronic overuse of sexual behaviors.

As an example of the manifestation of tolerance in sex addiction, many clinicians argue that, in clinical populations, patients report a tolerance to visual stimuli. This suggests that it takes more and more novel and intense content to achieve the same effect. In many of the conceptualizations of the diagnostic criteria for sex addiction, one aspect proposed by many researchers is the idea of escalation (82,83,87). For example, in one study, sexually compulsive behavior was defined as "a propensity to engage in sexually related activities that occur at escalating levels and have potential to result in negative consequences to one's self or others with higher score on measures of sexual compulsivity indicative of one's preoccupation with sex and perceived lack of control over their sexual impulses" (87). In one study (88), researchers made comparisons across four groups: nonsexually compulsive, moderately sexually compulsive, sexually compulsive (SC), and cybersexually compulsive (CSC). They found that the SC and CSC groups were significantly different from the other two groups in "higher sensation seeking" (e.g., the tendency to take part in new or dangerous activities) (88). In another study (89), researchers examined individuals who used the Internet for sexual purposes and found that 15% experienced a gradual increase of online sexual activity. Some might argue that this could be similar to the development of tolerance as seen in substance dependence. In addition, some researchers also believe that deviant pornography use may follow a "Guttman-like progression," which means that a nondeviant pornography user becomes a deviant pornography user over time (89). These researchers surveyed participants to determine if age of onset determined whether use of adult pornography would progress to deviant pornography, specifically bestiality and child pornography, to determine if desensitization occurred. The results indicated that those individuals who engaged in adult pornography at a younger age had a higher rate of transitioning to deviant pornography than those with a later onset, again suggesting a Guttman-like progression (89).

In addition to tolerance, many clinicians similarly reported that their clients experience the process of withdrawal when stopping compulsive sexual activities. Goodman (82) suggested withdrawal consists of "physiologically ... or psychologically described changes upon discontinuation of the sexual behavior" and that "the same (or closely related) sexual behavior is engaged in to relieve or avoid withdrawal symptoms." Carnes suggested that clients experience distress, anxiety, restlessness, or irritability when unable to engage in the behavior (81). These negative affective states reported by Goodman, Carnes, and many clinicians in the field directly fit with the description of withdrawal articulated by NIDA Director Volkow (6), as well as the antireward or dark side of addiction as articulated by Koob (9).

It is clear that the current definition and understanding of addiction has changed with the infusion of knowledge regarding how the brain learns and desires. Whereas sexual addiction was formerly defined based solely on behavioral criteria, it is now seen also through the lens of neuromodulation. Those who will not or cannot understand

these concepts may continue to cling to a more neurologically naïve perspective, but for those who are able to comprehend the behavior in the context of the biology, this new paradigm provides an integrative and functional definition of sexual addiction that informs both the scientist and the clinician.

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