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Pornography addiction – a supranormal stimulus considered in the context of neuroplasticity

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Addiction has been a divisive term when applied to various compulsive sexual behaviors (CSBs), including obsessive use of pornography. Despite a growing acceptance of the existence of natural or process addictions based on an increased understanding of the function of the mesolimbic dopaminergic reward systems, there has been a reticence to label CSBs as potentially addictive. While pathological gambling (PG) and obesity have received greater attention in functional and behavioral studies, evidence increasingly supports the description of CSBs as an addiction. This evidence is multifaceted and is based on an evolving understanding of the role of the neuronal receptor in addiction-related neuroplasticity, supported by the historical behavioral perspective. This addictive effect may be amplified by the accelerated novelty and the 'supranormal stimulus' (a phrase coined by Nikolaas Tinbergen) factor afforded by Internet pornography.

Keywords: *brain*; *addiction*; *pornography*; *neuroplasticity*; *sexuality*

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Much of the consternation regarding whether compulsive sexual behavior (CSB) is an addiction or some milder malady likely relates to how we define the term itself. It is evident that the word 'addiction' has been reluctantly utilized in mental health nomenclature; one need look no further than the *Diagnostic and Statistical Manual of Mental Disorders* (DSM) for evidence of this. In past versions, addictive behavior was more diffusely described in various sections; the DSM-5 has changed this and has added a classification using the word addiction.

The DSM manuals have historically been atheoretical, that is, based on behavioral observation and interview rather than focusing on biological etiology. The practical significance is that the DSM can thus function as a manual for clinicians in the field; they can diagnose and treat mental illness, including addictive behavior, based on observation and interview, rather than by relying on diagnostic scans and laboratory results.

To understand why the word addiction has met resistance in this context, it is useful to consider its historical meaning in the lexicon. An early, and possibly the first, recorded use of the word addiction in a medical context was a statement in the *Journal of the American Medical Association* in 1906: 'It matters little whether one speaks of the opium habit, the opium disease, or the opium addiction' (Jelliffe, 1906). While few now dispute using the word in relation to exogenously consumed substances of abuse, there has hitherto been a reticence regarding its application to what are now referred to as endogenous, process, or natural addictions.

In 1983, Patrick Carnes introduced the term 'sexual addiction' based on behavioral parameters (Carnes, 1983). Others have supported a behavioral model for sexual addiction; consider, for instance, the recent paper by Garcia and Thibaut, which stated, 'The phenomenology of excessive nonparaphilic sexual disorder favors its conceptualization as an addictive behavior, rather than an obsessive-compulsive, or an impulse control disorder' (Garcia & Thibaut, 2010).

Angres and Bettanardi-Angres (2008) defined addiction as 'the continued use of mood-altering addicting substances or behaviors (e.g. gambling, CSBs) despite adverse consequences', and Bostwick and Bucci (2008) have used the addition label in the context of Internet pornography. There is a growing tendency to apply the term sexual addiction to CSBs, with a realization that sexual motivation is complex, with affective, motivational, and cognitive factors affecting expression of the biologic drive to reproduce. For instance, Estellon and Mouras (2012) described a progressive convergence of psychoanalytic and neuroscientific perspectives as applied to sexual addiction.

Addiction neurobiologists increasingly support the concept of the existence of natural addictions, as functional and cellular evidence continues to accumulate.

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This model is based on a motivational platform emanating from a robustly conserved mesolimbic reward system, with a dopamine-mediated salience drive projecting from the midbrain to other systems essential to survival. This process enables and enhances neuronal learning through micro- and macro-neuroplastic change. Addiction is no longer defined simply by behavioral criteria.

Human consumptive behavior regarding food and sex is more complex than a simple stimulus–response reflex. Georgiadis (2012) stated that human sexuality demonstrates 'clear involvement of high end cerebral cortical areas, possibly hinting at high level "human functions", like perspective taking'. Executive input from frontal regions can modulate the mesencephalic dopaminergic reward impetus projecting to the nucleus accumbens–ventral striatal reward region. Nevertheless, the powerful drives to eat and to procreate are successfully expressed in species that survive, and lines that do not reproduce with net-positive fertility rates, for whatever reason, become extinct. Regardless of how higher cortical function colors sex with other recreational nuances, evolutionary procreative pressures eventually trump purely recreational motives in biologically successful species, including humans.

The evidence supporting the concept of natural addiction is multithreaded, with the behavioral thread being only one component of the growing tapestry of supporting research. Functional imaging studies, correlated with behavior, are of obvious interest, but metabolic and genetic factors are becoming more relevant. It was over a decade ago that realization began to increase regarding the existence of process addictions (Holden, 2001). This awareness has engendered a maturation in understanding the role of the mesolimbic dopaminergic reward pathways in both drug and natural addictions (Nestler, 2005, 2008), a process that culminated in the American Society of Addiction Medicine's (ASAM) definition in August 2011 (known as the ASAM long definition). The new ASAM definition describes addiction as a chronic disease of the brain that affects the reward, motivation, and memory systems, and combines both substance and behavioral addiction under a common umbrella.

The addition of a sub-section on behavioral addiction in the DSM-5 is also recognition of this change of perspective on natural addiction. However, this subsection includes only one process addiction, pathological gambling (PG) (Reuter et al., 2005), while relegating Internet gaming disorder, overconsumtion of food and sex, and other process addictions to a section titled 'Conditions for Further Study,' or ignoring them completely. While it is consistent with recent behavioral and functional data that PG is now recognized as more closely modeling substance abuse rather than obsessive–compulsive disorders (El-Guebaly, Mudry, Zohar, Tavares, & Potenza, 2011), thus meriting the addiction label, it is inconsistent to deny the same label to Internet pornography addiction. It is precisely this inconsistency that supports the premise that cultural and political biases tend to minimize addictive sexual behavior.

It is surprising that food addiction would not be included as a behavioral addiction, despite studies demonstrating dopaminergic receptor downregulation in obesity (Wang et al., 2001), with reversibility seen with dieting and normalization of body mass index (BMI) (Steele et al., 2010). The concept of a 'supranormal stimulus', invoking Nikolaas Tinbergen's term (Tinbergen, 1951), has recently been described in the context of intense sweetness surpassing cocaine reward, which also supports the premise of food addiction (Lenoir, Serre, Laurine, & Ahmed, 2007). Tinbergen originally found that birds, butterflies, and other animals could be duped into preferring artificial substitutes designed specifically to appear more attractive than the animal's normal eggs and mates. There is, of course, a lack of comparable functional and behavioral work in the study of human sexual addiction, as compared to gambling and food addictions, but it can be argued that each of these behaviors can involve supranormal stimuli. Deirdre Barrett (2010) has included pornography as an example of a supranormal stimulus.

Support for the existence of process addictions, though, has increased with our understanding of synaptic and dendritic plasticity. Is there evidence supporting the existence of pornography addiction? It depends on what one accepts, or can understand, as evidence, and this is a function of perspective and education. Perspective can introduce bias, and our perspectives are influenced by factors such as our personal educational and life experiences. What may be meaningless to one may be definitive proof to another depending on differences in knowledge that is esoteric to the field in question. As T. S. Eliot said, 'Where is the knowledge we have lost in information?' (T. S. Eliot, Choruses from *The Rock*, opening stanza, 1934).

Information, or data, becomes knowledge as it is organized into theory and as theory is coalesced into belief systems, or paradigms. Kuhn (1962/2012) noted that when established paradigms are challenged by anomalies, scientists tend to defend the status quo until it becomes apparent that emerging evidence and theory have rendered the status quo obsolete, thus precipitating a paradigm shift. Paradigm shifts are not painless, as Galileo, Ignaz Semmelweis, and others who challenged prevailing dogma learned.

Addiction's initial paradigm was defined solely based on behavioral criteria. What Kuhn would term a paradigmatic 'crisis' has emerged with neuroscience developing what is essentially a parallel – and, obviously to the

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strict behavioralists, a competing – paradigm with the introduction of the concept of behavioral (process) addictions. From the neuroscience vantage point, these are indeed parallel, and even contiguous, paradigms, as former diagnostic criteria defining substance addiction appear to some (Garcia & Thibaut, 2010) to dovetail with those defining behavioral addictions.

The crisis exists in the strictly behavioral paradigm, particularly with regard to labeling CSBs as addictive. For instance, a paper supporting the concept of natural addiction, specifically focusing on pornography (Hilton & Watts, 2011), argued that both micro- and macro-neuroplasticity substantiate the existence of such addictions. A response (Reid, Carpenter, & Fong, 2011) countered that the studies cited supporting macroscopic neuroplasticity in addictive behaviors, being correlative, have no bearing on causation with regard to addiction. Focusing on any changes likely relating more to metabolic effects (high blood sugar, high lipid levels, and so on), this response is dismissive of a neuroplastic effect relating to learning. Skeptical of any natural addiction causing morphologic changes, they discount evidence corroborating the existence of food or exercise addiction, and specifically the inference that these behaviors could affect morphological changes in the brain. Interestingly, they admit that they are more accepting 'of a causal mechanism ... when substances are involved', thus demonstrating the resistance that Kuhn predicted to changes in the old paradigm that substances alone can cause true addictions. This gap between the behavioral and biologic paradigms is further demonstrated in their assessment of the importance of molecular biology in the addiction debate. Strict behavioralists minimize the relevance of DeltaFosB, for instance, to addiction, and opine that DeltaFosB cannot inform the pornography debate because there are no studies in humans specifically investigating DeltaFosB in the context of pornography.

In discussing their perspective, Reid et al. cite their own work and avoid identifying sexuality as potentially addictive. They see problematic consumptive behaviors, whether to cocaine, food, alcohol, or sex, as separate disorders (as per the DSM) and therefore resist any generalization as being 'speculative not scientific' (Reid et al., 2011). This stance is not surprising when considered in the context of the paradigm in which they were trained, which has focused more on behavior rather than on integrating emerging biological evidence as well. The reader is encouraged to study the commentary on the Reid response by Hilton and Watts immediately following and contiguous with the response. That a separate neuroscience addiction paradigm has emerged has provoked a Kuhnian crisis, as these views merge into a new and cohesive biological–behavioral paradigm defining addictions both to substances and to behaviors.

Another summary of the arguments against the concept of addictive sexuality is found in *The Myth of Sex Addiction* by David Ley. The book also describes CSBs from a behavioral vantage point, with neurobiological evidence informing debate on the existence of natural addiction being dismissed with the previously referenced quote from the Reid response to the Hilton-Watts editorial: 'speculative not scientific'.

Interestingly, the brain is seen by Ley as a 'complex, multidetermined "black box" that we are just barely beginning to understand ... complex behaviors such as sex promise to be a riddle for many long years to come' (Ley, 2012). Again, this paradigmatic gap is seen in the veiling of neuroscience with a veneer of mystery and 'riddle', and a promise that we will not be able to understand sexual neuroscience for many years; certainly not now!

Rather than focusing on whether the addictive behavior involves injecting drugs or viewing highly arousing sexual images, an increased knowledge of cellular mechanisms allows us to understand that addiction involves and alters biology at the synaptic level, which then affects subsequent behavior. Addiction neuroscience is now as much about neuronal receptor reactivity, modulation, and subsequent plasticity as it is about destructive and repetitive behavior.

Some demand a higher standard of proof for sex than for other behaviors and substances when it comes to defining addiction. For instance, a strictly behavioralist perspective was illustrated in declaring that for pornography to be labeled addictive, we would have to prospectively addict one cohort of children, protect another, functionally scan both cohorts before and after, and compare behavioral outcomes (Clark-Flory, 2012). Obviously, this study cannot be conducted, given the ethical issues involved. Yet, we presume that even those supporting this behavioral perspective would accept the premise that tobacco is addictive without demanding the same prospective, child-based study. In other words, where is the comparative prospective study with tobacco in children? The one that divides the children, gives half cigarettes, protects the others, and follows them longitudinally? It does not exist, of course, and never will, and therefore some will still say that smoking is not addictive. So said the seven tobacco executives in front of Henry Waxman's subcommittee on Health and the Environment in 1994: in succession, each said 'No' when asked if smoking was addictive, included supporting expert testimony (UCSF Tobacco Control Archives, 1994). Yet based on an extensive body of research, virtually everyone – excluding these tobacco executives and their experts – believes that evidence exists for tobacco's addictive properties. For that matter, where are the prospective child-based cocaine, heroin, and alcohol studies?

The main difference is that we now understand learning-mediated neuroplasticity and neuronal receptor reactivity, including nicotinic acetylcholine, opioid, glutamate, and dopamine receptors, much better than we did in the past. We can now see addiction, whether to smoking, cocaine, or sex, through the lens of the neural receptor and subsequent neuroplastic change, and not solely from a behavioral perspective.

To accept the evidence supporting the concept of sexual addiction, it is necessary to have an understanding of the current concepts of cellular learning and plasticity. Dendritic arborization and other cellular changes precede gyral sculpting (Zatorre, Field, & Johansen-Berg, 2012) with learning, and reward-based learning is no different. Addiction thus becomes a powerful form of learning, with the associated neuroplasticity being detrimental (Kauer & Malenka, 2007). Addiction-related learning is merely an extension of reward-based learning in this model, and it therefore involves similar transcription factors and neurotransmitters. For instance, DeltaFosB was found over a decade ago to be chronically elevated specifically in the medium spiny neurons of the nucleus accumbens in the brains of drug-addicted laboratory animals (Kelz et al., 1999). Subsequent studies have shown it to be elevated in these same cells in animals manifesting pathologic overconsumption of natural rewards, including food and sex (Nestler, 2005).

Supraphysiologic levels of DeltaFosB appear to portend hyperconsumptive states of natural addiction (Nestler, 2008). That DeltaFosB is not only a marker but also a facilitator of hyperconsumptive behavior (as a neuroplasticity enabler) has been well demonstrated. Two closely related mechanisms have been used to genetically manipulate DeltaFosB independent of behavioral variables. One involves producing lines of bitransgenic mice that selectively overexpress DeltaFosB specifically in the striatal reward areas, and the second involves the transfer of genes through adeno-associated viral vectors into adult animals, which then induce over- or underexpression of DeltaFosB. These genetically altered animals exhibit addictive hyperconsumptive behavior involving food (Olausson et al., 2006), wheel running (Werme et al., 2002), and sex (Wallace et al., 2008). For instance, when overexpression of DeltaFosB was imposed through these viral vectors in laboratory animals, they exhibited a supraphysiologic enhancement of sexual performance (Hedges, Chakravarty, Nestler, Meisel, 2009; Wallace et al., 2008). Conversely, repression of DeltaFosB decreases performance (Pitchers et al., 2010), thus confirming that it has a role in normal physiologic homeostasis.

It now appears that DeltaFosB is a molecular transcription switch that turns on other gene sets, which then mediate neuroplastic change in these neurons; in other words, they promote neuronal learning. DeltaFosB increases dendritic spine density in medium spiny neurons in the nucleus accumbens in addicted animals during extended periods of abstinence through stimulation of the protein Cdk5, thus becoming a bridge to more extended neuroplasticity (Bibb et al., 2001; Norrholm et al., 2003). DeltaFosB has been shown to function in a positive feedback loop with Calcium/Calmodulin-Dependent Protein Kinase II to effect neuroplastic cellular responses in cocaine addiction. Significantly, this association was also demonstrated, for the first time, in human cocaine addiction (Robison et al., 2013).

Recent evidence has demonstrated that DeltaFosB is critical to this dendritic plasticity through its effect on the mesolimbic reward system in both sexual and drug rewards, an effect that is mediated by the D1 dopamine receptor in the nucleus accumbens (Pitchers et al., 2013). Dopamine is critical in assigning salience to sexual cues (Berridge & Robinson, 1998), and recent studies support a physiologic role in sexual function as well through its effect on and interaction with the hypothalamic oxytocinergic systems (Baskerville, Allard, Wayman, & Douglas., 2009; Succu et al., 2007). This influence has been broadly conserved across phyla (Kleitz-Nelson, Dominguez, & Ball, 2010; Kleitz-Nelson, Dominguez, Cornil, & Ball, 2010, Pfaus, 2010), ensuring that sex, which is essential to species survival, remains salient. Hypersexuality as a consequence of dopaminergic pharmacologic intervention is a known morbidity of such treatment, and it is related to 'exaggerated cue-triggered incentive salience-based motivation' (Politis et al., 2013). Addiction, of course, can be described as disordered salience. Instead of wanting that which will enhance survival, the addicted are motivated to want even when it is clearly harmful, a neuroplastic process that recalibrates the hedonistic set point.

We see this neuroplasticity at the cellular level through dendritic arborization and other cellular changes that provide a neuroplastic 'scaffolding' of sorts for new synapses to form. Severe craving states associated with subsequent satiation have produced these micromorphologic changes, as demonstrated by such diverse depletion–repletion models as cocaine (Robinson & Kolb, 1999), amphetamine (Li, Kolb, & Robinson, 2003), salt (Roitman, Na, Anderson, Jones, & Berstein, 2002), and sex (Pitchers, Balfour et al., 2012). Salt depletion–repletion craving models have been shown to robustly mobilize the same gene sets activated by cocaine models, and this mobilization is attenuated by dopamine antagonists, suggesting that drug addiction usurps ancient incentive pathways that are essential to survival (Liedtke et al., 2011).

Glutamate receptor trafficking is indicative of synaptic plasticity. Sex, as a powerful brain reward, has shown evidence of increasing silent synapses, which manifest as an increase in the NMDA–AMPA receptor ratio, a

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harbinger of subsequent synaptic plasticity and learning as these synapses are subsequently unsilenced, similar to what occurs with cocaine use (Pitchers, Schmid et al., 2012). Specifically, this ratio change was immediate and long-lasting, and it was found in nucleus accumbens neurons afferent to the prefrontal cortex, an area that is important in mediating CSBs (Pitchers, Schmid et al., 2012). In this, sex is unique among natural rewards, in that food reward did not cause this same persistent change in synaptic plasticity (Chen et al., 2008). Critically, neuroplastic changes in both dendritic morphology and glutamate receptor trafficking were correlated with increased sexual experience and increased amphetamine sensitivity, another hallmark of addiction. Even after 28 days, when these changes receded, the sex-induced hypersensitivity to amphetamine persisted (Pitchers et al., 2013), further strengthening the evidence for natural addiction.

Neuroplasticity as a result of learning is seen not only with microcellular changes, such as with arborization, but also macroscopically with gyral sculpting (Zatorre et al., 2012). Numerous studies over the last two decades have established the fact that learning physically changes the brain. Such diverse learning templates as music (Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995; Schwenkreis et al., 2007), juggling (Draganski et al., 2004), taxi driving (Maguire, Woollett, & Spiers, 2006), and intense studying (Draganski et al., 2006) have all been shown to affect morphologic alterations in gyri, and negative neuroplasticity has been seen with disuse (Coq & Xerri, 1999).

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' (Kauer & Malenka, 2007). 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 (Franklin et al., 2002), methamphetamine (Thompson et al., 2004), and opioids (Lyoo et al., 2005), and also for behavioral conditions associated with pathologic overconsumption of natural rewards and behaviors such as food (Pannacciulli et al., 2006), sex (Schiffer et al., 2007), and Internet addiction (Yuan, Quin, Lui, & Tian, 2011; Zhou et al., 2011).

Recovery from addiction has been correlated with positive neuroplastic changes as well, such as a return to more normal gyral volumes with recovery from methamphetamine addiction (Kim et al., 2006), and enlargement of gray matter after mindfulness therapy (Hölzel et al., 2011). This reversibility is supportive of causation despite the correlative intent of the study designs of these papers, as has been demonstrated in the learning plasticity studies previously cited.

Our brains naturally seek novelty, and sexuality can condition a powerful reward with novelty. Primitive organisms exhibit trophic behavior conducive to survival, and evidence exists of dopamine-related survival incentive in chordate ancestors. Dopamine-powered motivation projected in early amniotes from the primitive mesencephalon to the progressively complex telencephalon throughout the course of phylogeny (Yamamoto & Vernier, 2011). Obviously, human sexual drive and subsequent volitional motivation and reward procurement are much more complex (Georgiadis, 2012) than unicellular trophism, but the more primitive mesolimbic dopaminergic salience centers share these basic drives.

'Hypersexual syndrome', while descriptive behaviorally, falls short of the term 'sexual addiction' in describing the current state of understanding of CSBs. It ignores two decades of research about how learning changes the brain both micro- and macroscopically, and it does a disservice to both professionals and the public in inconsistently exempting the most powerful natural dopaminergic reward in the nervous system, sexual orgasm (Georgiadis, 2006), from neuroplastic learning.

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' (Tinbergen, 1951), 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 (Magnus, 1958; Tinbergen, 1951). 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' (Gaston, Shorey, & Saario, 1967).

Consider hypothetically two individuals, frantically fixated to their computers, both trying to win an intermittently reinforced reward. Both spend hours a night at their task, and have for some period, to the point of exhaustion. Work and personal relationships are affected negatively, yet they cannot stop. One is looking at pornography, searching for just the right clip for sexual consummation; the other is engrossed in an online poker game. One reward is masturbatory, and the monetary, yet the DSM-5 classifies only the poker as an addiction. This is both behaviorally and biologically inconsistent.

Even public opinion seems to be trying to describe this biologic phenomenon, as in this statement from Naomi

Wolf; 'For the first time in human history, the images' power and allure have supplanted that of real naked women. Today real naked women are just bad porn' (Wolf, 2003). Just as Tinbergen's and Magnus's 'butterfly porn' successfully competed for male attention at the expense of real females (Magnus, 1958; Tinbergen, 1951), we see this same process occurring in humans.

Even if pornography can become addictive, the question remains for some, can it be harmful? The content of the most popular pornography currently consumed does appear to overwhelmingly portray aggression toward women (Bridges, Wosnitzer, Scharrer, Chyng, & Liberman, 2010), and, in homosexual pornography, men (Kendall, 2007). The Hald meta-analysis supports the premise that pornography does indeed increase attitudes of aggression toward women (Hald, Malamuth, & Yuen, 2010), as does the paper from Foubert and colleagues (Foubert, Brosi, & Bannon, 2011). The Hald report concludes, 'In contrast to the earlier meta-analysis, the current results showed an overall significant positive association between pornography use and attitudes supporting violence against women in nonexperimental studies' (Hald et al., 2010). Consistent with this pattern of aggression in pornography, the Bridges et al.'s (2010) study found that a representative sample of scenes from the top 250 selling and renting pornographic films from 2004 to 2005 revealed that 41% of the scenes depicted rectal followed by oral penetration, thus exposing the woman not only to a misogynistic and demeaning role, but to potentially pathogenic coliform bacteria as well (Bridges et al., 2010).

This information has negative implications, in that the vast majority of college-aged males, and a growing number of females, use pornography regularly (Carroll et al., 2008). Indeed, pornography has passed from toleration and acceptance to preference, with many universities now hosting and sponsoring 'sex weeks'. Having dismissed any reticence to pornography as a Victorian moralistic, value-laden infringement on First Amendment rights, any objections to pornography are not taken seriously. Thus, potential harms to an individual's mental and emotional well-being are never discussed.

Since these young people, through the brain's mirror systems, 'resonate with the motivational state of individuals depicted' in these films (Mouras et al., 2008), the aggression increasingly inherent in pornography may portend negative emotional, cultural, and demographic effects. These issues warrant greater respect for the power of natural addictions, which can, as their substance counterparts do, 'change the stamp of nature' (William Shakespeare, *Hamlet*, Act 3, Scene 4). Sex, like drug rewards, places its stamp on neuronal receptors, dendrites, and gyri as it facilitates neuroplastic change, thus meriting the addiction label when compulsively and destructively expressed.

Paradigm shifts are usually best viewed historically, after those who cling to outdated paradigms have become irrelevant. During the shifts, crisis and tension predominate, clouding the significance of the shift in the present. Nevertheless, the new combined paradigm that amalgamates addictions to both substances and behaviors is beginning to assert itself, as seen in the new ASAM definition. The DSM's monopoly on defining all the parameters of mental illness, including whether or not biological considerations may contribute, is dissolving as a result of inconsistencies in the latest edition. It is not surprising that Thomas Insel, director of the National Institute of Mental Health, has lamented this continued deficiency in the DSM in stating, "A diagnostic approach based on the biology as well as the symptoms must not be constrained by the current DSM categories…" (April 29, 2013, http://www.nimh.nih.gov/about/director/2013/transforming-diagnosis.shtml). The dismissal of a biological contribution to mental illness through the DSM's silence and continued atheoretical stance is actually accentuating and accelerating the realization that a new combined paradigm is emerging. This is illustrated in the recent *Scientific American* article decrying the DSM's 'fundamental flaw: it says nothing about the biological underpinnings of mental disorders' (Jabr, 2013). As Bruce Cuthbert stated, 'We understand so much more about the brain than we used to. We are really in the middle of a big shift' (Jabr, 2013). Indeed, it is a paradigm shift, and as understanding of the power of the supranormal stimulus in the context of neuroplastic change continues to emerge, the contrast will be ever clearer.

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