

Letter to the Editor

Neuroscience research fails to support claims that excessive pornography consumption causes brain damage

Rory C. Reid, Bruce N. Carpenter¹, Timothy W. FongDepartment of Psychiatry and Biobehavioral Sciences, University of California, Los Angeles, ¹Department of Psychology, Brigham Young University, USAE-mail: *Rory C. Reid - rreid@mednet.ucla.edu; Bruce N. Carpenter - bruce_carpenter@byu.edu; Timothy W. Fong - TFong@mednet.ucla.edu

*Corresponding author

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Dear Sir,

In their editorial on excessive pornography use, Hilton and Watts^[3] offer some interesting neuroscience perspectives on their conceptualization of pornography problems as an *addictive* disorder. They highlight several parallels between dysregulated pornography consumption and other maladaptive behaviors, some of which are viewed as addictions. Although we believe these parallels are worthy of scientific inquiry, Hilton and Watts offered little, if any, convincing evidence to support their perspectives. Instead, excessive liberties and misleading interpretations of neuroscience research are used to assert that excessive pornography consumption causes brain damage. We wish to clarify what the research actually does suggest with several accompanying illustrations.

First, Hilton and Watts assert a “postulate” that “all addictions create, in addition to chemical changes in the brain, anatomical and pathological changes” which they state results in cerebral dysfunction. Depending on how *addiction* is defined, this is either well supported (e.g., brain atrophy arising from the neurotoxicity of alcohol) or wholly speculative as in the case of pornography consumption. A number of studies are cited in support of their position but the interpretation of the findings requires us to assume that cortical atrophy due to some type of excess (cocaine, obesity, or pedophilia) is universal and similarly distributed, and therefore the *type* of excess is irrelevant. Many of the studies cited merely compare groups on brain density scans in cross-sectional designs and inferences about causality cannot be made. For example, their citation of a 2007 study of pedophilia^[11] that used correlational data is reported as evidence that “sexual compulsion can cause physical, anatomic change

in the brain.” Even if such atrophy could be shown in relation to excessive pornography consumption, how much atrophy would actually be necessary before it would functionally impair (e.g., brain damage severe enough to cause behavioral dysfunction) a given individual? The notion that cerebral atrophy assessed through imaging is assumed to be synonymous with brain damage and therefore evidence of an addictive process is a perspective fraught with problems. For example, it is well established that cerebral atrophy occurs progressively as part of normal aging and if such a correlation is considered to be evidence of an addictive process then all of us are “addicted” to growing old. Illustrating a related concern, the imaging study of Miner and colleagues^[5] cited by Hilton and Watts does little to support neuroscientific perspectives on “pornography addiction” given that the majority of the sexually compulsive patient sample had a history of alcohol abuse or dependence and no provisions were made to control for patients with adult ADHD. As a result, it is difficult to determine whether cortical differences and performance on measures of impulsivity in the study were related to hypersexuality, substance misuse, or other pathology already known to be associated with frontal deficits and executive control. Most importantly, the Miner study did not report that any

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of the subjects had problems specifically with excessive pornography use. Collectively, references to neuroimaging studies by Hilton and Watts are unresponsive of their assertion that excessive pornography consumption parallels other maladaptive behavioral patterns such as substance-related disorders or causes significant atrophy in the brain leading to behavioral dysfunction. Even the authors of these studies refrain from drawing such inferences. For example, Franklin *et al*, state "... this study cannot address the etiology of the structural abnormalities. The observed differences may be related to preexisting dysfunction, either environmentally or genetically determined, or a result of the effects of chronic cocaine assault."^[2]

Hilton and Watts seem intent on skewing findings from the studies they cite to support their perspectives rather than evaluating several plausible explanations for the various results reported by study investigators. For example, several explanations exist for the finding of lower density frontal matter in the 2006 study^[6] on obese subjects including dysregulation of insulin or leptin resistance often found in obese individuals. It is also notable that even if the lower density in prefrontal matter of obese subjects, compared to healthy lean controls, was actually a result of atrophy (which this study was not designed to demonstrate), should it be interpreted as evidence demonstrating "visible damage in a natural endogenous addiction" as Hilton and Watts assert? They ignore the possibility that the grey matter differences and any possible frontal neurodegeneration could have predated obesity in the subjects or been an influence of the genetic or biological precipitating risk factors. Indeed, the most parsimonious explanation of the data cited is that frontal deficits may be a risk factor, i.e., preexisting and leading to the poor decision making and excessive indulgence characteristic of each clinical condition. This appears to be a preferred explanation of Schiffer *et al*,^[11] who—contrary to Hilton and Watts' interpretation—hypothesize that early neurodevelopment leads to the brain differences, which serve as a risk factor for the pedophiles they studied.

We are open to the notion that frontal impairment might make people vulnerable to a variety of over-indulgences, which can subsequently lead to substance dependence, maladaptive coping patterns, poor judgment, impulsivity or emotional disturbance, which people may seek to escape by turning to problematic behaviors, such as the case with many pathological gamblers. However, given the lack of studies designed to infer causality, we find it difficult to readily assume the converse — that these diverse dysfunctional behaviors lead to common frontal dysregulation or any cortical atrophy worthy of mention. Admittedly, a causal mechanism strikes us as more likely when substances are involved (e.g., cocaine, high blood sugar, or high lipid levels damaging brain cells), but such

causation is speculative for non-substance activities such as pornography use despite that likelihood that the sexual response cycle activated by pornography consumption also activates endogenous neurochemical reactions in the brain. If we consider that most people eat several times a day, are Hilton and Watson suggesting that the somewhat elevated activity of "eating behavior" is sufficiently different in obese persons to cause brain pathology? Similarly, would they argue that a "runner's high" from extensive exercise leads to brain damage? The parameters of what constitutes pattern, excess, cognitive reward, and the like need to be more clearly explicated and then studied within pornography users.

We are in agreement with Hilton and Watts that the study of executive deficits and frontostriatal systems in patients with dysregulated pornography use or hypersexual behavior is worthy of investigation. Using the proposed DSM-5 criteria for Hypersexual Disorder (HD), our research team has conducted two such studies that have yielded mixed findings. In one study, using neuropsychological self-report measures in a sample of hypersexual men (including those with excessive pornography problems), we found some evidence that executive deficits may exist in this population.^[9] However, when actual performance was assessed on neuropsychological tests sensitive to frontal deficits common in executive dysfunction, no differences were found between hypersexual patients and healthy controls.^[8] We interpreted these findings to support our theory that hypersexuality, including excessive pornography use, is a context specific phenomena which is expressed when triggered by a sexual cue or another stimuli, that when activated, is paired with sexual behavior (e.g., a learned behavior arising in response to dysphoric mood or stress such as been proposed in the current DSM-5 criteria for HD). Regardless, the current literature on excessive pornography use and hypersexuality diverge in many regards from those found in studies among patients seeking help for addictive disorders such as chemical dependency or among patients with impulse control problems such as pathological gamblers. Furthermore, our research on psychological profiles of hypersexual men, including those with pornography problems, failed to find evidence of elevations on addiction indices, but instead found characteristics common in populations with obsessive tendencies.^[7] These findings suggest that hypersexual patients with pornography problems may represent a distinct population and grouping these patterns of behavior with other addictive disorders constitutes a premature conclusion that lacks empirical support.

Hilton and Watts perspectives on pornographic activation of dopaminergic transmission in mesolimbic pathways of the nucleus accumbens, prefrontal cortex, and other brain regions associated with the pleasure reward system does

not offer meaningful insights given the variety of activities that engage this system. Watching the NCAA basketball play-offs will likely lead to similar neurochemical processes for many individuals. Some of us may even experience negative consequences in relation to viewing the play-offs and we may be willing to sacrifice important tasks in exchange for TV time. A few may even feel unable to resist the urge to view information online about the play-offs while at work despite possible violations of corporate policies about appropriate Internet use in the workplace. Are we to conclude that such patterns of behavior constitute an addictive disorder, given their potential relationship to activating dopaminergic transmission in the mesolimbic pathways? Alternatively, we prefer to clarify that substantial evidence suggests that dopamine release in these regions is not associated with a reward mechanism *per se*, but rather, it is part of an arousal process that alerts the brain to the presence of new or novel stimuli in the internal or external environment and such stimuli is not always associated with potential rewards.^[10] Subsequently, any release of dopamine in these brain regions in response to pornography exposure may very well be due to the novelty of the pornographic stimuli and would likely occur for individuals naïve to erotic content as well as seasoned consumers of such material. Regardless, it does not provide readers with any evidence that excessive pornography use is an addictive disorder.

It was unclear to us, and perhaps some of your readers, why Hilton and Watts elected to reference literature about increased Δ FosB in the nucleus accumbens in copulating laboratory rats. These hypersexual rats were engaged in relational sexual activities with female partners, not in autoeroticism in response to sexually-provocative stimuli. Although the rodent study is interesting, we dispute the notion that it is analogous to humans excessively masturbating to pornography and thus the generalizations of the results cited by Hilton and Watts are questionable. Furthermore, the degree of Δ FosB induction in the nucleus accumbens in response to the natural rewards (e.g., sex) was significantly less than that observed in studies of drug rewards suggesting possible differences, not similarities, between drug addiction and sexual activity. Additionally, the significance of Δ FosB in the accumbens appeared to be limited in its effects where sexually naïve rats required fewer intromissions for ejaculation. Notably, cellular changes associated with increased Δ FosB are also found in cells exposed to a wide variety of stimuli unrelated to pleasure or reward behaviors. For example, stressors, sensory stimuli involved in learning, and evoked memory have been associated with such changes.^[4] Given the fact that there are no human studies on Δ FosB in patients with excessive pornography problems and generalizing research from animal studies in order to provide evidence of biological

parallels between addictive disorders and pornography problems is once again, speculative not scientific.

A final concern related to the perspectives of Hilton and Watts is the lack of clarity about what is meant by the term *addiction*. Our research team, along with others, have reported elsewhere^[7-10] on various aspects of hypersexuality and excessive pornography consumption that diverge from commonly held ideas regarding persons addicted to substances.^[11] The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)^[11] completely avoids the term, instead referring to Substance-Related Disorders. By design, problematic behavioral patterns are treated elsewhere in the DSM. In the forthcoming DSM-5, a new category of HD is under consideration for possible inclusion and our research team is currently conducting an independent DSM-5 field trial of the proposed HD criteria to determine the validity of the construct the whether it can reliably be diagnosed. Hilton and Watts statement on this matter may be somewhat misleading to your readers. They state that the forthcoming DSM-5 “contains in this new addition the diagnosis of HD, which includes problematic, compulsive pornography use.” As a point of clarification, the decision whether or not to include HD as a disorder has not yet been made, but it is worth noting that the definitions under study intentionally do not include allusions to addictions, compulsions, or obsessions. Thus, although for some it is common to speak of pornography addiction or other sexual addiction, the lack of convergence of findings has led a growing number to take a more modest and careful position, wherein the connections to other classes of excessive behavior patterns are still under study. Further, delineation of what constitutes an addiction has no agreed-upon standard. Thus, it becomes particularly problematic that Hilton and Watts made no effort to clarify what definition they use and why the term as they use it applies to the participants in the studies they cite such as references to obese subjects as having a “natural endogenous addiction,” even though subjects were screened to be free of psychiatric disorders, including eating disorders.

Despite our criticism of their work, we are encouraged that Hilton and Watts have made an attempt to bring increased awareness to patients exhibiting problems with excessive pornography consumption. We agree, and have published findings demonstrating, that such patterns of behavior have been associated with numerous negative consequences including attachment ruptures in romantic relationships, loss of employment, and psychological distress. Yet much remains to be learned about patients seeking help for hypersexual behavior and excessive pornography problems. Neuroscience has the potential to offer meaningful contributions to our understanding of this phenomenon but such research is lacking at this time. The tone and content of the Hilton

and Watts article misleads readers to believe there is strong and convincing evidence based on neuroscientific research that excessive pornography problems constitute an addictive disorder causing brain abnormalities and cortical atrophy paralleling those found in substance abuse. Such assertions are speculative and unsupported by the studies cited by Hilton and Watts. Even if future research substantiates such claims, it is highly unlikely that such results will be generalized to all patients with excessive pornography problems given the consistent finding of heterogeneity in the characteristics of this population. We believe that addiction models may limit our understanding of this population and likely offer too simplistic a view of the vast array of complex issues encountered by patients with hypersexuality and pornography problems. In the interim, current research offers little support for conceptualizing excessive pornography problems as an addictive disorder. Research on tolerance or withdrawal, genetic associations, and neuroimaging in hypersexual patients with pornography problems are non-existent at this time. Although excessive pornography problems are part of the current proposed criteria for classification of HD in the forthcoming DSM-5, the field trial results have not been published and it is unclear whether such classification is valid or if it can be reliably diagnosed. Although the perspectives of Hilton and Watts may be appealing to some, we caution your readers in using their article to support or substantiate excessive pornography use as an addictive disorder based on the findings they attribute to neuroscience research. Collectively, their errors are egregious and detract from, rather than support, serious hypotheses for future research. In our own work with these patients, at least for those who seek treatment, the frequently attendant dysfunction in occupational, social, and other important activities, is sufficiently negative on its own, creating true dysfunction and significant clinical distress. We see no reason to exaggerate the known risks by suggesting

that excessive pornography consumption leads to brain damage or other neuropathology. Admittedly, some are prone to dismiss pornography use of any kind as a natural outgrowth of human sexuality; however, those who study and work with these extreme cases are well aware of the difficulties encountered by these individuals, including their sense of frustration about the inability to reduce or stop their problematic behaviors despite negative consequences. We look forward to future work offering empirically derived perspectives on these conditions, including the associated neurological correlates, but preferably insights that remain within the scope of what the research data supports.

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Commentary on: Neuroscience research fails to support claims that excessive pornography consumption causes brain damage

Perhaps the most startling aspect of the response to our editorial is the lack of understanding, perspective, or acknowledgment that there is a growing and credible body of research strongly supporting the existence of natural addiction, which encompasses pornography addiction. It is apparent the authors have rejected this premise of natural addiction that leading addiction neurobiologists continue to support, and therefore it is not surprising that they would view pornography addiction with skepticism.

Particularly noteworthy is their lack of awareness of the growing evidence of Δ FosB and its role as a molecular switch in addictive states, both drug and natural. For an understanding of the current perspective on Δ FosB, Dr. Eric Nestler's review paper published in the *Philosophical Transactions of the Royal Society* is most helpful. This paper was published in the issue titled "The Neurobiology of Addiction – new vistas," in summary of a discussion meeting of prominent addiction neurobiologists.^[24]

Because of confusion the commenters may have created with their comments, some of the main points of Nestler's paper, and even more recent work on Δ FosB in relation to sexuality will be reviewed in this response.

Δ FosB is a member of the Fos family transcription factors.^[24] Fos family proteins are induced by administration of drugs of abuse. These proteins, in general, are released and degraded quickly, with their point of action focusing on reward areas such as the nucleus accumbens and the dorsal striatum. They are unstable, and are gone within hours.

Δ FosB differs from other members of the Fos family in that it accumulates with drug abuse, across the spectrum of drugs of abuse.^[19] Thus Δ FosB continues to exert changes in gene expression even during periods of drug withdrawal. Nestler and others have proposed that Δ FosB is a "sustained 'molecular switch' that helps initiate and maintain an addicted state."^[24]

Bitransgenic mice can be induced to selectively produce Δ FosB in the dynorphin-containing medium spiny neurons, which is specifically where drugs of abuse are thought to exert their effect. They show an exaggerated behavioral response to drugs of abuse, as if they had been chronically given the drug, as compared to mice who do not inherently overexpress Δ FosB; this phenomenon is seen both with cocaine^[14] and opioids.^[33] Studies with mice which are induced to overexpress Δ FosB in a viral-mediated gene transfer model have replicated this response.^[33] In other words, drug naïve animals overexpressing Δ FosB behave as if they are already addicted.

So how does this relate to the existence of natural addiction? The purpose of the nucleus accumbens is integral in salience of natural reward behaviors such as food, sex and rewarding interpersonal interactions. Nestler discusses the evidence supporting a role for Δ FosB in the nucleus accumbens in "so-called natural addictions: e.g., pathological overeating, gambling, exercise, sexual addiction."^[24] Significantly, Δ FosB accumulates in the nucleus accumbens in mice that exhibit higher levels of wheel running than normal, a model for exercise addiction.^[32] This also occurs after chronic over consumption of sucrose or sex.^[30] The viral-mediated mice overexpressing Δ FosB referred to earlier which exhibit behavior consistent with drug addiction also "increases drive and consumption for these natural rewards."^[23] Blocking the action of Δ FosB in these animals with an antagonist (dominant negative Jun protein) prevents overconsumption of these natural rewards. Dr. Nestler summarizes: "These findings suggest that Δ FosB in this brain region sensitizes animals not only for drug rewards but for natural rewards as well, and may contribute to a state of natural addiction."^[24]

Other recent studies strengthen the premise that sexuality is strongly tied to Δ FosB, a marker of addiction. For instance, Δ FosB overexpression in the nucleus accumbens has been shown to enhance sexual reward in female Syrian hamsters.^[18] Pitchers *et al.*, published a paper last year demonstrating that sexual experience causes an accumulation of Δ FosB in limbic-associated brain regions, such as the nucleus accumbens core and shell, the medial prefrontal cortex, the ventral tegmental area and the caudate putamen. Significantly, "blocking Δ FosB attenuated experience-induced facilitation of sexual motivation and performance, while *overexpression of Δ FosB in the nucleus accumbens caused an enhanced facilitation of sexual behavior, in terms of increased sexual performance with less experience.*"^[27] (emphasis added) This is most interesting when considered in light of Nestler's comment in the Royal Society paper, that the level of Δ FosB may become a "biomarker to assess the state of activation of an individual's reward circuitry, as well as the degree to which an individual is 'addicted', both during the development of an addiction and its gradual waning during extended withdrawal or treatment."^[27] These perspectives are clearly supportive of a neurobiological marker for sexual addiction. Pitchers *et al.*, work summarizes "...these data are the first to indicate an obligatory role of Δ FosB in the acquisition of experience-induced facilitation of sexual behavior... *We propose that this long-term expression of facilitated behavior represents a form of memory for natural reward; hence, Δ FosB in NAc is a mediator of reward memory.*"^[16] (emphasis added) Another paper from Pitchers *et al.*, last year established that physiologic sexual experience, interrupted by a period of abstinence with resumption of sexual behavior actually increases numbers of dendrites and dendritic spines in medium spiny neurons. Again they summarize, "The structural alterations induced by sexual experience and subsequent abstinence resemble those seen after repeated exposure to psychostimulants... *the data presented here demonstrate that sexual behavior – a natural rewarding stimulus – can induce long-lasting neuroadaptation in the mesolimbic system.* Our findings suggest that behavioral plasticity, particularly a sensitized locomotor response, is an immediate and long-term outcome of sexual experience."^[26] (emphasis added)

Another metabolic parameter strongly supporting a neurobiological basis for natural addiction is found in studies examining dopamine receptor depletion. Wang *et al.*, demonstrated dopamine (D2) receptor downgrading with obesity similar to that seen in drug addiction, and the levels correlated with BMI.^[31] An animal study recently published by Johnson and Kenny found that rats exposed to "palatable, energy-dense food develop a profound state of reward hyposensitivity and compulsive-like eating. The maladaptive behavioral

responses in obese rats probably arise from diet-induced deficits in striatal D2R signaling. Overconsumption of drugs of abuse similarly decreases striatal D2 receptor density, induces a profound state of reward hypofunction, and triggers the emergence of compulsive-like drug taking behaviors. *Our findings therefore support previous work in indicating that obesity and drug addiction may arise from similar neuroadaptive responses in brain reward circuits.*^[20] (emphasis added)

Pathologic gambling has demonstrated decreased activation in the mesolimbic reward system as compared to controls,^[28] and administration of dopamine to patients with Parkinson's disease has iatrogenically induced both hypersexuality and pathological gambling.^[13] Reuter *et al.*, summarizes "...a decrease activation of the ventral striatum, which is a hallmark of drug addiction, and decreased VMPFC activation, which is related to impaired impulse control, favor the view that pathological gambling is a non-substance-related addiction."^[28] (emphasis added)

In our opinion the seminal work on Δ FosB by Nestle and others is pioneering, and changes the landscape in considering aspects of neuromodulation as related to natural addiction. It casts a biologic light on all aspects of this concept. We feel this data is confirmatory with regard to the existence of neuromodulation in natural addiction, especially considering the recent work exploring the relationship between Δ FosB and sexuality. The points we made on the VBM studies regarding hypoplasia of neuronal populations associated with reward centers emanated from this perspective. These correlative papers concluded that atrophy occurred in four different addictive states, two drug and two natural. Certainly the authors of these papers were not addressing causation, although the cocaine^[14] and obesity^[25] papers both recognize that *the areas of atrophy are associated with reward pathways*. Inherency, which certainly may be a factor, does not explain the reversibility, with recovery, of selective atrophy associated with the use of methamphetamine.^[22]

Our premise is that selective atrophy of cortical areas associated with reward pathways may be viewed in a neuromodulatory light, given current research confirming neuroplasticity in overindulgence in natural rewards, specifically sexuality. The inability of those challenging our conclusions to understand even the most basic of these concepts is illustrated by their comments about specific processes. For instance, their dismissal of the importance of Δ FosB is illustrated by their manifest lack of insight into the research concerning this protein. While mentioning that stress can induce Δ FosB, they fail to understand that the pattern of expression with stress extends broadly across both dynorphin+ and enkephalin+ medium spiny neurons and is not confined

to dynorphin+ medium spiny neurons as it is in the overexpression associated with overconsumption of natural rewards and with drug addiction.^[24] The following comment is illustrative of their lack of understanding the importance of Δ FosB as a molecular switch in addiction: "That's great news for the sexually inexperienced rats! Put your name on the list for more Δ FosB and your sexual performance will be on par with more experienced rats." They correctly point out that atrophy is associated with aging, and may be affected by comorbidities, neurotoxicity, and the like, but fail to appreciate selective atrophy in cortical areas associated with reward centers. The concept of upstream neuronal atresia as illustrated in figure 2 in the Nestler paper, focusing on a common pathway for drug and natural addiction,^[23] may be an important mediator in this process. This atresia is associated with decreased dopaminergic input into the NA from the VTA, and with decreased glutaminergic input from the cortex, both being associated with an increase in Δ FosB in the medium spiny neuron. The cortex can atrophy in response to decreased downstream stimuli.^[21] That there is a functional frontal deficit in addiction is the hallmark, whether drug induced or naturally induced, and the hypofrontal syndrome displayed is similar to that seen in traumatic brain injury.^[16] Another recently reported example of selective cortical atrophy in reward-associated regions in adolescents manifesting Internet "addiction" is of interest in this context.^[34]

While a role for inherency is obvious, to deny any role for causation is to envision a world of selectively preatrophied individuals destined to act out in addiction. We find this premise much less plausible than at least a partial role for causation given what we consider confirmatory data with regard to the role of Δ FosB in the induction and then perpetuation of addictive states.

Whether or not future structural studies confirm our premise that at least partial causation is supported in this regard, the question of neuromodulation with regard to natural addiction is independently supported by the Δ FosB studies, and strengthened by the D2R and fMRI studies on obesity and pathological gambling previously cited. Particularly convincing with regard to a causation role of subsequent addictive behavior after induction is the previously cited work on bitransgenic and virally induced mice which *behave as if addicted*, both in natural and drug addiction, overexpression of Δ FosB being the only variable.^[24]

As stated in our editorial, no less that the head of the National Institute for Drug Abuse (NIDA), Dr. Nora Volkow, called, in the journal Science, for changing the name of the NIDA to the National Institute on Diseases of Addiction, to "encompass addictions such as pornography, gambling, and food...She would like

to send the message that we should look at the whole field.”^[15] Dr. Eric Nestler at Mount Sinai uses the phrase “natural addiction” in describing what he calls “pathological overeating, pathological gambling, and sexual addictions.” Dr. Howard Shaffer at Harvard said, “I had great difficulty with my own colleagues when I suggested that a lot of addiction is the result of experience...” and continued, “Although it is possible to debate whether we should include substance or process addictions within the kingdom of addiction, technically there is little choice.”^[29] When scientists such as Drs. Volkow, Nestler and Shaffer use the word “addiction” with regard to processes such as food and sex, they are not using this term lightly. Neurobiologists understand that this word has neuromodulatory implication.

For Reid *et al.*, to suggest to the reader that it is irresponsible to use the word addiction in this context, we believe, is irresponsible. They seem to ignore substantial evidence that natural addictions do indeed exist, and that specifically sexual addiction can induce neuroplasticity. They fail to grasp the significance of neuromodulation in sexuality when they state, “...current research offers little support for conceptualizing excessive pornography problems as an addictive disorder.” If natural addiction exists, as we and others believe, then it strains credibility to argue that patients struggling with pornography addiction like the one described by Bostwick and Buccini are not prime examples.^[12]

Recently a colleague experienced in functional neurosurgery was visiting with another similarly experienced neurosurgeon. This latter surgeon opined that the next field which might be addressed through functional neurosurgery may be addiction. However, unlikely it appears now to some, we envision a day when drug addiction, severe obesity and sexual addictions with legal implications might be treated with limbic targeting, hence the relevance to our present subject.

We found the perspective and tone of these authors disappointing, in that they are desperately dismissive of any neurobiologic evidence supporting natural models of addiction. Particularly remarkable, in our opinion, is their blatant disregard for the context which leading neurobiologists view not only Δ FosB, but any data which supports neuromodulation in natural addiction. In refutation, the only evidence they cite is their own work, which is behavioral in nature, rather than neurobiologically based. Their perspective is permeated with an apologetic bias against any study suggesting pathologic neuromodulation on a macro or micro scale with regard to natural addiction.

As of this writing a report out of Yale published in the Archives of General Psychiatry titled “Neural Correlates of Food Addiction” describes activation in reward pathways using fMRI as being similar in obese individuals and in

those with substance addiction. They summarize, “the current findings suggest that food *addiction* is associated with reward-related neural activation that is frequently implicated in substance dependence. To our knowledge, this is the first study to link indicators of *addictive eating behavior with a specific pattern of neural activation.*”^[17] (emphasis added) Emerging data continues to strengthen and support the concept of neuromodulation with natural addiction.

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Donald L. Hilton Jr., Clark Watts¹

Department of Neurosurgery, University of Texas Health Science Center at San Antonio, San Antonio, ¹University of Texas School of Law, Austin, TX, USA

E-mail: cwatts@mindspring.com