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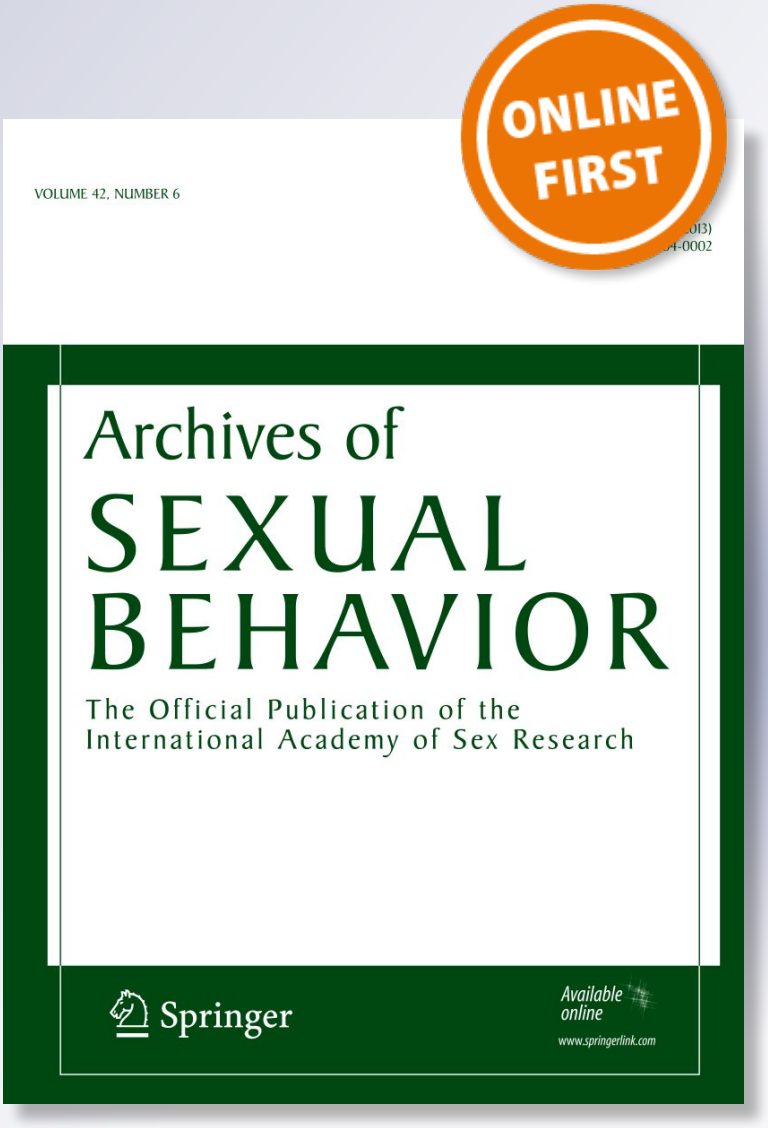

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COMMENTARY

Separating Models Obscures the Scientific Underpinnings of Sex Addiction as a Disorder

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A foundational concern we have about the Target Article by Walton, Cantor, Bhullar, and Lykins (2017) involves its structure. For example, the manner in which Walton et al. explore various theoretical approaches by dividing them into subtopics is problematic. Of greatest concern is the separation of research on sex addiction from research that utilizes other terms that describe the same basic phenomenon, and from the coverage of neurobiology as it relates to this issue.

Starting with the latter concern, Walton et al. present a largely phenomenological description of sex addiction, and the primary reference to neurobiology in this section is the problematic statement, "The conceptualization of sexual behavior as an addiction has long been criticized, as research has failed to substantiate physiological conditions of tolerance and withdrawal." We suggest a more accurate wording would be, "The conceptualization of sexual behavior as an addiction has been inaccurately criticized, as some researchers have failed to understand that tolerance and withdrawal are an affective process presenting in the same manner in sex addicts, as in gamblers and substance addicts."

Use of tolerance and withdrawal as diagnostic criteria for addictions is widely accepted and clearly articulated in the three-phase model of addiction put forth by the Directors of both NIDA and NIAA (Koob & Volkow, 2010; Volkow, Wang, Fowler, Tomasi, & Telang, 2011). Consider, for example, the American Psychiatric Association's (2013) first two criteria related to Gambling Disorder:

1. Needs to gamble with increasing amounts of money in order to achieve the desired excitement.
2. Is restless or irritable when attempting to cut down or stop gambling

Moreover, the neurobiology behind this has repeatedly been uncovered as part of an addict's neurobiological desensitization (Volkow et al., 2010). Regarding sexual behavior specifically, multiple studies, involving neuroimaging and neuropsychological, have found both tolerance and withdrawal, especially with Internet pornography addiction (Banca et al., 2016; Gola et al., 2017).

Another problematic assertion about neurobiology in the sex addiction subsection is the claim, "To date, the neurobiology of sex addiction has been difficult to empirically research and validate." We disagree. In fact, research on how chronic engagement in sexual behavior can lead to addiction is so plentiful that the American Society of Addiction Medicine (ASAM) includes sex as a specific behavioral example in their definition of addiction:

> 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 (American Society of Addiction Medicine, 2011).

Interestingly, the ASAM definition is briefly incorporated into the Walton et al. paper. However, it was only listed in the neurobiology section of their article, and the term "addiction" was excluded. Additionally, Walton et al. begin and end their section on neurobiology with research that supports the addiction model, but they do not use the word addiction, and they

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only minimally acknowledge that their information came from the field of generalized addiction research. For example, Robinson and Berridge (2008) are among the foremost leaders in addiction research, and their incentive salience model is an addiction model not specifically linked to hypersexuality. Nevertheless, in their paper, "The Incentive Sensitization Theory of Addiction," they state that "incentive sensitization can also sometimes spill over in animals or humans to other targets, such as food, sex, gambling, etc." (Robinson & Berridge, 2008, p. 3138). Still, Walton et al. place this paper in the neurobiology section only, separated and distinct from their discussion of sexual addiction. Other relevant addiction-related neuroscience is scattered elsewhere in the paper, outside of the sex addiction section, with, at best, a nominal connection to the overall issue.

On the plus side, we do agree with Walton et al.'s recognition of the difficulties presented by the lack of consistent nomenclature. Unfortunately, this paper does little to reverse the trend. In fact, by divvying up the research and spreading it around in differently named sections (Hypersexual Disorder, Compulsive Sexual Behavior, Sex Addiction, Neurobiology, etc.), this paper furthers the problem and leads to misinterpretation. For example, in the hypersexual disorder discussion Walton et al. state, "Although in its infancy, research has recently recognized the possible relationship between neurological systems, executive functioning, brain pathology, and hypersexuality." This is misleading; while the statement is technically accurate in that there are only a small number of neuroscience papers focused specifically on the term "hypersexuality," Walton et al. could have expanded their scope to all of the related terminology and thus could have included other recent and important research, including "Neurobiology of Compulsive Sexual Behavior: Emerging Science" (Kraus, Voon, & Potenza, 2016), "The Neurobiology of Internet Pornography Addiction: A Review and Update" (Love, Laier, Brand, Hatch, & Hajela, 2015), "Neuroscientific Approaches to (Online) Pornography Addiction" (Stark & Klucken, 2017), and "The Neurobiology of Behavioral Addictions: Sex Addiction" (Hilton, Carnes, & Love, 2016). Moreover, Walton et al. also failed to reference a major paper explicitly focused on hypersexuality, "Neurobiological Basis of Hypersexuality" (Kühn & Gallinat, 2016).

Just as unfortunately, in this incomplete literature review, there is minimal reference to and no discussion of the larger research base supporting the behavioral addiction framework. Instead, Walton et al. present the material in a vacuum, omitting the foundation of the relevant neuroscience (much of which, as discussed above, is also left out).

In actuality, research is robust in the area of natural rewards and behavioral addictions. In fact, there have been multiple reviews examining the research on behavioral addictions (Banz, Yip, Yau, & Potenza, 2016; Olsen, 2011). For example, Volkow, Koob, and McLellan (2016) openly acknowledge sex as having the potential to become a behavioral addiction. Some of this research gets all the way down to a molecular understanding. For instance, research published in *Nature: Neuroscience* specifically states that sexual addiction activates the same neural pathways as other addictions (Nestler, 2005).

The "Sexhavior" Cycle

We find the "sexhavior cycle" perplexing in its purported uniqueness, as it is unclear how this model is substantially different than the addictive system model proposed by Carnes (1983) over 30 years ago. Additionally, "cognitive abeyance" sounds very similar to what the addiction model already refers to as hypofrontality (Volkow et al., 2010). In their three-phase model of addiction, Koob and Volkow (2010) refer the third phase as "preoccupation and anticipation," and they go into explicit detail regarding the dysfunctional neuroadaptations of impaired executive functioning, down to the level of neurocircuits, synaptic systems, molecules, and epigenetics.

In some ways, the title of a 2011 article in *Nature Reviews Neuroscience* says it all, "Dysfunction in the Prefrontal Cortex in Addiction" (Goldstein & Volkow, 2011). The simple truth is there are plenty of neuropsychological studies focused on impaired executive functioning, at least a few of which are specific to the problem of Internet pornography addiction (Messina, Fuentes, Tavares, Abdo, & Scanavino, 2017; Negash, Sheppard, Lambert, & Fincham, 2016). Furthermore, Brand, Young, Laier, Wölfling, and Potenza (2016) recently presented their own highly detailed Interaction of Person-Affect-Cognition-Execution (I-PACE) model, wherein they discuss the interaction of cognitive variables and factors involved with Internet pornography addition. As such, it is hard to understand how cognitive abeyance is a unique explanation of the impaired thinking processes of sex addicts.

In the opening and closing of their article, Walton et al. posit the question of whether hypersexuality is a distinct clinical disorder. We suggest that future research should instead focus on consolidation of the various existing models used in current and ongoing research. Our belief is that the current language conflation is a huge problem in the field, as we see in this article. If we unify the terminology, however, it becomes abundantly clear that neurobiological and neuropsychological studies on the phenomenon of sexual addiction provide a definite convergence of findings falling well within the generally accepted bounds of the addiction model. From there, the question of whether "hypersexual disorder" exists beyond the addiction scope can be readdressed. Ultimately, that will be an important question, as Walton et al. do illustrate other valid, non-addiction-related examples of hypersexual behavior (dementia, Parkinson's, medication related, and expressions of other mental health disorders such as bipolar).

We think it is important to clarify that sexual behavior, in and of itself, should *not* be considered an addiction. Sexuality is a healthy and natural reward, similar to food, that the mammalian brain is organically wired to enjoy. However, as Doidge (2007)

states in his book, *The Brain That Changes Itself*, chronic engagement in natural rewards, including sexual behaviors, can result in addiction-related alterations to the brain.

It is also important to understand that sex/porn addiction is *not a sexual disorder*. It is not even "about sex." It is an addictive disorder. Continuing to use the lens of hypersexuality enables claims such as that subjects have "high sexual desire," when, in reality, people struggling with sex/porn addiction are no more "horny" than alcoholics are "thirsty" or food addicts are "hungry" (Hilton, 2014). This claim is well evidenced by the fact that the current head of the NIDA has become a leading researcher in, and proponent of, calling the compulsive use of food an addiction rather than an eating disorder (Volkow, Wang, Fowler, & Telang, 2008; Volkow & Wise, 2005).

Lastly, we wholeheartedly agree that case presentations are diverse. As such, we stress the critical importance of a proper differential diagnosis. It is possible that the various non-addiction-related conceptualizations Walton et al. have presented are valid explanations for some forms of volitional sexual behavior. Indeed, many conceptualizations for similar-appearing sexual behavior can simultaneously be true, and the expectation of a single model to explain all cases is reductionist and unrealistic.

That said, we are unclear on the need to add yet another model, "the sexhavior cycle," to the mix. Rather, we encourage Walton et al. to contribute to the field by helping to integrate existing science, instead of viewing the existing and continuing body of research via independent silos. Rather than framing an existential search for whether something "exists" (that clearly does), this black and white thinking needs to end for the field to move forward. It is time to accept the heterogeneity of presentation and see the commonality in both drug and behavioral addictions, including sexual addiction.

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