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The neurobiology of digital addiction: a review of the dopaminergic system, variable reward, and attention control.

The neurobiology of digital dependence: a review of the dopaminergic system, variable reward, and attentional control

Pedro Staciarini Silveira Rodrigues - UNAERP - pedrossr4@icloud.com
Giordano de Toledo Palumbo Walderrama - UNAERP - giordanotpwalderrama@gmail.com

Summary

The use of the internet and social media has become a central part of modern life. However, when used excessively, it can evolve into patterns similar to those observed in addictive disorders, such as craving, tolerance, and withdrawal. This work gathers and compares evidence from four selected studies to understand how the dopaminergic system relates to digital addiction and what the neurological and cognitive consequences of this continuous use are. The integrated analysis of the data shows that digital platforms—especially short video applications—
They utilize unpredictable reward schemes capable of intensely activating the brain's reward system. This repeated activation is associated with a reduction in dopamine transporters (DATs) in the striatum, indicating a possible dopaminergic dysfunction. In behavior, this alteration manifests as a compulsive search for rapid stimuli ("TikTok brain") and decreased attentional control. Despite the consistency of the findings, the available literature still presents methodological limitations, reinforcing the need for longitudinal studies.

Keywords: Digital addiction; Dopamine; Attentional control.

Abstract

The use of the internet and social media has become a central part of modern life. However, when used excessively, it can develop into patterns similar to those observed in addictive disorders, including craving, tolerance, and withdrawal. This paper brings together and compares evidence from four selected studies to examine how the dopaminergic system relates to digital addiction and to identify the neurological and cognitive consequences of chronic use. The integrated analysis of these studies shows that digital platforms—particularly short-video applications—rely on unpredictable reward schedules that strongly activate the brain's reward system. Repeated activation is associated with reduced dopamine transporters (DATs) in the striatum, indicating a potential dopaminergic dysfunction. Behaviorally, this alteration manifests as a compulsive search for rapid stimuli (“TikTok brain”) and diminished attentional control. Despite the consistency across findings, the available literature still presents methodological limitations, highlighting the need for longitudinal studies.
Keywords: Digital addiction; Dopamine; Attentional control.

1. Introduction

The use of the internet and social media has grown in the last decade, becoming a part
A holistic aspect of people's lives worldwide. Various platforms offer undeniable benefits.
such as global connectivity and easy access to information. However, maladaptive use and
The excessive use of these technologies has resulted in the emergence of new behavioral pathologies.
Internet Addiction Disorder (IAD), a concept first raised in the 1990s, has become "more prevalent
worldwide."1 Its impact is devastating, frequently associated
harm to psychological well-being, academic failure, and reduced work performance, especially among
adolescents.1 Recently, a new facet of this problem has emerged with

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Extreme speed: addiction to short videos, fueled by apps like TikTok. This

This media format already "exceeds 22 hours per user" monthly, surpassing mobile games and becoming the fastest-growing segment in the entertainment industry.1 The rapid

The growth and immersive nature of these apps have led to addiction, generating "concerns public" and the recognition of addiction to short videos as a "significant public health problem."1 The algorithmic and compulsive nature of these platforms has earned them nicknames such as "digital fentanyl" or "electronic opium."1 The central thesis justifying the investigation of these phenomena

The fact is that they don't merely represent "bad habits," but rather disorders with underlying causes. specific neurobiological abnormalities. Recent studies have found that IAD shares "abnormalities Neurobiological characteristics similar to other addictive disorders, notably substance abuse and pathological gambling.1 Individuals suffering from IAD exhibit the classic clinical features of addiction, including *craving , withdrawal ,* and tolerance.1 The central neural system implicated in this pathological process is the dopaminergic system.1 Dopamine (DA), a

Dopamine, a catecholaminergic neurotransmitter, is fundamental in reward pathways and motivated behavior.1 Often described as the "pleasure chemical , " dopamine

It is the central agent through which digital addiction takes hold and perpetuates itself, through "mechanisms of *feedback loops* that act directly on the dopamine reward system.1 The goal

The general objective of this work is to conduct a literature review to synthesize the neurobiological evidence. that link the dopaminergic system to internet and social media addiction, and discuss the pathological and cognitive consequences resulting from this chronic use.

2. Materials and Methods

This is a literature review with a focused scope. The methodological objective is not... Not to conduct a comprehensive systematic review of the entire field of digital addiction, but rather to synthesize. and to qualitatively analyze the specific data contained exclusively in the four sources of research provided [1] to construct a cohesive narrative on the topic.

The "materials" or "instruments" [1] The four articles used for this analysis were: pre-selected scientists, each providing a distinct methodological perspective on the problem:

1. A neuroimaging study by Hou et al. (2012), which used Single Photon Emission Computed Tomography (SPECT) to compare dopamine transporters in individuals with Internet Addiction Disorder (IAD) and healthy controls.1
2. A fundamental neuroscience study by Berns et al. (2001), which used Imaging by Functional magnetic resonance imaging (fMRI) to investigate how the predictability of primary rewards (juice and water) modulates brain response in humans.1

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3. A review article by Burhan and Moradzadeh (2020), which describes the role of the neurotransmitter dopamine (DA) and its *feedback loop* mechanisms in the development of addiction to social media.1

4. A quantitative (cross-sectional) study by Ye et al. (2025), which explored the relationships between the Intensity of short video use, addiction, the "TikTok brain" phenomenon, and decreased control. attention span in students.1

The "method of data analysis" [1] It consisted of a qualitative and comparative synthesis. The Findings from each source were extracted, analyzed, and juxtaposed to construct an explanatory model. unified. The analysis focused on identifying the neuropsychological mechanism. [1], neurobiological pathology resulting [1] and the observed cognitive-behavioral consequences [1], following a causal logic which goes from stimulus to symptom.

3. Results and Discussion

A clear progression can be observed from the psychological mechanism explored by The platforms lead to neurological pathology and, ultimately, to the cognitive deficits observed in users. A fundamental mechanism of social media addiction is the exploitation of the dopaminergic reward system.1 Burhan and Moradzadeh (2020) identify that addiction is perpetuated by " *feedback loop mechanisms".1* The specific method that these applications exploit is that of "Variable Reward Schedules *".1* This concept, originating from Behaviorism, as described, is analogous to gambling. The reward (a *like,* a message, an interesting video) is provided at random and unpredictable intervals.¹

The effectiveness of this mechanism lies in the neurobiology of anticipation. The authors highlight that, during the period of "intense anticipation" (i.e., the act of *scrolling* or checking the phone), dopamine neurons are "very active and firing."1 Most of the time, the user finds a neutral stimulus (an uninteresting post). However, occasionally, a "positive stimulus" occurs, leading to the "release of rewarding dopamine." It is this variability that keeps the user in a *loop,* creating a compulsion to check.1

The study by Berns et al. (2001) provides direct neurobiological validation for this theory. explaining *why* unpredictability is such a powerful trigger.1 Using fMRI, the researchers They administered primary rewards (fruit juice and water) to participants under two conditions: one a predictable sequence (alternating juice and water at fixed intervals) and an unpredictable sequence. (randomized order and time). [1] The main finding of the study was that activity in regions Key brain regions for reward processing, specifically the nucleus accumbens and the medial orbitofrontal cortex, were "larger when stimuli were unpredictable."1 Notably, the

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explicit subjective preference of the participants (for example, preferring juice to water)

It did not correlate with the activation of the nucleus accumbens; instead, it correlated with the sensorimotor cortex.1 This suggests that the dopaminergic reward system in the nucleus accumbens It is less attuned to the intrinsic value of the reward (liking) and more attuned to the surprise or predictive error of the reward (wanting).1

When combined, the findings of Berns et al. [1] and Burhan and Moradzadeh [1] they fit Perfectly. Infinite *scrolling* in a short video application is a functional [1] or notification verification [1] and replication in the real world of the experimental paradigm of unpredictability.1 The design These apps deliberately exploit the neurobiological mechanism that maximizes the activation of The nucleus accumbens, through unpredictability, creates a highly dopaminergic search cycle. compulsive. If this is the mechanics of overstimulation, the next question is: what are the What are the long-term pathological consequences of this chronic stimulation? The study by Hou et al. (2012) investigated exactly that, comparing individuals diagnosed with Internet Addiction Disorder (IAD) with healthy controls.1 Using SPECT neuroimaging to visualize and to quantify dopamine transporters (DATs) — proteins in the presynaptic membrane responsible for the reuptake of dopamine and the regulation of its synaptic levels — the results They were alarming.1

The study found that "striatal DAT expression levels were significantly decreased" in subjects with IAD compared to controls.1 The authors suggest that this A reduction in DATs "may possibly reflect a pronounced loss of dopamine terminals" or an "impairment in cerebral dopaminergic function."1 Essentially, in response to chronic flooding Through the release of dopamine (caused by the variable reward mechanism), the brain appears to regulate itself. negatively (down-regulation), resulting in measurable physical damage to the system. dopaminergic. Hou et al. [1] They claim that these neuroimaging findings illustrate that the IAD is associated with "dysfunctions in the brain's dopaminergic systems" and "support the claim that IAD "It may share similar neurobiological abnormalities with other addictive disorders." such as substance abuse.1

The magnitude of this damage is quantified in Table 1 of the study. [1]which compares the parameters of the striatum (Volume, Weight and Uptake Ratio) between the groups.

Table 1 – Comparison of striatal body parameters (V, W, Ra) between subjects with IAD and controls.

	Volume of corpus striatum V (cm^3)		Weight of corpus striatum W (g)		Ratio of corpus striatum/whole brain
	VL (left side)	VR (right side)	WL (left side)	WR (right side)	Ra (%)
Controls ($n = 9$)	16.69 ± 1.63	18.17 ± 2.00	17.53 ± 1.71	19.08 ± 2.10	7.93 ± 0.75
IAD subjects ($n = 5$)	11.72 ± 3.13*	12.51 ± 2.53**	12.30 ± 3.28*	13.14 ± 2.10**	5.38 ± 0.77**

Values are expressed as mean ± SD with $^{+}P < 0.05$ and $^{**}P < 0.01$.

(Source: Adapted from Hou et al., 2012, p. 4 1)

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The data in Table 1 are compelling, showing reductions in the IAD group of approximately 30% in the volume and weight of the striatum, and a 32% reduction in the uptake ratio (Ra), which reflects the density of functional DATs.1 The authors also describe the visual images, where the DATs of the controllers had a uniform "panda eye" shape and symmetrical, while subjects with IAD exhibited DATs that were "much smaller" and with irregular shapes ("dumbbell, thin band, moon shape, or sporadic dot").1

Finally, the study by Ye et al. (2025) [1] connects this neurobiological pathology [1] to the Cognitive and behavioral consequences seen in users of short videos. The study identified significant positive correlations between "intensity of use of short videos" and the "decreased attentional control."1 In addition, the study explores the construct "TikTok brain," defined as "perceived mood enhancement ."1 This phenomenon is described as a psychological pattern resulting from prolonged consumption of short videos, specifically "the need to continually obtain high emotional arousal through short videos" in order to experience pleasure.1

The final synthesis of the four sources allows for the following unified conclusion: The "TikTok brain" [1] It is the *behavioral description* of *craving.* [1] who relentlessly seeks the reward *mechanism* variable.1 A neurobiological "decrease in attentional [1] It is the *cognitive consequence* of the *pathology.* control" resulting from a reduction in DATs.1 A brain that has suffered a "loss of dopamine terminals" [1] will have a dysfunctional dopaminergic system. This system is essential not only for reward, but also for motivation, salience, and executive functions (such as focus). Therefore, when the An individual attempts to perform a low-reward but cognitively demanding task (such as studying). or work), your impaired dopaminergic system [1] failure to provide motivation and control attention spans are necessary. In contrast, the short video app offers a dopamine rush. easy, fast and unpredictable [1] that the damaged brain is still able to process, perpetuating the cycle. Ye et al. [1] They confirm that addicted users are "more prone to distraction" and that video addiction... Short strokes represent a "greater risk of cognitive deficits".1

Final Considerations

This literature review, based on a synthesis of the four sources analyzed, demonstrates that digital addiction, ranging from Internet Addiction Disorder (IAD) to addiction to Making short videos isn't a moral failing or a lack of willpower, but a neurobiological process. documented.1

It has been shown that the design of modern media platforms exploits the mechanism The fundamental aspect of reward *unpredictability* is that this mechanism maximizes dopaminergic activation in the nucleus accumbens, creating a compulsive seeking cycle.1

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Chronic use and the resulting overstimulation are associated with neurological pathology. Measurable: a significant reduction in dopamine transporters (DATs) in the striatum, a dysfunction analogous to that observed in substance use disorders.1

This dopaminergic dysfunction manifests cognitively as the "TikTok" phenomenon. "brain" — a compulsive search for instant gratification — and results in a "decrease in "Attention control," impairing individuals' ability to concentrate on cognitively demanding tasks.1

The main limitation identified is the "small sample size" (n=5 IAD) in the study, which "limits the neuroimaging by Hou et al. (2012) [1], generalizability" of the pathological findings.1

Additionally, the construct "TikTok brain" is described by Ye et al. (2025) [1] as "Preliminary and unproven," requiring further validation. The cross-sectional design of this study It also "prevents the establishment of causal relationships" between the use of short videos and the decline. attention.1

Therefore, future research, as suggested [1], should employ "experimental projects or "Longitudinal data" to confirm causality. Given the prevalence of use and the "potentials" adverse impacts [1], awareness campaigns about the neurobiology of digital addiction It is urgent to maximize the positive utility of these tools and minimize their harm to physical and mental well-being.1

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